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A PRACTICAL TREATISE

ON

DISEASES OF THE SKIN

FOR THE USE OF

STUDENTS AND PRACTITIONERS

BY

OLIVER S. ORMSBY, M.D.

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PREFACE TO THE SECOND EDITION.

In order to incorporate new material and yet keep this book of reasonable size, the author has reconstructed this edition to such extent as he deemed necessary. In consequence, approximately four hundred pages have been rewritten, fifteen new diseases described and the entire work brought up to date so far as possible.

The dermatological literature has been carefully reviewed in order that the pages of this book may reflect its subject as completely as the limits of a single volume permit. The opinions of experienced dermatologists are freely quoted. It has been possible to reduce the rapidly increasing literature references by selecting articles on the various subjects containing complete bibliography to the date of publication. These will serve as a guide to the investigative student in working up any particular subject.

Many of the older illustrations have been replaced and a large number of new ones added from the collection of the author and Dr. Mitchell, as well as through the generous contributions of colleagues. The author is deeply grateful to Drs. John A. Fordyce and George M. MacKee for a large number of new illustrations in addition to those employed in the previous edition. He is further indebted to Drs. Howard Morrow, Fred Wise, Martin F. Engman, William H. Mook and Udo J. Wile for photographs kindly furnished, and his thanks are due the following gentlemen for the privilege of producing those heretofore used: Drs. George Henry Fox, Howard Fox, A. D. Mewborn, Douglas W. Montgomery, Howard Morrow, Ernest E. Tyzzer, Stopford Taylor, David Lieberthal, Heman Spalding, Ernest L. McEwen, and the late M. L. Heidingsfeld.

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51321

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He further desires to express to the publishers, Messrs. Lea & Febiger his appreciation for the uniform courtesy extended while these pages were passing through the press.

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Chicago, Illinois, 1921.

CONTENTS.

Introduction 17
I. Anatomy and Physiology of the Skin 17
II. General Symptomatology 53
III. General Etiology 62
IV. General Pathology 68
V. General Diagnosis 74
VI. General Prognosis 86
VII. General Therapeutics 87
Classification 114

DISEASES OF THE SKIN.

CLASS I.

HYPEREMIAS AND INFLAMMATIONS.

Hyperemia 115
Erythema 115
  Erythema Hyperemicum (*seu* Simplex) 116
  Erythema Traumaticum 116
  Erythema Caloricum 116
  Erythema Venenatum 116
  Erythema Paratrimma 116
  Erythema Nuchæ 116
  Erythema Ab Igne 116
  Erythema Pernio 117
  Erythema of Jacquet 118
  Erythema Symptomatic 118
  Erythema Læve 118
  Erythema Fugax 119
  Erythema Urticans 119
  Erythema Infectiosum 119
  Roseola Infantilis 119
  Erythema Scarlatiniforme 119
  Shedding of the Skin 122
  Erythema Intertrigo 123
  Erythema Multiforme 125
  Erythema Nodosum 135
  Erythema Perstans 137
Granuloma Annulare 138
  Erythema Elevatum Diutinum 141
Urticaria 141

Urticaria:

- Urticaria in Infants and Children 144
- Urticaria Pigmentosa 149
- Angioneurotic Edema 153

Prurigo 156
- Prurigo Nodularis 159

Dermatitis 161
- Dermatitis Traumatica 163
- Dermatitis Venenata 163
- Dermatitis Calorica 170
- Dermatitis Congelationis 172
- Dermatitis Medicamentosa 174

Dermatitis Exfoliativa 186
- Dermatitis Exfoliativa (Wilson) 189
- Pityriasis Rubra (Hebra) 193
- Dermatitis Exfoliativa Neonatorum (Ritter) 196
- Erythoderma Desquamativa 197
- Dermatitis Exfoliativa Epidemica (Savill) 198

Feigned Eruptions 200

Neurotic Excoriations 203

Radiodermatitis 205

Purpura Annularis Telangiectodes 208

A Peculiar Progressive Pigmentary Disease of the Skin 210

Eczema 212
- Eczema Erythematosum 214
- Eczema Papulosum 215
- Eczema Vesiculosum 215
- Eczema Pustulosum 217
- Eczema Rubrum 219
- Eczema Squamosum 220
- Eczema Fissum 220
- Eczéma Craquelé 221
- Eczema Verrucosum 221
- Eczema Nummularis 221
- Acute Eczema 222
- Chronic Eczema 222
- Topical Varieties of Eczema 223
  - Eczema of the Scalp 223
  - Eczema of the Face 223
  - Eczema of the Lips 224
  - Eczema of the Nostrils 224
  - Eczema of the Ears 225
  - Eczema of the Eyelids 225
  - Eczema of the Beard 225
  - Eczema of the Genital Region 225
  - Eczema of the Anus and Anal Regions 226
  - Eczema of the Nipple and Breast in Women 226
  - Eczema of the Umbilicus 226
  - Eczema Crurum 227
  - Eczema of the Hands and Feet 227
  - Eczema in Infants and Children 228

Infectious Eczematoid Dermatitis 247

Psoriasis 249

Psoriasis:

Parakeratosis Scutularis 269
Parapsoriasis 270
Parapsoriasis en Gouttes 270
Parapsoriasis Lichénoïde 272
Parapsoriasis en Plaques 272
Pityriasis Rosea 274
Lichen Ruber 279
Pityriasis Rubra Pilaris 280
Lichen Planus 287
Lichen Planus Hypertrophicus 292
Lichen Planus Obtusus 293
Lichen Planus Linearis 293
Lichen Ruber Moniliformis 293
Lichen Planus Annularis 293
Lichen Planus Erythematosus 293
Lichen Planopilaris 294
Lichen Planus of the Mucous Surfaces 294
Lichen Planus Sclerosus et Atrophicus (Hallopeau) 296
Lichen Planus Morphœicus (Crocker) 296
Lichenification 301
Lichen Nitidus 304
Impetigo 305
Impetigo Contagiosa 305
Ecthyma 311
Veldt Sore 314
Dermatitis Vegetans 314
Furunculus 315
Carbunculus 319
Multiple Abscesses of the Skin of Infants 322
Phlegmona Diffusa (Cellulitis) 323
Dissection-wounds and Animal Poisons 324
Gayle 324
Anthrax 325
Equinia 328
Erysipelas 331
Erysipeloid 336
Solid Edema of the Face 337
Dermatitis Repens 337
Acrodermatitis Perstans 339
Dysidrosis (Tilbury Fox) 340
Acrodermatitis Vesiculosa Tropica 343
Keratolysis Exfoliativa 344
Dermatitis Gangrenosa (Sphaceloderma) 344
Diabetic Gangrene 346
Dermatitis Gangrenosa Infantum 347
Symmetrical Gangrene of the Extremities 349
Diphtheria of the Skin 350
Herpes Simplex 351
Herpes Zoster 355
Dermatitis Herpetiformis 361
Herpes Gestationis 365
Impetigo Herpetiformis 368

Pemphigus 370
  Pemphigus Vulgaris 373
  Pemphigus Foliaceus 379
  Pemphigus Vegetans 383
Hydroa Vacciniforme 387
Epidermolysis Bullosa 390
Pellagra 393
Acrodynia 403
Exanthemata 405
  Scarlatina 405
  Rubeola 410
  Rötheln 413
  Variola 415
  Varicella 426
  Vaccinia 429
Rocky Mountain Spotted Fever 432

CLASS II.

HEMORRHAGES.

Purpura 435
  Purpura Simplex 436
  Purpura Rheumatica 436

CLASS III.

HYPERTROPHIES.

Keratosis Pilaris 439
  Ulerythema Ophryogenes 442
Lichen Pilaris *seu* Spinulosus (Crocker) 442
Keratosis Senilis (Senile Keratoma) 444
Keratosis Follicularis 446
Keratosis Follicularis Contagiosa 450
Keratodermia Palmaris et Plantaris 451
  Mal de Meleda 456
Keratodermia Blennorrhagica 456
Porokeratosis (Mibelli) 458
Angiokeratoma 460
Acanthosis Nigricans 463
Callositas 466
Clavus 467
Cornu Cutaneum 468
Verruca 470
  Verruca Acuminata 475
  Synovial Lesions of the Skin 477
Nevus Pigmentosus 478
  Giant Nevus 479
  Linear Nevus 479
Folliculitis Ulerythematosa Reticulata 486
Ichthyosis 487
  Ichthyosis Hystrix 489
  Ichthyosis Congenita 490

Ichthyosis:
  Ichthyosis Follicularis . . . 491
  Érythrodermie Congénitale Ichtyosiforme . . . 494
Scleroderma . . . 495
  Circumscribed Scleroderma . . . 498
  Morphea Guttata (White-spot Disease) . . . 499
Sclerema Neonatorum . . . 504
Edema Neonatorum . . . 505
Hereditary Edema of the Legs (Milroy's Disease) . . . 507
Elephantiasis . . . 507
Acromegaly . . . 512
Myxedema . . . 512
Dermatolysis . . . 515
Cutis Verticis Gyrata (Unna) . . . 517

CLASS IV.

ATROPHIES.

Atrophia Senilis . . . 519
Diffuse Idiopathic Atrophy of the Skin . . . 520
Atrophia Maculosa et Striata . . . 523
Atrophy of the Fatty Layer of the Skin . . . 525
Glossy Skin . . . 527
Perforating Ulcer of the Foot . . . 528
Multiple Benign Tumor-like New Growths of the Skin . . . 529
Morvan's Disease . . . 529
Kraurosis Vulvæ . . . 530
Ainhum . . . 532

CLASS V.

PIGMENT ANOMALIES.

Lentigo . . . 535
Chloasma . . . 537
  Chloasma Idiopathicum . . . 537
  Chloasma Symptomaticum . . . 537
  Chloasma Uterinum . . . 537
  Hemochromatosis . . . 538
  Mongolian Pigment Spots . . . 538
  Argyria . . . 541
  Tattooing . . . 542
  Blue Pigmentation . . . 542
Generalized Pigmentation . . . 543
Albinismus . . . 543
Vitiligo . . . 544

CLASS VI.

NEW GROWTHS.

Cicatrix . . . 549
Keloid . . . 551

Keloid:
- Cicatricial Keloid 553
- Acne Keloid 555

Fibroma 557
Paraffinoma 564
Chondrodermatitis Nodularis Chronica Helicis 568
Lipoma 569
- Adiposis Dolorosa 571

Neuroma 571
Myoma 573
- Dartoic Myoma 573
- Simple Myoma 573

Osteoma Cutis 574
Angioma 575
- Nevus Vasculosus 576
- Telangiectasis 581
- Nevus Anemicus 583

Angioma Serpiginosum 584
Granuloma Pyogenicum 587
Lymphangioma 589
- Circumscriptum 591

Molluscum Contagiosum 594
Xanthoma 598
- Xanthelasma 603
- Xanthoma Diabeticorum 605

Pseudo-xanthoma Elasticum 607
Colloid Degeneration of the Skin 608
Calcification of the Skin 609
Adenoma Sebaceum 610
Multiple Benign Cystic Epithelioma 614
Syringocystoma 617
Xeroderma Pigmentosum 619
Carcinoma of the Skin 625
- Carcinoma Lenticulare 625
- Carcinoma Tuberosum 627
- Melanotic or Pigmented Carcinoma 627
- Epithelioma 629
  - Superficial Variety 629
  - Rodent Ulcer 631
  - Morphea-like Epithelioma 632
  - Multiple Epitheliomata 632
  - Deep-seated Variety 633
  - Papillary Epithelioma 635
  - Epithelioma of the Lip 636

Paget's Disease 650
Precancerous Dermatoses 653
Endothelioma Cutis 654
- Endothelioma Capitus 654

Rhinoscleroma 655
Tuberculosis Cutis 657
- Lupus Vulgaris 658
- Tuberculosis Cutis Verrucosa 665
- Scrofuloderma 668
- Tuberculosis Cutis Orificialis 671

Erythema Induratum 683
Lichen Scrofulosorum 685
Tuberculides (Darier) 687
Acnitis 689
Folliclis 689
Acne Scrofulosorum 691
Acne Cachecticorum of Hebra 691
Lupus Erythematosus 693
Multiple Benign Sarcoid (Boeck) 706
Syphilis 709
Chancre 711
Syphilodermata 715
Syphiloderma Maculosum 719
Pigmentary Syphiloderm 720
Syphiloderma Papulosum 723
Small Acuminate Miliary Papular Syphiloderm 723
The Lenticular Papular Syphiloderm 725
Moist Papules 729
Palmar and Plantar Syphiloderm 732
Syphiloderma Vesiculosum 735
Syphiloderma Pustulosum 735
Small Acuminate Pustular Syphiloderm 736
Large Acuminate Pustular Syphiloderm 737
Flat Pustular Syphiloderm 739
Pustulo-ulcerative Syphiloderm 740
Syphiloderma Bullosum 741
Nodular Syphiloderm 741
Gummatous Syphiloderm 746
Reinfection and Superinfection in Syphilis 750
Congenital Syphilodermata 750
Lepra 781
Granuloma Fungoides 798
Leukemia Cutis 810
Pseudoleukemia Cutis 814
Sarcoma Cutis 815
Non-pigmented Sarcoma Cutis 815
Melanotic Sarcoma 819
Idiopathic Multiple Hemorrhagic Sarcoma (Idiopathic Multiple Pigment-sarcoma) (Kaposi) 822
Oriental Sore 827
Frambesia 830
Verruga Peruana 834
Gangosa 835
Ulcerating Granuloma of the Pudenda 838

CLASS VII.

NEUROSES.

Pruritus 841
General Pruritus 841
Senile Pruritus 842
Local Pruritus 842
omelalgia 851

Dermatalgia 853
  Causalgia 854
Meralgia Paresthetica 855
Trichotillomania 856

CLASS VIII.

PARASITIC AFFECTIONS.

DISEASES DUE TO VEGETABLE PARASITES.

Favus 857
Trichophytosis 865
  Trichophytosis Corporis 865
  Trichophytosis Capitis 874
  Trichophytosis Barbæ 878
  Histology of the Skin when Affected with the Fungus 891
  Tinea Imbricata 898
Tinea Versicolor 900
Erythrasma 905
Pinta 907
Mycetoma 909
Actinomycosis of the Skin 912
Blastomycosis 916
  Phialophora Verrucosa 927
Protozoan and Coccidoidal Infections 928
Sporotrichosis 928
Nocardiosis Cutis 932

DISEASES DUE TO ANIMAL PARASITES.

Pediculosis 932
  Pediculosis Capitis 934
  Pediculosis Corporis 936
  Pediculosis Pubis 937
Cimex Lectularius 939
Grain Itch 940
Copra Itch 943
Brown-tail Moth 943
Pulex Irritans 945
Ixodes 946
Scabies 947
Leptus 956
Pulex Penetrans 957
Dracontiasis 958
Cysticercus Cellulosæ Cutis 960
Echinococcus 961
Distoma Hepaticum 961
Uncinarial Dermatitis 962
Trypanosomiasis 963
Demodex Folliculorum 964
Myiasis Cutanea 965
Creeping Eruption 966
Craw-craw 967

CLASS IX.

DISEASES OF THE APPENDAGES.

DISEASES OF THE SWEAT-GLANDS.

- Hyperidrosis 969
- Anidrosis 973
- Bromidrosis 974
- Chromidrosis 976
- Uridrosis 978
- Sudamen 980
- Miliaria Rubra 981
- Miliary Fever 983
- Hydrocystoma 983
- Hydradenitis Suppurativa 986
- Granulosis Rubra Nasi 986

DISEASES OF THE SEBACEOUS GLANDS.

- Seborrhea 988
- Dermatitis Seborrheica 997
- Asteatosis 1008
- Milium 1009
  - Milium Congenitalé 1011
- Steatoma 1012
- Comedo 1014
- Acne 1019
  - Acne Rosacea 1030
- Acneform Eruptions 1036
  - Acne Artificialis 1036
  - Acne Keratosa 1036
  - Acne Urticata 1037
  - Acne Varioliformis 1037

DISEASES OF THE HAIR AND HAIR FOLLICLES.

- Hypertrichosis 1039
  - Trichiasis 1044
  - Districhiasis 1044
  - Plica Polonica 1044
  - Plica Neuropathica 1045
- Atrophia Pilorum Propria 1045
  - Fragilitas Crinium 1045
  - Trichorrhexis Nodosa 1046
  - Monilethrix 1049
- Trichonodosis 1050
- Piedra 1051
  - Chignon Fungus 1052
  - Piedra Nostras 1053
- Lepothrix 1053
- Trichostasis Spinulosa 1054
- Canities 1055
- Alopecia 1057
  - Alopecia Congenita 1058

Alopecia:

Alopecia Prematura 1060
Alopecia Pityrodes 1063
Alopecia Senilis 1064
Alopecia Areata 1069
Alopecia Cicatrisata 1080
Folliculitis Decalvans 1083
Dermatitis Papillaris Capillitii 1084
Coccogenous Sycosis 1087

Diseases of the Nails.

Onychauxis 1093
Atrophia Unguium 1097
Hapalonychia 1097
Koilonychia 1098
Onychoschizia 1098
Onychomadesis 1099
Leukonychia 1099
Onychorrexis 1100
Transverse Furrows 1100
Morbid Condition of the Nails Induced by Cutaneous Diseases 1103
Psoriasis 1103
Eczema 1104
Syphilis of the Nails 1105
Syphilis of the Nail-plate 1106
Syphilis of the Nail-bed and Matrix 1106
Onychomycosis 1109
Trichophytosis Unguium 1109
Onychomycosis Favosa 1111
Congenital Abnormalities and Diseases of the Nails 1112
Anonychia 1112
Polydactyly 1112
Syndactyly 1112
Onych-heteropia 1112

Diseases of the Mucous Membranes.

Leukokeratosis Buccalis 1113
Grooved Tongue 1116
Transitory Benign Plaques of the Tongue 1117
The Hairy Black Tongue 1119
Chronic Superficial Excoriations of the Tongue 1120
Cheilitis Glandularis 1121
Cheilitis Exfoliativa 1122
Fordyce's Disease 1123
Perlèche 1125
Aphthous Stomatitis 1126
Vincent's Disease 1127
Periadenitis Mucosa Necrotica Recurrens (Sutton) 1128
Atrophy of the Mucous Membranes of the Tongue and Mouth 1129

INTRODUCTION.

PRECEDING the description of the various diseases of the skin, several chapters are devoted to subjects of vital interest to the student of dermatology, and which, if mastered, will materially simplify the study of the individual diseases. Without a good working knowledge of the microscopic anatomy and of the physiology of the skin, an understanding of the pathological changes which occur in disease is rendered more difficult. In the description of cutaneous diseases in the text, the different symptoms are described in terms entirely different from those used ordinarily, and the student is therefore urged to master the chapter devoted to General Symptomatology. In the chapter on General Pathology, many terms are defined and pathological processes common to a large number of diseases are outlined which greatly facilitate the special study taken up later. In the chapter on General Diagnosis, special methods pertaining to cutaneous disease are detailed, such as the Wassermann test for syphilis, Noguchi's luetin test for syphilis, and the tuberculin, von Pirquet, and other tests for tuberculosis, in addition to much other information of value in the general and special examination of the patient. The chapters on General Etiology and Prognosis should also receive attention, and the one devoted to General Treatment is of value both for reference on special occasions, and also to obtain some knowledge of drugs, chemicals, and other means found useful in the management of cutaneous diseases. Among other methods described here will be found radiotherapy, which includes *x*-rays and radium; phototherapy, vaccine therapy, and the technique for treatment with liquid air and solidified carbon dioxid.

I. ANATOMY AND PHYSIOLOGY OF THE SKIN.

THE skin is essentially one of the vital organs of the body, performing functions necessary to health and life. It is closely associated with underlying structures, and by its situation is brought into intimate relation with the external world. The skin is a complex, elastic, and sensitive organ, varying greatly in different conditions of climate, age, sex, health, and race; and varying also in the characteristics exhibited in different localities upon the same individual. Thus, in color there is a wide range between the fair skin of the blonde and the black skin of the negro, between the rosy pink of the infant's palm and the dark-brown hue of the genital region of the aged. The skin varies also in pliability and thickness, being delicate and lax over the eyelids, the lips, and the prepuce, and much thicker and more firmly attached over the palms and soles.

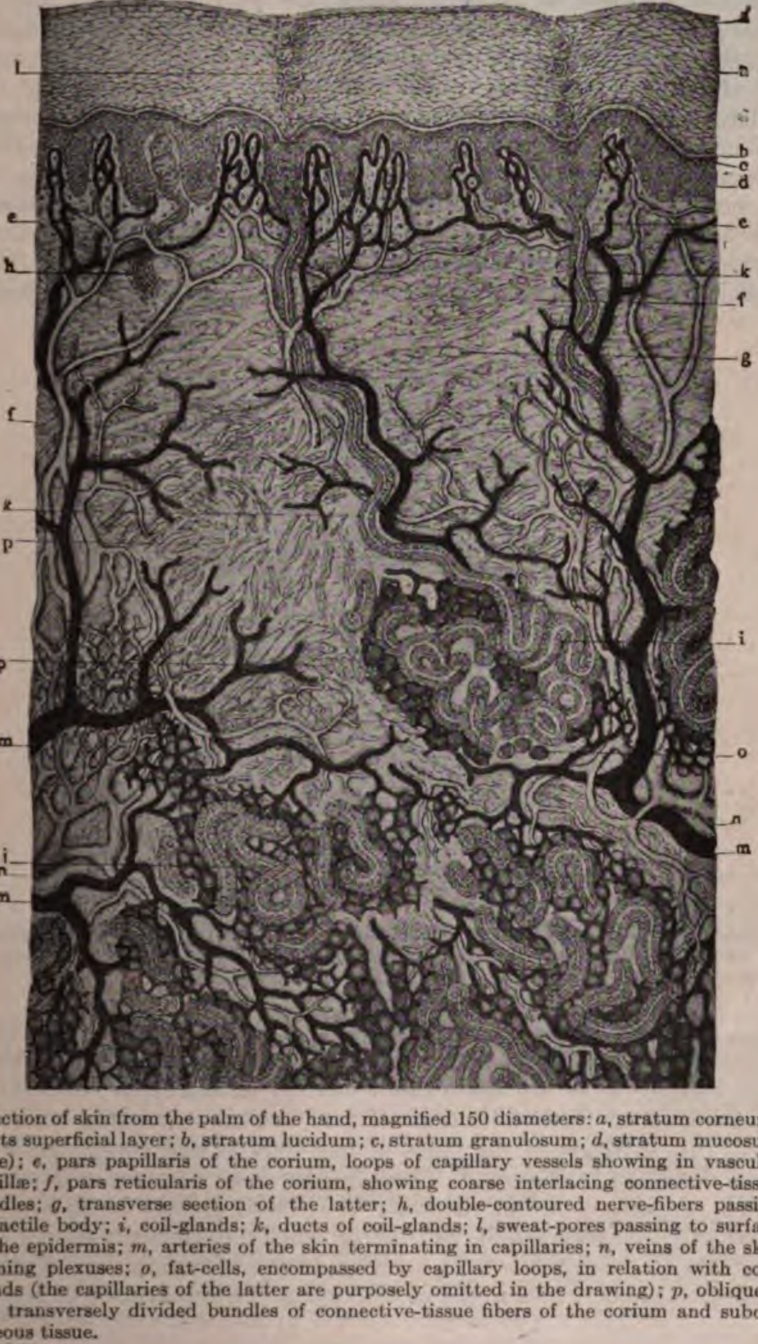
Ridges and Furrows.—Viewed externally, the skin is seen to be traversed by superficial and deep furrows, which vary in arrangement and size according to their situation. They are formed by the attachment of the skin to the deeper structures, by the movement to which one part is subjected, and by the arrangement of the fibrous structures of the corium. In some situations (the palms and soles), the fine furrows have a regular arrangement and run parallel with each other. The pattern thus outlined is constant in the individual, of which fact use is made in the identification of criminals. Between these fine furrows are ridges dotted with numerous depressions representing the openings of sweat-pores. The entire body is traversed by fine furrows, which form an irregularly diamond-shaped network.

Coarse furrows are found chiefly in situations where the skin is subjected to movement, such as about the joints, and they are due to the fixation of the skin to deeper structures by fibrous bundles. It is in such situations that fissures occur, when the normal pliability is lost through inflammatory thickening. The shape of many of the lesions of the skin is determined by the ridges and furrows above described.

The skin is divided, on account of anatomical differences, into three general layers: the epidermis or cuticle; corium or true skin; and subcutaneous tissue or hypoderm. In these various layers are found the coil- and sebaceous-glands, bloodvessels, lymphvessels, muscles, pigment, nerves, and the appendages of the skin—the hairs and nails. A clear conception is best had in a study of these component parts by progressing from within outward.

Subcutaneous Tissue (*stratum subcutaneum, panniculus adiposus*).—The subcutaneous tissue, or hypoderm, is differentiated from the corium between the third and fourth months of fetal life. It is a structure serving a mechanical purpose as a receptacle for fat, and for the support of vessels and nerves passing from the tissues beneath to the corium above. It contains, also, coil-glands, some of the hair-follicles more deeply situated than their fellows, and Pacinian corpuscles. There is no distinct boundary line between the upper limits of the subcutaneous tissue and the overlying corium, to which it projects columnar masses of fat, extending obliquely to the coil-glands and the hair-follicles above, often with lateral, horizontally disposed prolongations of similar shape. It is built up of loose connective-tissue bundles, which are attached to the aponeuroses, fasciæ, and the membranes lying beneath.

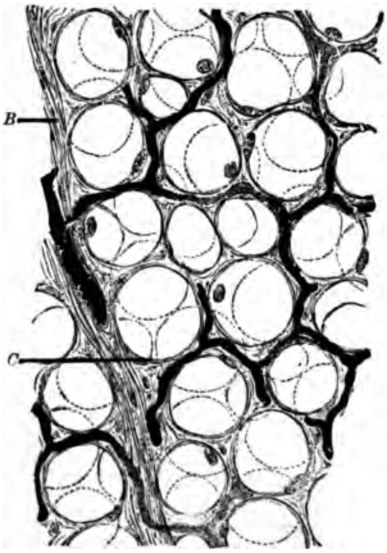
FIG. 1



Section of skin from the palm of the hand, magnified 150 diameters: *a*, stratum corneum; *a'*, its superficial layer; *b*, stratum lucidum; *c*, stratum granulosum; *d*, stratum mucosum (rete); *e*, pars papillaris of the corium, loops of capillary vessels showing in vascular papillæ; *f*, pars reticularis of the corium, showing coarse interlacing connective-tissue bundles; *g*, transverse section of the latter; *h*, double-contoured nerve-fibers passing to tactile body; *i*, coil-glands; *k*, ducts of coil-glands; *l*, sweat-pores passing to surface of the epidermis; *m*, arteries of the skin terminating in capillaries; *n*, veins of the skin forming plexuses; *o*, fat-cells, encompassed by capillary loops, in relation with coil-glands (the capillaries of the latter are purposely omitted in the drawing); *p*, obliquely and transversely divided bundles of connective-tissue fibers of the corium and subcutaneous tissue.

The subcutaneous tissue is attached firmly to the skin over the extensor surfaces of the articulations, the palms and soles, and the groins by short, coarse bundles, between which are single or multilocular spaces lined with endothelia secreting a mucoid fluid. Some of these are congenital; others result from evolution later in life. They are most frequent and largest where necessary movements occur, as where the skin is stretched over a bone or tendon. These spaces constitute the bursæ mucosæ. In the eyelids, the penis, the scrotum, and the auricle of the ear, the attachment to the skin is by loose, delicate connective tissue, containing no fat-globules. In other situations, the fibrous tracts are arranged obliquely. They admit by their extension of various degrees of pliability, and inclose rhomboidal spaces containing more or less numerous fat-globules. These spaces are divided into lobes by fibrous tissue called trabeculæ, and the lobes are again subdivided by fibrous septa into lobules. In addition to these collections of fat, columns extending obliquely from this situation to the bases of hair-follicles and coil-glands have been described by Warren.[1] They are known as *columnæ adiposæ*.

Fig. 2



Subcutaneous fat-tissue, the fat having been extracted with turpentine: *B*, bundles of fibrous connective tissue, carrying injected blood-vessels; *C*, capsules of fat-globules, with oblong nuclei. Magnified 500 diameters. (After Heitzmann.)

The deposit of fat in the body is reduced greatly in all diseases productive of emaciation, but never wholly disappears during life. In cases of obesity, fat is deposited in excess of normal limits, and it may then be concerned in the production or the aggravation of disease.

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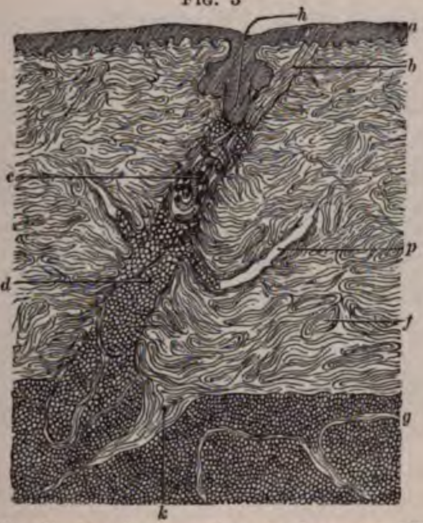
[1] Boston Med. and Surg. Jour., April, 1877.

It is due largely to the greater or lesser volume of the panniculus adiposus that the natural outlines of the body are made to the eye graceful and attractive or the reverse.

The Corium (*derma, cutis, cutis vera, or true skin*).—The corium is a mesoblastic structure, made up largely of connective tissue and cellular elements. It is rich in bloodvessels and capillaries, especially in the papillary layer, and contains many nerves, nerve-endings, and terminal nerve organs. It also contains lymphatics, small muscle-fibers, hairs, sweat glands, and sebaceous glands.

The fibrous elements are of two varieties: collagen and elastin. The collagen occurs as bundles of fibers held together by a semifluid, interfibrillary substance. The fibers are about $\frac{1}{3200}$ of an inch (0.79μ) in breadth, and according to Clarkson (quoted from MacLeod) are made up of fibrils that are approximately $\frac{1}{50000}$ to $\frac{1}{20000}$ of an inch (0.05μ to 0.12μ) thick. The collagenous bundles are only slightly extensible, but as their arrangement presents a wavy appearance, on longitudinal section, they admit of stretching of the skin. The individual fibers do not branch, but such an appearance is simulated by the joining of parts of different bundles.

FIG. 3

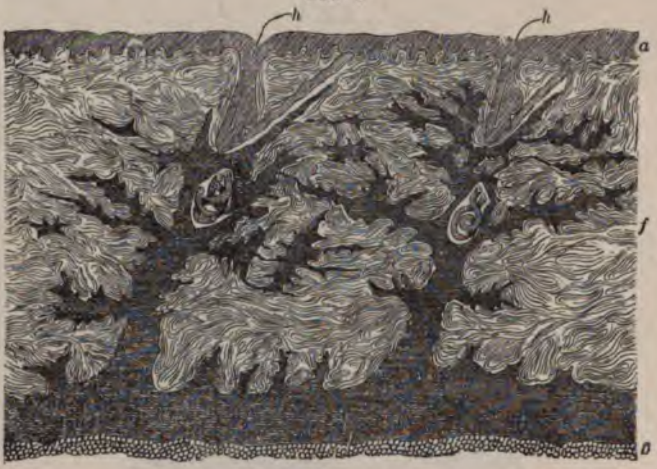


Vertical section of the skin showing: *a*, epidermis; *b*, erector pili muscle; *d*, columnæ adiposæ; *c*, coil-gland suspended in the columnæ adiposæ; *h*, sebaceous gland; *p*, horizontal prolongations of the column; *f*, fibrous bundles of the corium; *g*, panniculus adiposus; *k*, band of fibrous tissue extending into the panniculus adiposus. (After Warren.)

The elastic fibers (elastin) occupy the entire corium and extend throughout the subcutaneous tissue. These fibers, by anastomatic branches, form a network which surrounds the collagenous bundles and all the other elements of this region, acting as a supporting framework. These fibers vary in thickness from imperceptible fineness up to 11μ in breadth (Stöhr) and have little elasticity. They are the first to rupture when the skin is stretched, as is demonstrated in the *lineæ albicantes*, and their chief function appears to be that of support.

The cellular elements of the corium consist of connective-tissue corpuscles, vacuolated cells (Schäfer), mast-cells, and migratory blood-cells. (Description of these cells, p. 71.) The fibers and bundles of connective tissue are coarsest toward the subcutaneous tissue, and finest toward the outermost portion, which comes in contact with the epidermis above They form the mesoblastic portion of the hair-follicle, the capsules around the coil-glands, and the layers which surround their ducts.

FIG. 4



Vertical section of skin after injection (from beneath) of areolar tissue with Berlin blue: *a*, epidermis; *f*, corium; *g*, panniculus adiposus; *h*, sebaceous gland. (After Warren.)

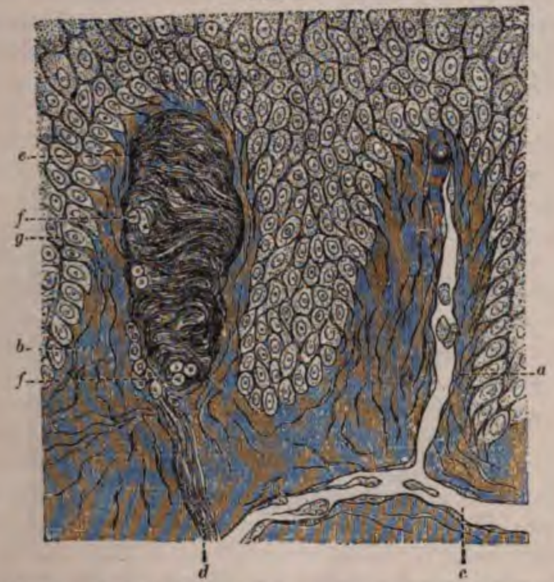
Corresponding with their anatomical structure, the upper and lower portions of the corium are called respectively the "papillary layer" and the "reticular layer." There is no sharp dividing line between these layers, the pars reticularis passing gradually into the pars papillaris above and into the subcutaneous tissue below.

Pars Reticularis.—The reticular layer of the corium is made up of interlacing connective-tissue bundles, which are coarser below and separated by larger spaces. The bundles become finer and the spaces smaller toward the surface.

Pars Papillaris.—The papillary layer lies in contact with the rete above, and is connected below with the deeper reticular portion of the corium. Between the rete and the papillæ of the corium, a hyalin substance is interposed, which is supposed to be identical with the cement substance surrounding and separating the fibrillæ of the corium. The basal membrane, once thought to be stretched between the rete mucosum of the epidermis and the papillary layer of the corium, cannot be demonstrated to exist. Viewed obliquely with an amplification of about 300 diameters, it will be seen that long, slender filaments from the prickle-cells of the mucous layer of the epidermis encircle in a spiral direction both nervous and vascular papillæ. At the apices of the latter, these threads completely surround the connective-tissue fibers.

The name of this portion of the corium is intended to describe its chief characteristic, the existence of numerous digital prolongations or nipple-like prominences of the corium, made up of delicate connective-tissue fibers, which do not interlace and which are abundantly provided with nuclei. The papillæ spring each from a single, or several from a common, ovoid base. Their bulbous, conical, or blunt apices reach into the rete, which also dips down between them in prolongations termed "rete-pegs." The papillæ vary in size in different parts of the body, and also in their disposition and shape, being in places arranged in linear series, and in others in concentric whorls, with definite centers, thus producing corresponding furrows, visible to the naked eye as markings upon the outer surface of the epidermis. The largest are found on the palms and soles and over the inner faces of the digits. It has been estimated that 100 are developed within each square millimeter of the body surface.

Fig. 5



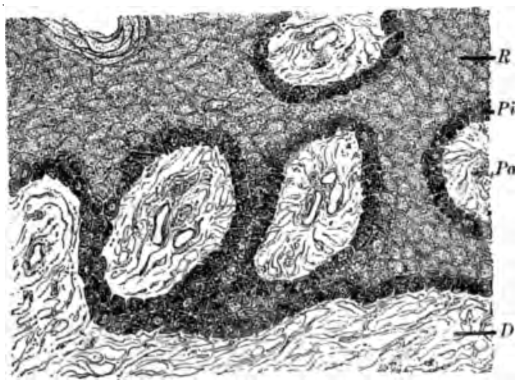
Vascular and nervous papillæ: *a*, vessel; *b*, nervous papilla; *c*, vessel; *d*, nerve-fiber; *e*, corpusculum tactus; *f*, transversely divided nervous filaments; *g*, epithelia of rete. (After Biesiadecki.)

In horizontal sections of the skin, the papillæ, being transversely divided, appear as circular or ovoid areas, in which can be recognized a centrally and transversely or obliquely divided capillary loop. Between these areas is seen the interpapillary reticulum of the mucous layer.

The growth of the rete downward and of the corium upward results in mutual effects of pressure and counter-pressure, the equilibrium of which is constantly adjusted by the mechanical and vital necessities of such union.

When the papillæ are completely exposed, after removal of the overlying cement substance and of the epidermis above, their exterior surface is seen to be uniformly and delicately marked with series after series of alternating furrows and ridges, more or less concentrically disposed. Into the grooves are admitted corresponding dentations, that can be recognized on the under surface of the layer of epithelial cells next to the corium. They may, however, be the furrows left after separation of the long prickles wrapped about the papillæ and traceable to the mucous layer.

FIG. 6



Scalp of a negro—horizontal section: *R*, rete mucosum; *Pi*, row of columnar epithelia (cut obliquely) supplied with dark-brown pigment-granules; *Pa*, papilla (cut transversely); *D*, derma. Magnified 500 diameters. (After Heitzmann.)

Two varieties of papillæ are distinguished, the vascular and the nervous. The former contains the terminal loops of a minute artery and vein, and the latter the terminations of medullated nerve-fibers.

The greater number of the papillæ are of the vascular variety, being traversed by a vertically disposed loop of vessels, consisting of an arterial and a venous capillary. The office of the vascular loop is evidently not merely to supply nutriment for the epidermis above, but also to provide for the cooling of the blood when brought in large quantities to the surface of the body. Occasionally, two or more of such loops can be recognized in a single papilla.

The nervous papillæ contain the tactile corpuscles, which serve an important purpose in providing for the sensibility of the integument.

Ultimate terminations of nerves can be seen in the papillæ largely occupied with the corpuscles of touch.

Lines of Cleavage.—Puncture of the skin with a rounded instrument leaves an irregularly longitudinal slit. This phenomenon occurs as the result of the arrangement of the connective-tissue bundles and fibers of the corium. Dupuytren[1] studied this in the skin of the palm, and Langer and Heitzmann[2] later mapped out the special directions over the entire body in which these lines occur.

The Epidermis (*scarf skin or cuticle*).—The epidermis is the most external of the several layers of the skin, being in close contact on one side with the corium and exposed on the other to the atmosphere by which it is surrounded. The latter surface is therefore relatively drier, while the former is constantly moistened by fluids from the vessels which ramify beneath it. It is of epiblastic origin and made up of superimposed strata of epithelial cells, and varies in aspect and thickness according to its anatomical situation and the age of the subject.

The epidermis is composed of the following principal layers, named in order from within outward: the stratum mucosum, the stratum granulosum, the stratum lucidum, and the stratum corneum. All the cells composing these various layers are derived from the basal layer of the rete. Besides these, Ranvier and others recognize a stratum germinativum, a stratum filamentosum, a stratum intermedium, and a stratum disjunctum.

Rete Mucosum (*mucous layer, prickle layer, stratum mucosum, rete malpighii or malpighianum*).—This is the deepest of the epidermal layers, and rests upon the corium below. It is generally designated as "the rete." The corium is intimately united with it by a series of interdigitations, which are commonly described as prolongations of the derma into the substance of the rete; but it is equally true that the rete sends down prolongations (the "rete-pegs") into the derma. The two, in the need of an intimate union to resist friction and to insure vascular supply, are thus closely locked together.

The stratum mucosum is built up of nucleated epithelial cells, chiefly of polyhedral shape. These cells are masses of granular protoplasm, living matter, which by their relation to one another form a protoplasmic network enveloping the entire surface of the body and lining all channels and cavities in direct or indirect communication with the surface. There are lymph spaces between the epithelia, from which the nutritive fluids are conveyed to the cells, and the individual cells are united by delicate protoplasmic threads, called prickles, spines or thorns. The epithelia are not provided with either bloodvessels or lymphvessels, but are supplied with a large number of nerves, which, in the shape of very minute beaded fibers, traverse the intercellular substance, and which are in direct communication with the reticulum of living matter within the protoplasmic bodies themselves.

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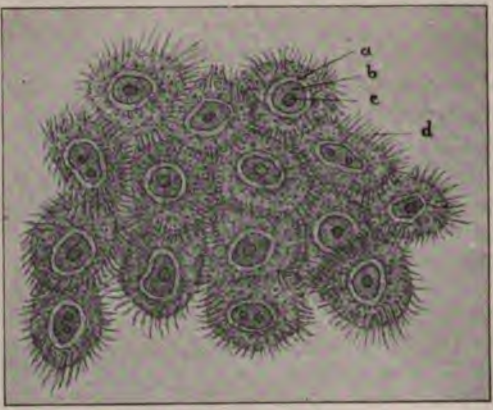
[1] Ueber die Verletzungen durch Kriegswaffen aus der Franz, 1836, p. 27.

[2] Archiv, 1890, xxii, p. 3.

Next to the corium is a layer (*basal layer, stratum germinativum*) of cells, columnar in form, often largely provided with pigment, and arranged with their long axes nearly at right angles to the plane of that portion of the corium upon which they are superimposed. The cells of this layer are dividing constantly by mitosis, the daughter-cells pushing outward to form the succeeding layers. The entire epidermis is thus derived from this single (occasionally double) row of columnar cells. More externally, the cells are rounded or cuboidal in form, with large, distinct nuclei. They are not arranged in definite strata except in the outermost layers, where the cells become more flattened and elongated (*stratum filamentosum*).

FIG. 7



Prickle-cells from a condyloma (magnified about 625 diameters): *a*, cavity of cell nucleus; *b*, nucleus; *c*, nucleolus; *d*, prickles—these are greatly developed on the protoplasm of the cells. The dots on the surface of the protoplasmic mass represent the appearance of the prickles when directed toward the eye of the observer. Some of the protoplasmic threads are seen passing from one cell to another.

Langahan's Cells.—These are elongated, irregularly stellate, non-nucleated bodies, found chiefly in the deeper parts of the rete. They have been looked upon as pigment cells devoid of pigment, as wandering cells, lymphoid cells, and as colorless tissue-corpuscles.

Stratum Granulosum (*granular layer*).—The stratum granulosum of the epidermis lies immediately above the stratum mucosum, and is built up of three or four rows of horizontally disposed cells, united to one another by short, broad threads, and containing granules. Between the cells, the spaces are so contracted that nutritive fluids cannot easily filter outward, and the nuclei of the cells are usually shrunken. These cells have been classified and studied by Ranvier, Kölliker, Waldeyer, and others. According to these observers, the roundish granules which give this layer of the epidermis its name and peculiar appearance consist of keratohyalin, a substance which plays a part in the process of cornification. These granules first appear in the neighborhood of the nuclei of some of the large prickle cells in the rete, but they are best studied in the granular layer, the cells of which are often completely filled with them. Keratohyalin is a solid or semisolid substance, being chemically closely allied to hyalin. According to Unna, the color of the skin of the white races depends upon this layer alone.

Stratum Lucidum (*septum lucidum*) of Oehl lies immediately above the stratum granulosum, and appears under the microscope as a delicate, brightly colored line consisting of two or three rows of transversely disposed, glistening epithelia, differing in translucency from those situated on either side. The stratum lucidum thus marks with tolerable distinctness the boundary lines of the rows of cells above and below it. Its cells no longer contain the granules of keratohyalin, conspicuous in the stratum granulosum below, but in their place have acquired an oily-looking substance termed *eleidin*. Eleidin, though oily-looking, is not a fatty substance. It differs from keratohyalin physically and chemically, but MacLeod suggests that it may be a derivative of keratohyalin.[1]

The cells of the uppermost layer of the stratum lucidum have been termed the *stratum intermedium* by Ranvier, for the reason that they take a reddish stain after treatment with picrocarmine. In this layer the process of keratinization is first detected.

Stratum Corneum (*horny layer*) of the epidermis is its outermost and widest layer. In its lower portion, the cells still retain to a degree the shape of the cells below, indicating their relationship. Nuclei exist in this layer only as shriveled and inconspicuous relics. Occasionally, on the edges, rudiments of prickle cells may still be recognized. More externally, the dried, lifeless, horn-like plates of which this layer is composed become mere cornified shells, generally lying in horizontal strata and becoming more curled and wrinkled as the surface of the skin is reached, often being imbricated, but preserving the polygonal outlines of epithelia relieved of the forces of pressure and counter-pressure exerted in the deeper part of the epidermis. No pigment is present in this layer, except in the colored races. The cells contain a fatty material, which adds to the suppleness of the skin and prevents undue evaporation, and also absorption from the outside. The hard, dry character of the cells is due to a resistant substance termed *keratin*, which has replaced the keratohyalin of the stratum granulosum and the eleidin of the stratum lucidum. Keratin is insoluble in 50 per cent. dilution of mineral acids, and resists digestion in a solution of pepsin containing weak hydrochloric acid, but is soluble in weak alkaline solution.

After digestion with pepsin and trypsin, the horny cells may be seen to be connected by more or less persistent threads, forming a large-meshed reticulum, with strands formed from a double row of cornified filaments united by short, horny bridges.

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[1] *Cf.* MacLeod's Pathology for complete discussion and description of keratohyalin and eleidin, p. 61.

Stratum Disjunctum.—This is the most superficial layer of the stratum corneum, and only differentiates by staining methods.

Spiral Fibers.—Herxheimer's[1] spiral fibers are found chiefly between the cells of the rete and the basal layer of the epidermis. They are most abundant normally in the lower part of the rete, and become increased in number in inflammatory conditions. They lie for the most part parallel with the long axes of the rete-cells. They sometimes are found between the cells of the inner root-sheath of the hair-follicle. Opinion differs as to their nature. Jadassohn, Ehrmann,[2] MacLeod[3] and others believe them to be spirals of fibrin. This seems probable, since they are increased in number when an inflammatory reaction is present, and since they occupy the lymph spaces between the cells and in size correspond with these spaces. They have in the past been regarded as elastic fibers protruding from the corium below; as parts of a canal system for the conveyance of nutriment to the cells of the epidermis, etc.

Cornification.—The process by which the epithelial cell from the basal layer of the rete becomes transformed into the hard, resistant cell of the stratum corneum has been studied at length, and the part played by the keratohyalin of the granular layer and eleidin of the stratum lucidum in the formation of keratin has caused much controversy. While, as a rule, when cornification is perfect both keratohyalin and eleidin are present normally, and when these two are absent or imperfectly formed cornification is incomplete, yet cornification may occur without the intercurrence of these substances. MacLeod considers keratohyalin as "a separation product of the protoplasm of the cell which appears as the vitality of the cell is diminishing; eleidin, a further product of the same substance; and the ultimate product of both is probably the fatty or waxy substance which is present in the horn cells." "The intercellular bridges or prickles would, according to this hypothesis, become hardened into keratin by an inherent power of their own, in much the same way as the fibro-vascular system of a leaf at the fall of the year becomes hardened into a brittle leaf skeleton."

Bloodvessels.—The skin is richly supplied with bloodvessels, particularly on its flexor surfaces. It is customary to describe the bloodvessels in the skin as consisting of an upper and a lower plexus or ridge-net. A more simple explanation of the vascular supply than that formerly made has been given by MacLeod.[4] Practically the entire supply comes from a system of bloodvessels situated in the subcutaneous tissue. There is in addition a horizontal plexus located in the papillary layer of the corium, the latter being formed by a series of arteries and capillaries running more or less vertically from the subcutaneous plexus and branching horizontally in this region. Taken as a whole, the larger arteries divide and subdivide, eventually becoming capillaries, which join venous capillaries and return by anastomosis to the subcutaneous veins by the junction of the various tributaries, thus following a return course similar to that taken by the arteries. In the subcutaneous tissue the course of the vessels lies between the lobes and lobules, and after dividing into small branches these surround and supply the fat-cells. A plexus of vessels, chiefly derived from the subcutaneous group, is also found about the coil glands, the remainder of the sweat-apparatus being supplied largely from vessels in the corium. The sebaceous glands, hair follicles, and arrectores pilorum are also freely supplied with plexuses of vessels. The major portion of the vessels in the subcutaneous tissue have an endothelial lining and a muscular and adventitious coat, while those of the corium are supplied with an endothelial lining only.

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[1] Arch., 1889, p. 645

[2] Arch., 1892, Erganzungsheft, i, p. 307; and Monatshefte, 1897, xxiv, p. 549.

[3] MacLeod's Histopathology of the Skin, 1903, p. 59.

[4] Ibid.

From the horizontal plexus in the papillary layer loops are sent to each papilla. These are either single or compound, the returning venous capillary being about four times the diameter of the arterial capillary. Special venous sinuses lined with endothelium are found in the tips of the fingers and toes, the lobes of the ears, the nostrils, the lips, and the nail-beds. In inflammatory and other pathological conditions of the skin, most marked perivascular infiltration occurs in the regions about the glands and hair follicles and in the papillary layer, thus indicating the distribution of the greatest number of vessels.

Nerves.—The skin, in view of the number and mode of distribution of its nervous elements, may be regarded as a vast area of sensitive nerve-terminals. Non-medullated and medullated nerve-fibers, each in places being substituted for the other, are supplied to the skin from horizontally disposed bundles of nerve-twigs in the subcutaneous tissue. These fibers traverse the corium in connection with the bloodvessels, and become finer as they ascend, until they form a subepithelial plexus below the epidermis.

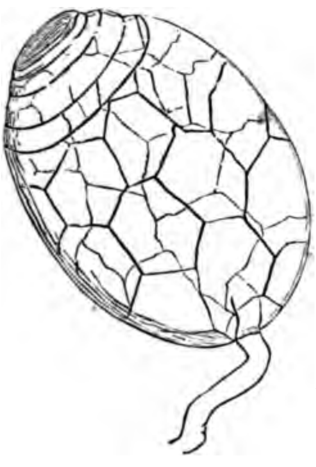
Non-Medullated Fibers are exceedingly delicate fibers, penetrating in great abundance to the epidermis between the epithelia, and are not to be confounded with the migratory cells found in this situation. Here, traversing the intercellular substance by the side of the juice-spaces, these fibers either terminate between the prickle-cells as ultimate bulbous terminations of finely beaded fibrillæ, or penetrate the epithelia themselves in pairs. Each prickle-cell is supplied with a pair of these beaded filaments, which may be either applied to the nucleus of the cell or be seen to encircle the nucleus more or less completely. Above the stratum granulosum these nervous threads cannot be recognized.

Similar nerve-filaments are supplied to the sheaths of the hairs and the ducts of the coil glands. It is by means of these numerous and delicate fibers that the perception of sensation in the skin is accomplished.

Motor filaments are also distributed to the sheaths of the blood-vessels (vasomotor nerves), in which they are finally lost. Other motor filaments supply the muscles, and trophic nerves are distributed to all the secreting organs of the skin and to all its protoplasmic formations.

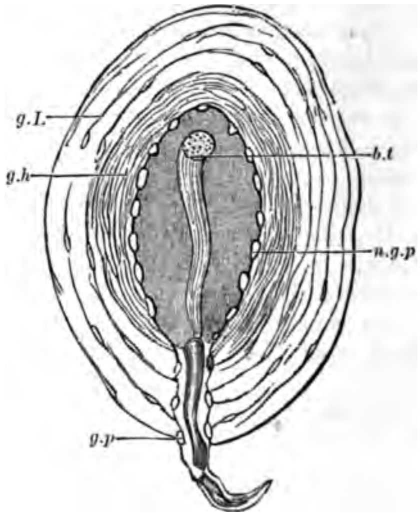
Medullated Nerve-fibers of the skin in one or several loops pass upward into the papillæ, and then turn backward to the subpapillary region. Some of these fibers, after such reversion, again ascend to an adjacent papilla; others are supplied to the Pacinian and tactile corpuscles.

Fig. 8



Pacinian body, after silver staining, showing superimposed endothelial layers. (After Renault.)

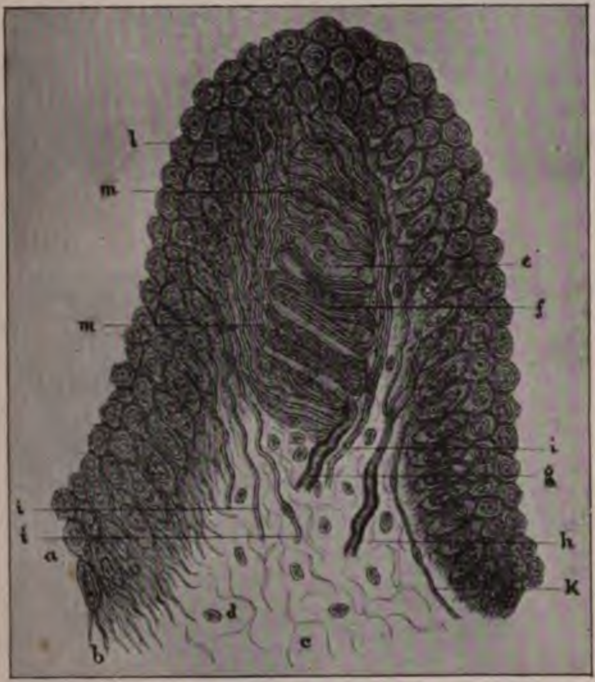
Fig. 9.



Section of Pacinian body from a duck's bill: *g. L*, lamellar envelope; *g.h*, hyaline zone of the lamellar envelope; *b.t*, terminal bulb of the nerve; *g.p*, *n.g.p*, layer investing the cavity of the body. (After Renault.)

Pacinian Corpuscles (named from the anatomist Pacini), also called *Corpuscles of Vater*, exist subcutaneously only upon nerves intended for cutaneous supply. They are ovoid bodies, two or more millimeters in diameter. Each corpuscle consists of a series of concentric, nucleated, vascular capsules, arranged after the manner of the capsules of the onion, more closely united at the periphery than at the centre, and surrounding a protoplasmic core. The medullated nerve to which the body is attached gradually loses its myeline envelope, and terminates in the center of this core, after traversing the greater part of its axis, in one or several minutely club-shaped filaments. The myeline sheath is lost in the tissue of the concentric capsules. The nerve may, after supplying one capsule, penetrate a second or even a third. In such cases the nerve regains its sheath as it issues from the corpuscle at its opposite pole. Robinson believes that the nerve forms a plexus or loop within the corpuscle, and escapes from it at one of its poles.

FIG. 10

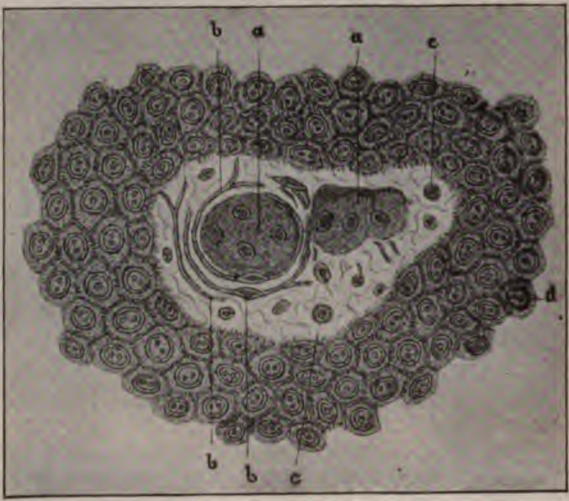


Section of a papilla still covered by a portion of the stratum mucosum and containing a tactile body (from the skin of a finger). The corpuscle of Meissner is seen to consist of minute lobules, made up of a homogeneous protoplasm, with numerous oval nuclei and nervous fibrillæ wound in a spiral direction about the mass of the corpuscle. The extension of the fibrillæ to the mucous layer is shown. The courses of the nerve-filaments are demonstrated to be: (1) the axis-cylinders of one or two double-contoured nerve-fibers, splitting into their original fibrillæ on arriving at the corpuscle, winding about the latter in characteristic spirals, and passing to the palisade-layer of the prickle-cells of the rete, between which, on account of the long prickles of the latter and the general resemblance of the two in thickness and contour, it is difficult to trace them further; (2) filaments from another double-contoured nerve-fiber (*h*) pass directly to the inferior layer of cells in the rete without establishing relations with the tactile body; (3) fibrillæ derived from the network of nervous fibrillæ in the pars papillaris of the corium (*K*), also passing more or less directly to the stratum mucosum; *a*, cells of the rete; *b*, prickles of the latter; *c*, body of papilla; *d*, nuclei of connective tissue forming papilla; *e*, protoplasmic part of the tactile body with its nuclei; *f*, fibrillæ of the corpuscle; *g*, double-contoured nerve-fibers directly supplying the rete; *k*, nervous fibrillæ derived from the network in the pars papillaris; *l*, nervous fibrillæ entering the epidermis between the rete-cells leaving the corpusculum tactus at *m*.

The precise function of the Pacinian corpuscle is unknown. Its connection with the tactile sense is suggested by its location, since these bodies are most numerous in the subcutaneous tissues of the nipple, the penis, the digits, and in parts similarly sensitive. These corpuscles bear an analogy to the organ of vision, each body having a capsular character; each being provided with a special nerve-filament, which enters the corpuscle at one pole; each also receiving its impressions at the extremity of the capsule opposite that at which it receives its nervous supply.

According to Krause, the Pacinian corpuscles aid in the appreciation of impressions produced by pressure and traction. Whether specially concerned in distinguishing sensations of heat, cold, moisture, pressure, traction, or weight, it is evident that they contribute but little, if at all, to the perception of ordinary impressions upon the skin, and they are not known to play any part in cutaneous diseases.

FIG. 11



Transverse section of nervous papilla surrounded by cells of the stratum mucosum: *a*, protoplasmic lobules of the corpusculum tactus; *b*, nervous fibrillæ spirally wound about the latter; *c*, transverse section of double-contoured nerve-fibers; *d*, cavity of nucleus (out of focus).

Tactile Corpuscles (*corpuscles of Meissner or of Wagner*) are ovoid bodies found in about one in four of the papillæ in the pars papillaris of the corium. Each corpuscle is composed of from one to three capsules. Minute lobules of a homogeneous protoplasm with oval nuclei are found in each. These corpuscles receive medullated nerve-fibers, and are made up of closely compressed, flat, connective-tissue fibers with minute nuclei, which are so packed together as to form a spindle-shaped mass occupying the greater part of the papilla in which each corpuscle is found and surrounded by a somewhat denser connective-tissue capsule. The myeline sheath of the nerve-fibers is lost in the fibrous tissue of the corpuscle. Externally viewed they seem to be transversely striated.

The axis-cylinder of the nerve-filament distributed to each corpuscle divides into numerous delicate nerve-threads, which in part encircle the corpuscles and also penetrate within. Each corpuscle is provided with an afferent and an efferent nerve, the former approaching the corpuscle from the subpapillary region and entering at or near its base. Occasionally the afferent fiber is furnished by an adjacent papilla. As the filament that enters the corpuscle frequently divides, two or more efferent fibers may then escape from it. Afferent fibers reach the rete above after encircling the tactile corpuscles; others, side by side, arrive at the rete without coming into contact with the former.

The discovery of nerve-filaments in and among the epithelia of the epidermis in such abundance as to provide fully for tactile sensation in the skin leaves the exact function of these corpuscles in partial obscurity. There can be little doubt, however, as to their association with the perception of certain qualities of foreign bodies with which the skin may be brought into contact.

Touch-Cells.—Merkel's touch-cells are oval, nucleated bodies found in the lower animals, but also in man. They are supposed to be connected with the ultimate nerve-fibers. They resemble cells in a mitotic state, and are found in the upper parts of the corium as well as the epidermis, and in regions in which the tactile corpuscles are few, as over the abdominal surface.

Corpuscles of Krause (*bulb-corpuscles: kolbenkörperchen*) are rounded or oval-shaped bodies formed of a connective-tissue envelope and a non-nucleated bulb, to which some delicate nerve-fibers penetrate. These bodies are found chiefly along the borders of the lips, over the glans penis, the clitoris, and the tongue.

Lymphatic Vessels.—The skin in all its parts is provided with a closed system of lymphatic channels, designed to subserve the necessities of the important processes of absorption, and is traversed by lymph, the currents of which are continuously directed to the large vessels of the structures beneath the skin. These channels include: first, juice-spaces, provided or not with independent walls, usually without, and not freely communicating with the endothelium-lined vessels; second, lymphatic vessels proper. These conduits do not connect with bloodvessels.

The juice-spaces, or lymph-spaces, separate the epithelial bodies which make up the stratum mucosum of the epidermis, and they also extend between the protoplasmic threads, or prickles, that unite them. Such conduits may be regarded either as delicate excavations in the cement-substance between the epithelia, or as irregular channels in a soft, viscid, albuminoid, and readily coagulable substance between the protoplasmic threads. At times this intercellular substance seems capable of obstructing the conduits by which it is tunnelled. These juice-spaces exist in the papillæ of the corium, and encircle the several glands, hair-follicles, and nail-beds of the skin. They also sheathe the connective-tissue fibrillæ of the corium and surround the fat-cells. According to Darier, the derma is a "true lymphatic sponge."

The lymphatic vessels are relatively few, but they form a continuous meshwork, with transversely and vertically disposed branches supplying all parts of the skin below the epidermis. The juice-spaces communicate with these vessels in the papillary portion of the corium through minute orifices in the vascular walls, the vessels themselves being here represented by blind terminal loops. As these vessels pass to the deeper portions of the corium and below it they increase in size. The current of the lymph flows from the papillary apices to all parts of the rete, like the currents in the delta of a river, a reflux occurring at the lower limit of the interpapillary depressions of the rete downward, possibly through the sweat-pores which traverse the epidermis at these points. Thence the current flows freely downward to the lymphatic vessels in the corium, but the stream from the juice-spaces about the coil glands and fat-tissue is retarded by reason of a more restricted communication with the lymphatic vessels below. In consequence of the retardation due to this anatomical peculiarity the formation of fat by filtration is facilitated.

Muscles.—Striated Muscular Fibers extend from the subcutaneous tissue into the derma; in the case of man they are found chiefly upon the face and neck, where they are the analogues of more powerful skin-moving muscles possessed by several of the lower animals. Some, as those in the region of the face, serve to give expression to mental emotion by the production of facial movements.

Non-Striated Muscular Fibers exist either as minute oblique fasciculi in connection with the glands and follicles of the skin; as annular bands, such as those which surround the nipple; or as radiating and more or less parallel rods, such as antagonize the orbicularis in the eyelids.

Arrectores (*Erectores*) *Pilorum.*—These muscles are found usually in connection with the hair-follicles. They originate by minute multiple fasciculi from the papillary portion of the corium, and are inserted at several points into the outer layer of several adjacent hair-follicles, just above the plane of the apex of the hair-papillæ. Their general direction is oblique, and their muscle-bundles are embraced and traversed by elastic fibers, which form a dense network about them. Elastic threads also connect them intimately with the connective-tissue bundles of the corium, and serve as tendons at either extremity of each muscular fasciculus.

The muscles, by virtue of their oblique direction and mode of attachment, include in the angle subtended by their muscular fibers the sebaceous glands connected with the hair-follicles. It follows, therefore, that by their contraction they aid in the expulsion of the sebaceous secretion formed in the gland; but their intimate union with the elastic tissue, which is evenly and generally distributed throughout the framework of the corium, results in their discharge of a still more important function in connection with the regulation of the body-temperature; since by virtue of direct compression exerted upon the skin the blood may be driven from the surface in a centripetal direction and its cooling in a great degree prevented, as in the well-known phenomena resulting in the production of the *cutis anserina*, or "goose-flesh." The reverse of this naturally follows when the muscles expand under the influence of external heat. The anatomical connections of the arrectores pilorum are such that their contraction serves to approximate several of the papillæ of the corium, including the hair-papillæ. Thus, by their contraction, the sebaceous secretion may be extruded, or, as is more particularly exhibited in the lower animals, such hairs as the bristles of the boar may be erected.

Muscular membranes exist in the skin of the scrotum, over the penis, about the nipple, and elsewhere. They are simply layers of smooth muscular fibers, which suffice when contracting to move the portions of skin to which they are distributed.

Pigment.—The hue of the living integument is due in part to the degree of vascularity and distention of the vessels in the corium, and in part also to pigmentation of the epidermis. The coloring matter of the skin in health is deposited chiefly in from one to four rows of cells in the lower stratum of the rete, the fine granules of pigment staining both the cell-body and the nucleus, the latter more vividly. The pigment of the skin depends for its hue upon a substance called *melanin*. Its office is obvious. It is designed to absorb rays of light, and thus to aid in the protection of the body from undue insolation.

The degree of vascularity of the skin is responsible for most of the flesh-tints, but the colors seen in the various races of men are wholly related to the character and quantity of pigment found in the rete. Rarely, pigment cells are found in the corium in a state of health. This pigment depends upon a distinct and uniform coloration of the epithelia, and also upon minute granules of melanin entangled in the reticulum of living matter in the same part. Extreme variation in the distribution of pigment is noticeable both in health and in disease, and in individuals and races, being at times related to climatic and similar influences. This fact is well illustrated by the wide range between the flaxen-haired, pink-eyed albino and the blackest specimens of the negro, each, with small exception, being of African descent.

It has already been noted that in the colored races the pigment may stain the epithelial cells and their nuclei as high as the granular layer; and that to this layer only is due the characteristic color of the skin of the white races. Pigment is not normally found either in the horny layer of the skin or in the subepithelial tissues.

The source of the pigment in the skin has been the subject of much study. Karg,[1] Kölliker,[2] and Ehrmann[3] all support the view of the derivation of the pigment from the hemoglobin and its transference to the basal layer of the epithelium by special cells (*melanoblasts, chromatophores*). Ehrmann considers these pigment-bearing cells derivatives from the mesoderm and different from connective-tissue cells or leukocytes. Delépine,[1] Jooss,[2] and more recently Meirowsky,[3] Helmich,[4] McDonagh,[5] Dyson,[6] and Kreibich[7] consider that the pigment is independently produced in the epithelial cells.

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[1] Archiv f. Anat. u. Physi., 1888, p. 369.
[2] Zeitsch. f. Wissensch. Zoolog., 1887, xlv.
[3] Vierteljahr., 1886, xiii.

The experimental work carried on by these observers appears to prove conclusively that the epidermal pigment is formed in the individual epithelial cells, the formation beginning in the nucleus and spreading from this situation throughout the cell. It is further believed by some that independent pigment cells may be produced in the corium.

Hairs.—The study of the anatomy of the hair-apparatus includes the structure of the hairs themselves and the follicles in which they are implanted. Hairs are distributed over the entire cutaneous surface, except on the palms and soles, the dorsal surface of the distal phalanges of the hands and feet, and the skin of the penis. They occur in three classes: the fine, downy hairs, or lanugo, covering the face, the trunk, and the limbs; the long, soft hairs found on the scalp, over the pubes, and in the axillæ; and the short hairs, which include the soft varieties found on the brow and the stiff hairs of the eyelids. The wide variation in color depends upon the amount of pigment granules, the soluble coloring matter, and the air which they contain. Hairs, on cross-section, are round, oval, or more or less flattened. The portion of the hair included in the skin is termed the *hair-root*, and its lowermost portion the *hair-bulb*. That portion extending from the surface to the free extremity is termed the *shaft*, and the terminal end the *point*.

The bulb is a club-shaped expansion of the lower end of the hair, implanted upon and surrounding a nipple-like projection of the corium termed the *hair-papilla*, which resembles the vascular papilla of the papillary layer of the corium. When uncut and normal, the external extremity of the hair ends in a sharp point.

The distribution of the hair is determined by the fibrous tissue of the corium, and corresponds closely with the lines of cleavage of the skin. In certain areas, such as the vertex of the scalp, the hairs are arranged in a circular manner, forming whorls.

On cross-section the hair presents a cuticle, a cortex, and a medulla, and is composed of epithelial cells.

Cuticle.—The external layer of cells of the hair is termed the cuticle. This is composed of cells quadrilateral in shape, flat and regularly overlaid, resembling the tiles on the roof of a house. They have their long axes directed upward and outward at an acute angle with the shaft.

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[1] Proceedings of Physiological Society, 1890, vi, p. 23; quoted by MacLeod, Histopathology, p, 306.

[2] Münch. med. Abhandl., I, Heft. xvi; quoted by MacLeod, Histopathology, p. 306.

[3] Monatshefte, August 15, 1906, p. 155; abstr. Jour. Cut. Dis., 1907, xxv, p. 192.

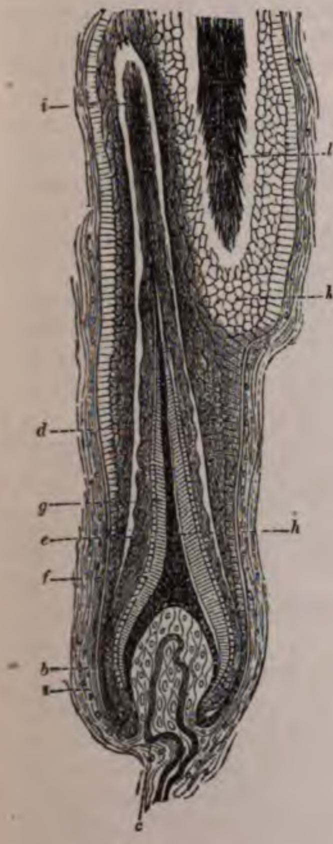
[4] Monatshefte, Bd. xlv, No. 4, 1907, August 15, p. 184; abstr. Brit. Jour. Derm., 1908, xx, p. 169.

[5] Brit. Jour. Derm., 1910, xxii, p. 316.

[6] Ibid., 1911, xxiii, p. 205

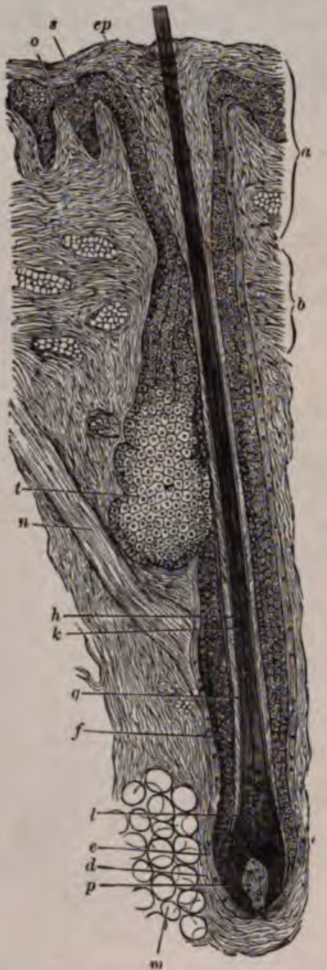
[7] Archiv, cxviii, No. 3; abstr. Brit. Jour. Derm., 1914, xxvi, p. 171.

FIG. 12



Section of a hair-follicle during the formation of a new hair: *a*, external and middle root-sheaths; *b*, vitreous membrane; *c*, papilla with vascular loop; *d*, external root-sheath; *e*, internal root-sheath; *f*, cuticle of hair-follicle; *g*, cuticle of hair; *h*, *i*, young hair; *l*, bulb of old hair; *k*, débris of external root-sheath of hair recently expelled. (After Ebner.)

FIG. 13

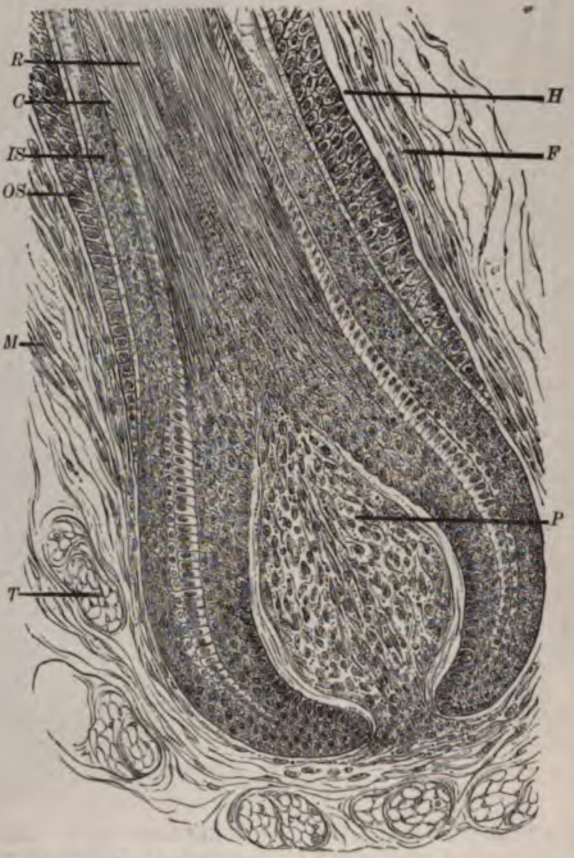


Hair-follicle in longitudinal section: *a*, mouth of follicle; *b*, neck; *c*, bulb; *d*, *e*, dermic coat; *f*, outer root-sheath; *g*, inner root-sheath; *h*, hair; *k*, its medulla; *l*, hair-knob; *m*, adipose tissue; *n*, hair-muscle; *o*, papilla of skin; *p*, papilla of hair; *s*, rete mucosum, continuous with outer root-sheath; *ep*, horny layer; *t*, sebaceous gland.

Cortex.—In its upper portion, the hair consists entirely of the cortex. Lower down a medulla is present. The cells of the cortex are spindle-shaped and have a fusiform nucleus. They contain coloring matter, and between them are found pigment-granules and air-spaces. The strength, elasticity, and extensibility of the hair are due to the arrangement of the cells of the cortex.

Medulla.—The medulla occupies the center of the hair and extends a variable distance up the shaft. In its upper portion, it consists of several rows of flattened epithelial cells, which near the bulb become cubical in shape and contain keratohyalin. In the lanugo hairs the medulla is absent.

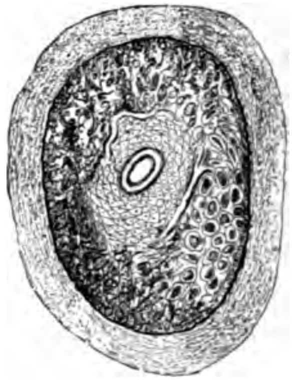
FIG. 14



Lower portion of hair-pouch from the lip of a kitten: *F*, follicle; *T*, transverse section of connective-tissue bundles of derma; *M*, arrector pili muscle; *IS*, inner root-sheath; *OS*, outer root-sheath; *P*, papilla; *C*, cuticle; *R*, root of hair; *H*, hyaline, or so-called "structureless," membrane. Magnified 500 diameters. (After Heitzmann.)

Hair-follicle.—The hairs are implanted in the skin in a series of invaginations called hair-follicles. Usually, only one hair springs from a single follicle, but occasionally there may be two or more. The hair-follicle is an elongated, cylindrical pouch, dipping down into the corium, and at times into the subcutaneous tissue. Its upper portion, termed the mouth, is funnel-shaped and opens directly upon the cutaneous surface. The neck of the follicle is its narrowest portion, and is situated at the junction of the middle and upper thirds, and at this point occur the orifices of the sebaceous glands. Below this the pouch gradually enlarges to end in a bulbous extremity, which contains the hair-bulb and hair-papilla. The follicle has a fibrous portion derived from the corium, and an epithelial portion made up of the various layers of the epidermis. The fibrous coat of the hair-follicle is divided into three layers: an external, thin, dense layer of connective-tissue fibers and cells, arranged in a longitudinal direction; a middle, thicker layer, with a circular arrangement of the fibers and bundles; and an internal, condensed layer, presenting a homogeneous appearance. This last-mentioned layer is commonly described as the *hyalin layer*, or vitreous layer, and is present chiefly in the lower part of the follicle. The epithelial portion of the hair-follicle is composed of the various layers of the epidermis. The layer next the hair, corresponding to the stratum corneum, is termed the cuticle. External to this is a complicated structure termed the internal root-sheath; and outside of this is the external root-sheath, which is virtually a continuation of the prickle-cell layer of the epidermis.

FIG. 15



Transverse section of hair and follicle.

Cuticle.—The cuticle is composed of a single layer of elongated cells, with their long axes arranged downward and inward. By recalling the arrangement of the cells of the cuticle of the hair, it will be seen that by the special arrangement of the cells of the two cuticles they interlock and thus make a firm union between the hair and its follicle. This anatomical arrangement accounts for the removal of a part of the root-sheath when a hair is epilated.

Internal Root-sheath.—The internal root-sheath occupies the lower two-thirds of the hair-follicle. The inner part of this sheath is known as the "sheath of Huxley," while its outer portion is termed the "sheath of Henle." The cells of the inner portion (sheath of Huxley) are nucleated and contain granules of keratohyalin. Those of the outer portion (sheath of Henle) are smaller, have lost their nuclei, and have become cornified. There are spaces between these cells through which processes from the cells of the inner layer extend, on account of which this layer has been described as a fenestrated membrane. In the upper part of the follicle, these two layers lose their identity through the cornification of the cells of the inner layer.

External Root-sheath.—This is represented by a continuation of the stratum mucosum downward into the follicle. The stratum corneum and the stratum granulosum accompany the stratum mucosum downward as far as the opening of the sebaceous gland at the neck of the follicle. From here downward to near the papilla the stratum mucosum consists of several layers of polyhedral epithelial cells, while near the base of the papilla it is reduced to a single layer of cells.

Hair Change.—During life, the hairs are being constantly shed and replaced by new ones. In the scalp, they are said to have an existence of from two to four years, when they are replaced by new ones. In addition to the intermittent falling of the hairs, there are certain periods during which great changes occur. Immediately after birth, the hairs grow actively on the scalp. At puberty, hairs appear in certain regions, such as the beard, the axillæ, and about the genitalia. In middle life and later years, hairs grow in and about the ears and nostrils. In females, after the menopause, there is frequently an increase of hairs on the face. All these hairs (called "periodic hairs" by MacLeod) are pigmented and coarse, while those of the rest of the body retain their unpigmented condition.

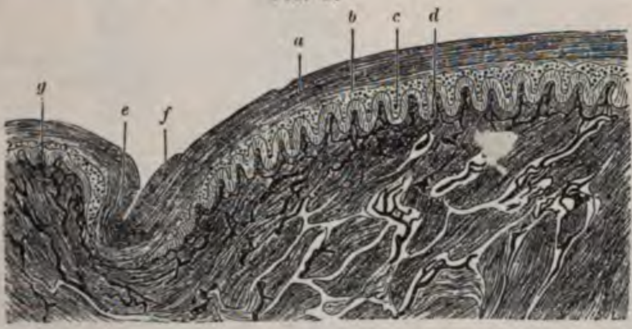
Nails.—Nails are compact, solid plates of highly cornified epithelial cells, situated on the dorsal surface of the distal phalanges of the fingers and toes. They are convex from side to side, also from before backward, but to a less degree. Three edges of the plate are inserted into the skin, one, the anterior, being free. The posterior edge is slightly concave, the lateral edges straight and parallel, and the anterior edge convex. The visible portion of the nail-plate is termed the *body*, the posterior one-fifth of which is occupied by a semilunar, whitish portion, termed the *lunule*. The portion of the nail-plate embedded posteriorly is called the *root*. The three edges of the nail-plate rest in a depression termed the *nail-groove;* and the skin extending from this over the nail for a short distance laterally, but further posteriorly, is called the *nail-fold* or *nail-wall*. The thin, crescentic membrane extending from the posterior wall a short distance over the lunule represents the remains of the *eponychium*. The *hyponychium* is that portion of the epidermis upon which the nail-body rests, and the *perionychium* is that portion of the epidermis surrounding the entire nail-border.

The following structures are presented for microscopic study: the nail-fold, nail-matrix, nail-bed, and nail-plate.

Nail-fold.—The nail-fold is divided into an upper and a lower portion by the nail-root. The upper portion, extending backward, consists of the various layers of the epidermis forming a roof for the nail-root. The stratum corneum lies upon the nail-root, and the finger-like projections of the rete are obliterated and represented here by a straight line. The lower portion, extending forward beneath the nail-root, represents the nail-matrix.

Nail-matrix.—The nail-matrix corresponds to the lower portion of the nail-fold, and extends forward to the anterior margin of the lunule. It is composed of a prickle-cell layer continuous with the same layer behind forming the roof of the nail-fold, and in front with the prickle-cell layer of the nail-bed. The prickle cells in this situation are larger than those of the same layer anteriorly and posteriorly. Interpapillary ridges are present and are more marked posteriorly. The matrix is composed of layers of cells analogous to those of the epidermis, with some variation noted, as follows: A basal layer, composed of regular, cylindrical epithelial cells. Immediately above this are from three to ten rows of cells of polygonal shape, and above this several rows of flat, closely-packed cells, having shriveled nuclei and containing fine granules. Above this are found the horny complete nail-cells. The nature of the granules mentioned in the transitional layer is not settled. Ranvier (quoted from MacLeod) terms them *onychogene*. Okamura[1] regards them as keratin granules within the cells. A third hypothesis is that they are shrunken prickles "seen in relief" (MacLeod).

FIG. 16



Vertical section of one-half of nail and matrix: *a*, nail-substance; *b*, horny layer; *c*, mucous layer; *d*, papillæ of corium; *e*, nail-furrow destitute of papillæ; *f*, horny layer of the ungual furrow rising above the nail; *g*, papillæ of skin of dorsal surface of the finger.

Nail-bed.—The nail-bed extends forward from the matrix to the horny layer of the pulp which joins the nail at its free edge. The nail-plate rests upon the nail-bed. The nail-bed has a basal layer of cells presenting longitudinal edges, several layers of polygonal cells, and above these flattened cells, upon which the nail-plate rests.

Nail-plate.—The nail-plate (*true nail*) is produced entirely from the matrix and is pushed forward over the nail-bed. The upper cells spring from the posterior portion of the matrix, while those beneath are produced by the anterior portion. It is suggested that the pressure exerted by the roof of the nail-fold helps to direct the growth forward in place of upward. The nail-plate is made up of flattened epithelial cells arranged in lamellæ, and each cell contains the remains of a nucleus. This cellular structure is best seen near the matrix; the cells further forward being so closely associated as to become an almost homogeneous mass. The under surface of the nail-plate is traversed by fine longitudinal ridges, which fit into corresponding depressions in the nail-bed.

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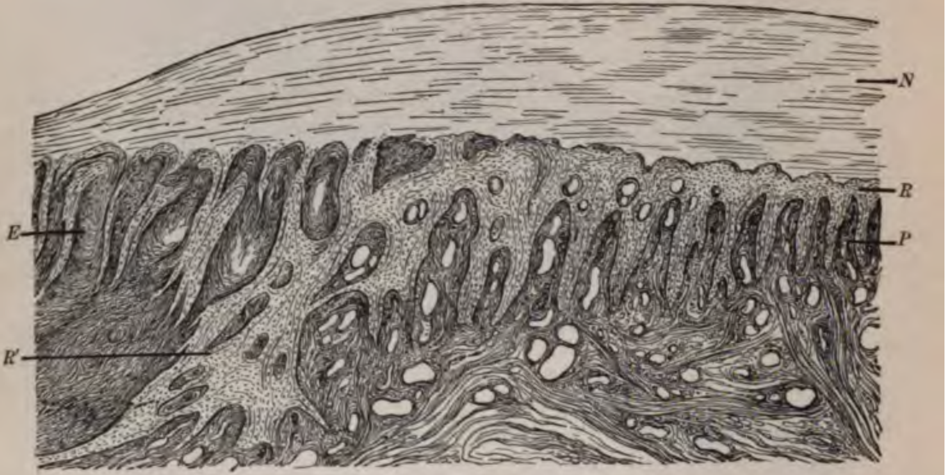
[1] Archiv, 1900, lii, p. 223; quoted from MacLeod.

The process of cornification occurring in the nail is apparently accomplished without the interposition of keratohyalin and eleidin, and the final product differs from keratin in the degree of hardness and otherwise. Finally, the presence of shrunken nuclei in the cornified cell is different from the usual process of cornification described in the epidermis. The structure of the corium and subcutaneous tissue of the nail region presents certain peculiarities. Collagen-bundles radiate vertically from the periosteum of the phalanx to the epidermis of the nail-bed, closely binding these structures. Other collagenous bundles are present having a horizontal arrangement. Elastin is present below the matrix, and to a less degree beneath the nail-bed. Bloodvessels and lymphatics are present in this meshwork, and also a moderate amount of fat (MacLeod). The papillary body has a special arrangement, described by Hans v. Hebra[1] (quoted from MacLeod). Beneath the matrix posteriorly occur from three to six rows of small, isolated papillæ. Anterior to this is seen a series of ridges, which are curved at the sides, but parallel with the long axis of the nail in the center. On the edges of these ridges, rows of papillæ are present, giving this region the appearance of a cock's-comb. From here forward to the anterior part of the lunule the ridges flatten and papillæ are absent. The papillæ of the nail-bed are arranged on a large number of ridges running parallel with the long axis of the nail, and anterior to this the ordinary papillæ of the

FIG. 17



Implantation of a nail at its border: *P*, papillæ decreasing in size toward the middle line; *R*, rete mucosum, which broadens toward the border of the nail, and forms irregular prolongations; *R'*, *E*, epidermal layer; *N*, plate of the nail. Magnified 500 diameters. (After Heitzmann.)

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[1] Wiener med. Jahrb. 1880, p. 59.

corium of finger-pulp occur. A copious supply of bloodvessels is found in the papillæ and ridges above described. Beneath this is a rich plexus of bloodvessels, on which various sinuses lined with endothelium are present. Into these sinuses the capillaries of the papillæ empty their contents.

The growth of the nail is continuous during the life of the individual, being more active in the young and during the summer season. From 100 to 160 days are required for the reproduction of a finger-nail and about three times that period for a nail of the toe.

Fig. 18

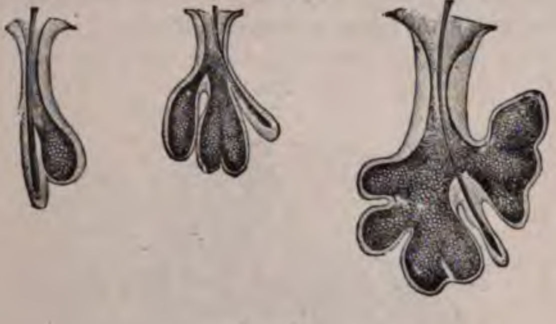
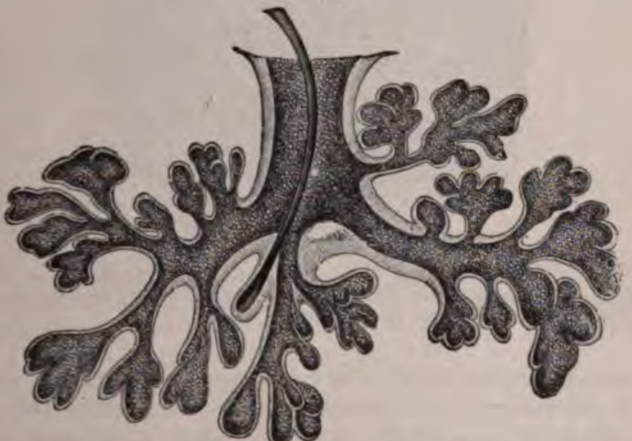


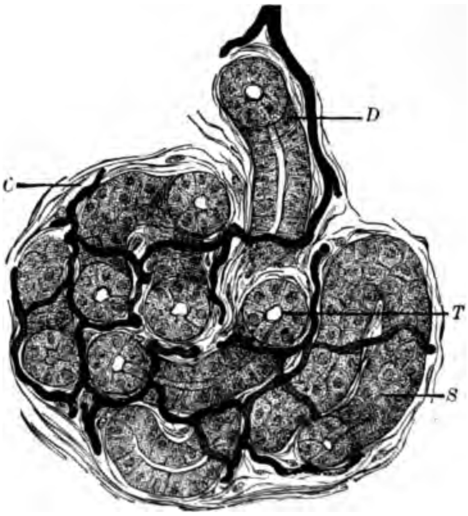
Fig. 19



Sebaceous glands of the second class, from the alæ of the nose. (After Sappey.)

Glands.—**Sebaceous** glands, or sebiparous glands, are pyriform bodies, usually racemose in development, situated in the corium. They furnish a more or less consistent and fatty secretion, destined to anoint the skin and the hairs. They are divided into three classes. The first class includes the sebaceous glands that are appendages of the hairs and hair-follicles. They are developed early in fetal life from minute, lateral, bud-like prolongations from the outer root-sheath of the hair. From two to six of these prolongations spring from the prickle layer of the hair-follicle, and the prickle cells in the axis of each bud speedily undergo fatty metamorphosis. In the mature gland, each acinus is formed of a *membrana propria* supporting layers of nucleated cuboidal epithelial cells furnishing fat. Gradually the fatty cells are pushed outward toward the duct of the gland, where, sooner or later, their rupture releases the drops of fat (sebum) just where the hair emerges from the closely applied follicle below to the funnel-shaped mouth of the hair-pouch above. Externally, each gland is provided with a layer of connective tissue provided with blood- and lymphvessels and nerves. Sebaceous follicles are found in connection with the long, soft hairs, as those of the scalp and axillæ, several being grouped around a single hair-sac.

FIG. 20



Coil of a sweat-gland: *S*, tubule lined with cuboidal epithelia; *T*, central caliber of the tubule; *D*, beginning of the duct; *C*, connective tissue with injected blood-vessels. Magnified 500 diameters. (After Heitzmann.)

The second class includes the large and complex glandular structures to which the lanugo hairs seem accessory, the orifices of their respective ducts opening directly upon the cutaneous surface.

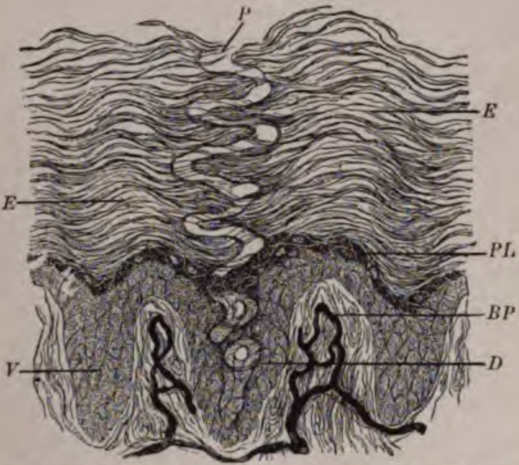
The third class includes those sebaceous glands opening directly upon the surface and unconnected with hairs or hair-follicles. Such are the glandulæ odoriferæ of the male and female genitalia, and those existing about the lips and in the areola of the nipple. These glands might be designated as "glands of the mucous orifices" (Unna).

Meibomian and Tysonian Glands.—These are of the largest order of sebaceous glands. The former exist within the free border of the eyelids; the latter upon the glans penis and the inner surface of the prepuce. They are unconnected with hairs, and differ in this respect from other types of sebaceous glands.

Glandulæ Ceruminosæ.—These are situated in the subcutaneous tissue of the meatus of the ear, and produce the waxy secretions found in this situation. The "glands of Moll" found in the eyelid are to be classed with the sweat-glands.

Coil Glands.—Coil glands (*sweat or sudoriparous glands, glandulæ glomiformes*) are found within the skin of all regions of the body, being exceptionally numerous in the palms and soles. They are situated in the subcutaneous tissue, as a rule, with an occasional one in the deeper part of the corium. In certain regions, such as the axillæ, the groins, the palms, the soles, and about the anus, the coil glands are multiple and of unusual size, and often of peculiar arrangement. For descriptive purposes, the coil gland apparatus is divided into the gland (the coil proper), the sweat duct, and the sweat pore.

FIG. 21

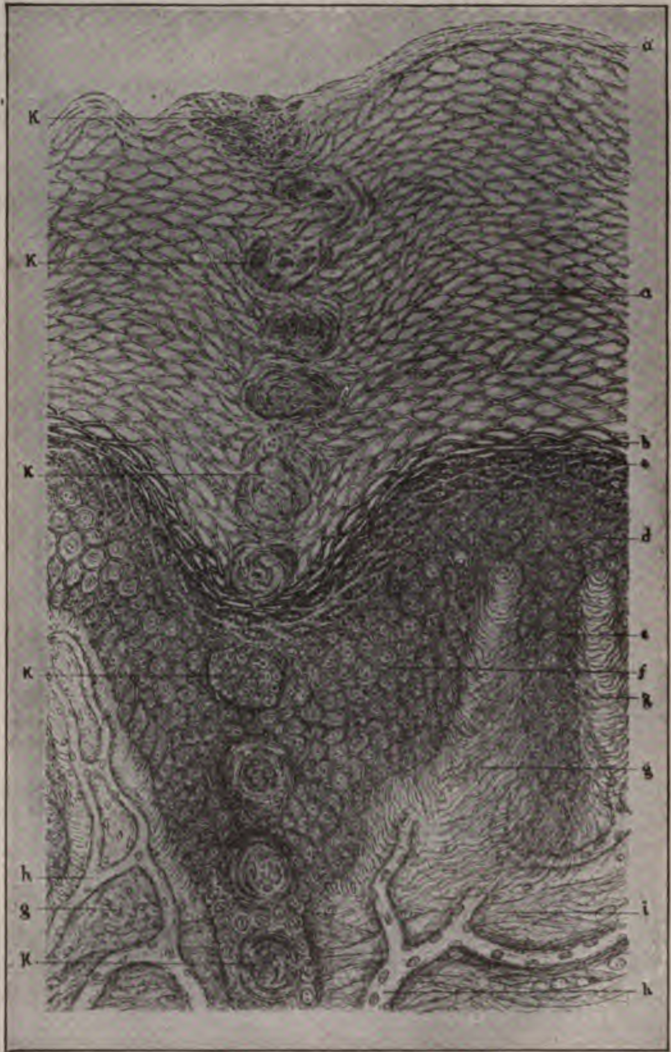


Sweat-pore traversing the epithelial layers of the skin: *BP*, papilla with injected blood-vessels; *V*, valley between two papillæ; *D*, duct in the rete mucosum; *E*, *E*, epidermal layer; *PL*, coarsely granular epithelia, deeply stained with carmine; *P*, duct with cork-screw windings in the epidermal layer. Magnified 200 diameters. (After Heitzmann.)

Coil.—The coil is simply the end of a tube coiled upon itself from a few to several times. The tube terminates in a cecal pouch and is lined with a single layer of nucleated cuboid epithelial cells, having a granular appearance. Outside of the tube are smooth, muscular fibers, running parallel with or in a spiral direction about the coil. Surrounding both muscle-bundles and epithelium, a connective-tissue membrane is described. The glomerulus or coil is globular in outline and reddish-yellow in color. In the larger glands, irregular dilatations and constrictions of the tube are conspicuous.

Coil Duct.—The excretory duct of the coil gland passes from the glomerulus below to the epidermis above in a straight or a spiral course. It is lined with delicate hyalin cuticle (Heynold), beneath which is a double layer of cuboidal epithelial cells. Externally is a membrana propria, unprovided with muscular fibers. The external sheath consists of connective tissue. At the border line of the epider-

Fig. 22



Section of the skin from the palm of the hand (hardened in Moeller's fluid and treated with glacial acetic acid), magnified 300 diameters, showing epidermis and pars papillaris of the corium traversed by the excretory duct of a coil-gland terminating in a sweat-pore: *a*, stratum corneum; *a'*, its superficial layer, the cells in the upper and lower layers somewhat larger than those situated between the two; *b*, stratum lucidum; *c*, stratum granulosum; *d*, stratum mucosum; *e*, rete-pegs; *f*, interpapillary process of rete meeting duct of coil-gland; *g*, *g*, papillæ embraced by long prickles extending from lower palisade-layer of the rete; *h*, blood-vessels of papillæ; *i*, bundles of connective-tissue fibers of pars papillaris; *k*, section of spiral duct of coil gland and sweat pore.

mis, the cuticle and external connective-tissue sheath are lost, and the duct here becomes a sweat-pore. It occasionally opens within a hair-pouch.

Sweat Pore.—This is a continuation of the excretory duct of the coil-gland after the loss of its cuticle and connective-tissue sheath. The sweat pore traverses the epidermis usually in a spiral direction, and terminates in a funnel-shaped opening through the stratum corneum. It is a wall-less channel, and therefore in free communication with the juice-spaces of the epidermis. This anatomical peculiarity provides fully for the needs of evaporation at the surface of the body.

The alternation of muscular fibers with the secretory cells of the ducts of the coil glands is a provision for the extrusion of the gland secretion onward. The same anatomical arrangement permits communication between the epithelial cells and the lymph spaces which reach into the connective-tissue sheath of the gland. As a result, the lymph flows freely among the secreting elements of the gland and its duct. This lymph, loaded with fat, streams away from the coils, and before it reaches the lymphatic trunks its fat-globules are filtered away in the subcutaneous tissue. The total number of coil glands in the body is estimated to be between 2,000,000 and 3,000,000, and the total length of the uncoiled glands to be eight miles. These figures serve to give an approximate idea of their very great physiological importance and of the extent to which violation of the rules of hygiene possesses interest from a pathological view-point.

DEVELOPMENT OF THE SKIN.

From an embryological standpoint, the skin is composed of two layers, the epidermis and corium; the epidermis developing from the epiblastic layers, and the corium from the mesoblastic layers, of the blastodermic vesicle. The corium and subcutaneous tissue are now regarded as layers of a mesoblastic structure maturing from within outward, increasing by cell-division and by the formation of fibrous bundles and the deposition of fat in the deeper layers.

The epidermis early in the blastodermic vesicle consists of a layer of polygonal nucleated cells, termed "Rauber's layer," beneath which is a layer of cubical cells with large, round nuclei. Between the first and second months of fetal life three layers are distinguished most superficially: Rauber's layer, the cells of which have become flat; and two layers of columnar cells. About the third month, the "bladder cells" of Zander become demonstrable in the regions of the tips of the fingers and toes. These latter appear to be certain cells of Rauber's layer which have become swollen and dome-shaped (Bowen).

Epitrichial Layer.—Welcker, Minor, and Bowen[1] have described a layer of large cells, with round nuclei much larger than those of the epidermal layers beneath, covering the entire body of the human embryo during the early months of its existence. This layer, histologically, is quite distinct from the outer cells of the stratum corneum, and corresponds with the epitrichium of certain animals. It usually disappears before the sixth or seventh month of intra-uterine life.

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[1] Jour. Cut. Dis., 1895, xiii, p. 485.

At the end of the fourth month, the epidermis consists of five or six rows of cells: a superficial row of flattened cells, beneath which are several rows of cells of polygonal shape. In the lowermost layer, mitotic figures may be demonstrated. By the sixth month, marked proliferation has taken place in the epidermis, and the epitrichium has disappeared except in the fingers and toes and over the developing nails. By this time, also, the proliferating epidermis has formed the papillæ, and a short time later (about the seventh month) the prickle-cells take on their peculiar characteristics. Keratohyalin granules become evident about the eighth month, and keratin is also present in the cells of the stratum corneum.

Coil Glands.—The first evidence of the coil glands may be found in the palms and soles, between the fifth and sixth months, as a conical down-growth of the epidermis. Very shortly afterward, these gland rudiments spread over the entire subcutaneous surface, except that part occupied by hairs. The rudiment gradually pushes downward into the corium and becomes constricted near the upper part, forming a neck, and widens out beneath into a globe-shaped thickening. These down-shoots occur over the interpapillary processes of the epidermis, and the cells are those composing the epidermis from which it sprang. Between the sixth and seventh months, the central cells of the developing gland break down and the beginning of a canal becomes appreciable. When this rudiment reaches a certain position in the corium, the end turns and the subsequent growth produces a coil.

Sweat Pores.—The sweat pores are first demonstrable between the seventh and eighth months, some time after the gland is fully developed. Fat-globules may be seen in and between the cells of the coil before there is any external outlet. The fat appears to pass out from the cells of the coil to the surrounding tissue. As no sebaceous glands are present on the palms and soles, it has been suggested that the vernix caseosa has been produced by coil glands.

Hairs.—The first change noticeable in the area which is to form a lanugo hair is a down-growth of the epithelium into the corium, occurring between the second and third months of fetal life, and situated about the forehead and eyebrows. From here these down-growths spread to all regions of the body, except the palms and soles. Between the fourth and fifth months, the hair itself begins to form in this early rudiment. By the sixth month, a lateral outgrowth from the hair-rudiment has developed into a sebaceous gland. Immediately beneath this is a small projection for the attachment of the fibers of the arrectores pilorum. When the hair becomes sufficiently rigid, it pierces the horny layer of the epidermis and makes its appearance on the surface. At about the seventh or eighth month, the lanugo hairs begin to exfoliate and are replaced by new hairs growing out of the old follicles. This change is sometimes completed after birth. A very clear conception of the development of the hair as it occurs in the rabbit is quoted from MacLeod:

"A solid process of epithelial cells grows down from the epidermis into the underlying connective tissue of the corium; a small, vascular papilla of connective tissue forms below the down-growing process; the papilla increases in size, indents the fundus of the process, and from it a sheath of connective tissue spreads up to envelop the process and form a follicle; the axial cells of the process then become cornified and a hair is formed; the hair grows and finally breaks through the overlying epidermis, and the development is complete."

Sebaceous Glands.—The sebaceous glands appear first about the fifth month of fetal life as merely knob-like outgrowths of the hair rudiments, and are composed of cells of the same type. As they grow, they become pear-shaped, and have their long axes directed obliquely downward. The original process buds to form a number of saccules enclosed in a connective-tissue sheath continuous with that of the hair-follicle. From two to six glands develop in connection with each follicle.

PHYSIOLOGY OF THE SKIN.[1]

The skin, through its various component parts, renders great service to the body as a whole through performing many physiological functions vital to life. Most important of these are protection, heat regulation, secretion, sensation, and respiration, the last-named only to a small degree. The skin is not simply an inert envelope in which the structures of the body are confined, but is a living organ comparable in importance to the liver, kidneys, and other similar organs.

Protection.—The epidermis is a poor conductor of electricity and light. In the tropics, where the light is greatest, the natives are provided with an increased amount of epidermal pigment to screen them from the light. Inhabitants of the temperate zone, while visiting tropical countries, require such clothing and shelter as will assist the epidermis in checking light-penetration.

The fatty matter in the stratum corneum prevents evaporation of the fluids of the body. The impermeability of the stratum corneum protects other organs of the body from the absorption of water and other fluids. Keratin is a substance which enjoys great power of resistance to chemicals of all kinds. Resistance to microörganisms which are normally present in the epidermis and to those also that are pathogenic that find accidental lodgment in the skin is provided by the impenetrability of the granular scales, their coherence, and the fatty matter present. The points offering least resistance to their entrance are the glandular orifices. In sweat-glands there may be an acid secretion (the sweat) exuding from a narrow, tortuous canal. The weakest point in the epidermis is the pilo-sebaceous system, where the sebaceous secretion, which is pasty and fatty, offers some resistance. The fibrous corium, by its strength and elasticity, with the loose, fat-containing subcutaneous tissue, acts as an ideal support and protective apparatus against external injury of the delicate nerves, with their special endings, the blood- and lymph-vessels, the glands and hair-follicles of these regions. The brain has an extra protection through the abundant hair-growth of the scalp.

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[1] In the preparation of this chapter the following works were consulted: La Pratique Dermatologique, Tome I; Tigerstedt's Physiology, 1905; Duhring's Cutaneous Medicine, 1896, vol. i.

Heat Regulation.—In health, the temperature of the blood is maintained at nearly a given point, though the body be exposed to temperature changes of wide variation. This is accomplished, in the main, by the skin through radiation, conduction, and evaporation. When the body is overheated, either from external or internal causes, the blood is determined to the surface capillaries, with loss of heat by conduction and radiation; and at the same time increased activity of the coil glands is stimulated, with outpouring of sweat, which by evaporation dissipates such heat. When the body is exposed to the cold, the cutaneous capillaries contract, sweat secretion is diminished or stopped, and loss of heat is thus prevented. In addition, heat loss is lessened through contraction of the arrectores pilorum, which occurs when the body is chilled, by lessening the exposed cutaneous surface. The vasomotor system regulating the blood supply is intimately concerned in the phenomena above described, and apparently may act by direct excitation from without (heat and cold), or reflexly from within by fever, hot drinks, shock, drugs, etc.

Secretion.—The secretory function of the skin is carried on chiefly by the sebaceous and coil glands. Their office is to furnish oil and moisture to render the skin soft and pliable, and, to a small degree, give off waste material. They play an important part in the temperature. The sebaceous glands secrete in health greasy and oily matter (sebum), which anoints the greater part of the cutaneous surface, including the hairs. The palms and soles are not anointed by this secretion, and they are the only parts which show the effect of water even after prolonged immersion. In the glands, sebum is a fluid or semifluid substance, which may be of firmer consistency in the ducts, and is, according to Starling, rather a wax than a true fat, and composed of fatty acids united with the stronger alcohols, including cholesterol. The expulsion of sebum from the glands to the surface is accomplished largely by contraction of the arrectores pilorum muscles which surround the sebaceous glands.

Sweat is composed largely (98 to 99 per cent.) of water. It is colorless, and has a specific gravity of 1003 to 1008. Its reaction may be acid, alkaline or neutral. It has an unpleasant odor and a salty taste. The odor varies according to the part of the body from which it is secreted. Under ordinary conditions it is acid, but after profuse perspiration its reaction becomes neutral or alkaline. The chemical composition of sweat is difficult to ascertain, owing to the admixture with material from the sebaceous glands, which necessarily cannot be eliminated. The quantity daily excreted is variable and depends largely upon the requirements for heat regulation. The amount in twenty-four hours is probably between a pint and a half and two pints. Urea is ordinarily present in very minute quantity, but in certain pathological conditions, such as uremia, may become appreciable in amount. While the entire skin secretes sweat, certain portions, such as the brow, face, neck, axillæ, genital regions, palms, and soles, are the chief areas of such activity. That there is a division of work between the skin and the kidneys in excreting water is shown by the light color of the urine in winter, when perspiration is at a minimum, while the urine is heavy and darker in color in summer, when the skin is actively secreting. The sweat secretion is influenced both by reflex and central stimulation. The secretion is increased by elevation of the external temperature, by copious warm draughts, by certain drugs, such as pilocarpin, strychnia, camphor, ammonia, etc., and by such psychic phenomena as fright and anxiety. It is diminished by external cold and such drugs as morphin and atropin, and in certain pathological conditions, such as diabetes. The nervous centers for its regulation are located both in the medulla and spinal cord. The sweat centers may be stimulated by venous blood caused by dyspnea preceding death, and reflexly by exciting the mucous membranes of the mouth with mustard and other condiments (Halliburton). While increased perspiration occurs as the result of vasomotor dilatation of the vessels in the skin due to heat or muscular activity, it may occur independently of this, as is shown in the psychic causes mentioned.

Absorption.—It is generally admitted that the intact skin is impermeable to water and other solutions. Normal fats, on the other hand, are absorbed to a certain degree, as well as chemicals incorporated in such fats. Advantage of this is taken in the treatment of syphilis by inunction. When the epidermis is removed, absorption may readily take place, and this fact should be borne in mind when making applications containing toxic agents to large inflammatory areas in which the stratum corneum has been removed.

Sensation.—This function of the skin provides a means of protection and discrimination. It was formerly thought that different sensations were awakened by various degrees and kinds of irritation applied to a single nerve-ending. Through the work of Blix (1833) and Goldschneider (1886), it is now known that there is a disassociation of sensation. There are different nerve-endings for heat, for cold, for tactile sense, and for pain.

Temperature Sense.—There are scattered over the surface of the body certain points which convey only the sensation of cold, whether the irritation be electrical, mechanical, chemical, or by a hot or cold needle. There are other points which transmit only the sensation of heat. Where the tactile sense is most acute, as in the hand, the temperature sense is diminished. The topographical areas of heat and cold must be studied from charts.

Tactile Sense.—This sense is not equally distributed over the surface of the body. It is keenest in the finger-tip and the point of the tongue. It includes relative perception, with discriminations as to roughness, smoothness, hardness and softness, dryness and moistness. Relative perception is often an aid to vision. Pressure sense is often increased by the presence of hairs.

Pain Sense.—Where the surface is denuded of epithelium, it is not apt to receive other sensation than that of pain. Pain, too, is experienced from thermal, mechanical, chemical, or electrical irritation of severe character. There are nerve-terminals which receive no other impression than that of pain.

The nature of sensations of itching, tingling, and creeping has not yet been fully determined. Clinical observation suggests that one of the important factors in the production of itching is pressure on the epidermis. Any inflammatory or serous exudate occurring in just the right location to produce an outward pressure on the epidermis will cause itching. If the exudate is more deeply located, other sensations are experienced. Tingling and creeping sensations are apt to denote deranged innervation.

Respiration.—The respiratory activity of the skin depends on its permeability to gases and vapors, in which function it is accessory to the lungs. The manner in which this is accomplished is through a diffusion between the circulating blood in the capillaries and the atmosphere. Physiologically, oxygen is absorbed as well as some other gases. Water, carbon dioxid, and a trace of nitrogen are expelled. The respiratory function of the skin is not important and is far less in man than in amphibious animals (frog), whose skins are more like mucous membranes. The skin of these animals cannot be transplanted to man.

II. GENERAL SYMPTOMATOLOGY.

The symptoms in diseases of the skin are divided into two groups, subjective and objective. The former are those appreciated by the patient alone in consequence of his sensations. The latter are those seen by the eye or detected by the touch of another who undertakes the investigation of the disease. Some diseases of the skin have associated general symptoms in which the cutaneous lesions are merely one expression of the pathological process. It is therefore necessary to study in detail not only those symptoms seen on the skin and described by the patient relative to the skin, but also to detect any departures from the normal in the condition of the viscera and properly to interpret these findings.

SUBJECTIVE SYMPTOMS.

The subjective symptoms of a disease of the skin are those manifested to the patient by sensations other than those connected with his vision or his own sense of touch. Chief among these are itching, burning, smarting, prickling, and tingling. Other sensations are increased or diminished susceptibility to contact with foreign bodies; sensations of increased or diminished temperature; pain in various grades of severity, and paresthetic sensations such as those suggesting the crawling of insects over the part, or the passage of currents of hot or cold air and the compression of portions of the skin as by cords or bands. The subjective sensations occurring in many cutaneous diseases are frequently characteristic, either in their form, time of appearance, degree of severity and persistence, and are of material assistance in arriving at correct conclusions as to diagnosis.

OBJECTIVE SYMPTOMS.

The importance of the study of the objective symptoms of cutaneous disease cannot be overestimated. It is only through a clear understanding of these features that a correct diagnosis can be made. The signs of skin disease are called lesions and it is usual to classify them as primary and secondary. They are sometimes called elementary and consecutive.

Elementary (or Primary) Lesions.—In describing the average size of cutaneous lesions it is more convenient to convey an approximate idea by comparison with familiar objects of relatively fixed dimensions than to state the measurements in fractions of a meter. The objects usually selected for this purpose, beginning with the smallest, are seeds of the poppy, mustard and rape; the pea, the coffee-bean, the bean, the cherry, the finger-nail, the chestnut, the egg of the hen and of the goose, and the orange. The point and the head of the pin are also frequently used for comparison.

Maculæ.—Macules (*spots, stains;* Fr., *taches;* Ger., *Flecke*) are circumscribed alterations in the color of the skin of varying size and shape, unaccompanied by elevation or depression. Macules may be congenital or acquired and may be the only cutaneous symptoms present, or may constitute only a part of an eruption. They are usually some shade of red, yellow, or brown, except occasionally, when they are white. They may be due to arterial or venous hyperemia, examples of which are seen in the exanthematous eruptions and in erythema hyperemicum; also in the halo which is seen surrounding a vaccine vesicle, which is called an *areola.* They may be due to escape of the coloring matter of the blood into the skin which occurs in the various purpuras. When these extravasations occur in linear streaks they are called *vibices;* in punctate and large extravasation they are termed *petechiæ* and *ecchymoses.* They may be due to pigment anomalies, illustrated by freckles and the macules of urticaria pigmentosa or to enlargement of bloodvessels in localized area, illustrated by acquired telangiectases, or to enlargement of bloodvessels in localized areas, or to hyperplasia of bloodvessels, illustrated by vascular nevi of the port wine type.

Macular stains are also seen in those who come in contact with dyes. Spots of various colors and figures are also produced by the intentional or accidental introduction of pigmented particles beneath the epidermis, as illustrated in the process of tattooing, or of explosion of gun powder. White macules are seen in vitiligo.

Extensive changes in the color of the skin are seen in association with several systemic diseases, such as yellow fever, cancer, chlorosis, albinism, Addison's disease, argyria and icterus. Subjective symptoms are rarely associated with macules. In occasional instances scaling occurs, as in pityriasis rosea. At times a macule becomes elevated and is then said to be a maculo-papule.

Papulæ.—Papules (Fr., *papules;* Ger., *Knötchen*) are circumscribed solid elevations of the skin varying in size from a pin point to a split pea. The base of the papule may be round or irregular. The top may be pointed, acuminate, conical, rounded, flat, or depressed in the center to form an umbilication. In color they may be bright or dark red, violaceous, brown, or dead white. They may be located anatomically about hair follicles or on any portion of the surface. They may be comparatively transitory or persistent. They may be scale covered, or surrounded by a collar of scales; a vesicle or pustule may develop on their surface, producing a compound lesion, or they may be transformed into other lesions of the skin.

The small plugs of epidermal debris that occur about hair follicles in keratosis pilaris, and the dried sebum that collects in the mouth of hair follicles coming from the sebaceous glands (comedone) are often called papules, although they are not, strictly speaking, such lesions.

Papules may be few or numerous, and be discrete or occur in patches. They may be accompanied by marked subjective sensations, or be entirely devoid of such symptoms.

Pomphi.—Wheals (*urticæ;* Fr., *plaques ortiées;* Ger., *Quaddeln*) are circumscribed, edematous, more or less transitory elevations of the skin varying in size from a pin-head to that of the palm or larger. Wheals are commonly evanescent lesions which appear and disappear quickly and are usually accompanied by itching, tingling, or pricking sensations. They are pinkish or darker red, or white, surrounded by an areola. They are usually pea to finger-nail sized elevations, oval or circular in contour. They may be few or abundant and occur as discrete lesions, or in patches, circles, or gyrations. Their rapid development and disappearance is characteristic.

"Giant wheals" may have the dimensions of an egg or cover a large part of a limb or the trunk. Occasionally they are complicated by vesicles or bullæ which develop on their summits, and while as a rule only transitory hyperemia remains after their disappearance, occasionally pigmentation is a sequel.

Wheal-like lesions frequently result from the bites of insects, reptiles, horses and dogs. The disease in which wheals occur classically is urticaria.

Tubercula.—Tubercles (*nodules;* Fr., *tubercules;* Ger., *Knoten*) are circumscribed, solid elevations of the skin varying in size from a pea to a cherry. The term tubercle is rarely used at the present time on account of its association with tuberculosis. The title here relates chiefly to the size of the lesion, but the term nodule is preferable. These lesions vary as to size, shape, color and development in much the same way that papules do. They differ from papules largely in their deeper situation in the skin and their size. They commonly occupy the deeper portions of the corium and the subcutaneous tissue. At times they are only slightly elevated above the cutaneous level, the major portion of the lesion being deeply situated. They frequently undergo necrosis and develop into ulcers and are then followed by cicatrices. They occur characteristically in syphilis, leprosy, sarcoma, and carcinoma.

Phymata.—Tumors (*tumors;* Fr., *tumeurs* Ger., *Geschwülste;*) are new growths of varying size and composed of any of the structures of the skin or subcutaneous tissue. They may be solid or more or less compressible, differing in size, shape and color. Some are benign, others malignant, and they may be located within or beneath the skin, or merely be attached to the latter and project from it a variable distance. In size they vary from that of a walnut to those of great volume and weight. They are not common in cutaneous diseases but are seen in fibroma, sarcoma and carcinoma.

Vesiculæ.—Vesicles (*phlyctenæ; phlyctenulæ;* Ger., *Bläschen*) are circumscribed elevations of the skin containing fluid and varying in size from a pin's head to a split pea. Vesicles may be discrete or grouped. They may be comparatively transitory, as in eczema, or of long duration, as in herpes. They may contain pus, blood or chyle. In color they are opalescent, yellowish, blood-tinged, or darker. They may be acuminate, round, conical, flattened or umbilicated. They may be tense or flaccid. Their content may escape through accidental or spontaneous rupture or by absorption. They may be transformed into pustules or bullæ. They may occur as secondary lesions on the apex of papules. They frequently dry and become crust-covered. Minute vesicles are sometimes seen as the external apex of larger accumulations of fluid beneath. Such lesions are seen in lymphangiectases. A moderate amount of itching may or may not be present; other sensations sometimes occur. Vesicles are found typically in herpes zoster, herpes simplex, varicella, dermatitis herpetiformis, eczema and other disorders.

Vesico-pustules and vesico-bullæ are intermediate forms of elementary lesions representing types designated by these names.

Pustulæ.—Pustules (Fr., *pustules;* Ger., *pustellen*) are circumscribed, elevations of the skin varying in size from a millet seed to a filbert and containing pus. Pustules, like vesicles, may be round, globoid, conical, or umbilicated and are frequently surrounded by an areola. They may be superficial or deeply situated. They may terminate by rupture or by desiccation. Their color is yellow, yellowish-green, or brownish-green, according to the admixture of the contents with blood. They may be situated on the free surface of the skin or about hair follicles. They may originate as pustules, or be transformed from vesicles. They frequently become crust covered, the crusts assuming the color of the contents of the pustule.

Pustules are seen in syphilis, variola, eczema, scabies, acne, some forms of dermatitis medicamentosa, and some other diseases.

Transitional forms between vesicles and pustules and papules and pustules are termed respectively vesico-pustules and papulo-pustules.

Bullæ.—Blebs (*blisters;* Fr., *bulles;* Ger., *Blasen*) are superficial or deep seated elevations of the skin containing fluid and varying in size from a split pea to a goose egg or larger. Blebs are similar to vesicles in many respects. They usually contain serum or lymph, or they may contain blood or pus, and are variously colored according to their content. They may be globoid, hemispherical, oval, crescentic, semicrescentic, or conical or may even be angular. They may be seated upon an apparently normal integument or upon an inflamed base, and may or may not present an areola. They may be tense or flaccid, persistent or easily ruptured. They may dry and form crusts, or degenerate into ulcers. They are seen classically in pemphigus, and are seen also in leprosy, erysipelas, syphilis, gangrene, and in scalds and burns.

Consecutive or Secondary Lesions.—**Squamæ.**—Scales (Fr., *squames;* Ger., *Schuppen*) are dried epithelial lamellæ, usually resulting from imperfect cornification. There is constantly in progress over the surface of the body physiological desquamation which is not perceptible in skins properly cleansed. Pathological scaling is a prominent feature of many cutaneous diseases. Scales may be fine, branny, dirty white or yellowish, or large, pearly white and shiny. They may be dry or fatty, few or abundant, firmly or loosely attached. They may be aggregated so as to resemble flaky pie crust, or exfoliate in extensive sheets, as from the entire palm or sole. They may fall spontaneously in such quantity as to cover the clothing of the patient or the bed linen. They may occur on an erythematous or non-inflamed skin as well as on papules and other lesions. They may cover an entire papule, in an imbricated manner, as in psoriasis, or form a firmly adherent covering, as seen in lichen planus, or form a collarette around the base of a papule, as in syphilis. They occur, as a rule, as the result of inflammation, but an exception to this rule is seen in ichthyosis.

Scales occur in addition to the above in dermatitis exfoliativa, eczema and several of the parasitic disorders of the skin. The scales occurring in pityriasis steatoides are flattened plates of dry sebum with epithelial debris.

Crustæ.—Crusts ("*scabs;*" Fr., *croutes;* Ger., *Krusten, Borken*) are the dried products of exudates induced by pathological processes in the skin. Crusts are composed of dried serum, pus, blood, epithelial debris and extraneous matter in varying combinations. Crusts may be yellowish, straw color, or reddish-yellow in hue when composed of dried serum. Yellow, greenish-yellow, or greenish when composed largely of dried pus. Brownish or darker colored when blood is admixed. At times they are comparable in appearance to gum, honey or Venice turpentine. They may be delicate and thin, bulky and thick, friable or mealy, firmly attached to the underlying structures or easily separable. They may have the form of the concavo-convex lid of the watch-case, or resemble the half shell of the oyster. They are formed on ulcers, both superficial and deep and are seen on the reddened and tender but largely healed epidermis. In size they vary from minute dimensions to lesions covering a large portion of the limb. The wide range in characteristics exhibited by crusts is noted between the superficial "stuck on" crusts of impetigo and the deeply seated and bulky crusts of the rupioid syphiloderm.

Crusts may be formed of dried sebum, in which case they are greasy to the touch, dirty yellow in shade, and usually situated on a slightly inflammatory non-infiltrated base.

Crusts are common in eczema, syphilis, impetigo, leprosy and many other diseases of the skin.

Excoriations.—Excoriations (*abrasions, erosions;* Fr., *excoriations;* Ger., *Hautabschürfungen*) are superficial solutions of continuity, usually involving portions of the skin, accompanied with itching and resulting from mechanical violence. Excoriations are most commonly produced by the finger-nails in itching dermatoses. At times they are produced by rubbing the skin with foreign objects. They occur as punctate, linear or irregular wounds of the skin, oozing serum or blood. Later, after desiccation, they form reddish-yellow or darker colored crusts. Occasionally these lesions are accompanied by those induced by secondary pyogenic infection.

Excoriations occur in essential pruritus independent of preceding lesions. As complicating lesions they are seen in eczema, scabies, prurigo and many other itching diseases. They are conspicuous in pediculosis corporis. A skin long scratched frequently becomes thickened and pigmented.

Rhagades.—Fissures (*cracks, rinæ;* Fr., *fissures;* Ger., *Hautschrunden*) are linear solutions of continuity, usually occurring in infiltrated portions of the skin. Fissures occur, as a rule, in a skin which has lost its elasticity and extensibility through infiltration and thickening. They may be dry or discharge a small quantity of serum or blood. At times they are covered with crusts. They occur most frequently where extensibility of the skin is essential to accomplish the necessary movements of the parts. They are seen about the mucocutaneous orifices of the body, about the small articulations of the hands and feet, as well as on the finger ends, palms and soles. They are frequently tender and painful and interfere with the occupation of the individual, or with walking. They may be superficial or comparatively deep, and vary as to length and outline. They are seen in eczema, syphilis, intertrigo, keratoderma and other affections.

Ulcers.—Ulcers (Fr., *ulcères;* Ger., *Geschwüre*) are losses of substance resulting from pathological processes involving the corium. Ulcers differ greatly in shape, size, color, edges, base and career. Every ulcer has an outline, a base, a floor, edges and secretion. The outline may be circular, crescentic, reniform, ovoid, serpiginous, horseshoe-shaped, or irregular. The base or underlying tissue may be soft, supple, infiltrated or indurated. The floor may be shallow, deep, excavated, cup- or funnel-shaped, "worm eaten," crateriform, sloughy, or glazed, covered with a secretion which may be tenacious or readily removed, granular, puriform or hemorrhagic. The edges may be clean-cut, have a punched-out appearance, undermined, everted, ragged, irregular, or contracting with a whitish inner border of advancing cicatrization. The secretion may be scanty, profuse, limpid, puriform, ichorous and odorless, or exhale an offensive smell. Ulcers may or may not be crust covered. They may be acute or chronic; insensitive, or productive of intense pain. They may heal by cicatrization or remain open indefinitely. Ulcers occur in syphilis, leprosy and carcinoma. They occur on the lower extremities as the result of secondary infection associated with congestion due to varicose veins; also in hypostatic congestion (bed-sores) and in cases of general debility with impaired resistance. Ulcers terminate after healing with cicatrization.

Cicatrices.—Scars (Fr., *cicatrices;* Ger., *Narben*) are new formations of connective tissue replacing tissue lost through destructive processes involving the corium. Scars usually follow ulcerative processes, in which case their contour is that of the preceding lesions. This is of diagnostic importance.

Scars may be minute or cover extensive areas. In the early stages they are red, later becoming pigmented or white. They may be attached to the underlying tissues, or be freely movable, depressed, or raised above the plane of the surrounding skin. They may be smooth, soft and pliable, or present an irregular surface and be indurated and inflexible. Scars following burns are characteristic and frequently undergo contracture, producing marked deformity. The normal lines of the skin, the orifices of sweat pores and ducts of sebaceous glands and the hairs are absent in scars. Subjective sensations are rarely present. At times itching or pain occurs. As implied in the definition given above, scars may result from any disease or injury of the skin that involves loss of connective-tissue elements of the corium. Scars or atrophic areas occur frequently without preceding ulceration in such diseases as lupus erythematosus, scleroderma, some cases of erythema induratum and syphilis, favus, and other disorders. Here the process is the result of absorption of pathological processes which have destroyed the deeper portions of the skin, or the result of pressure atrophy.

Unclassified Lesions.—To the several lesions defined above, Bazin adds, as elementary forms, the mucous patch of syphilis, the cuniculus or furrow produced in the skin by the *Acarus scabiei*, and the sulphur-colored crusts of favus. Among the elementary lesions of the skin, Brocq includes the gumma, or firm, deeply situated, often subcutaneous mass commonly degenerating centrally rather than, as may the tubercle, from without; while among the consecutive or secondary lesions of the skin the same author considers "lichenization" or "lichenification." These are terms chiefly employed by French writers to designate the changes in the skin produced by long-continued external irritation, the thickened and infiltrated integument assuming a yellowish-brown or reddish-brown tint, the exposed surface being studded with pinhead, pinpoint, or slightly larger, shining and flattened isolated elevations, with delicate furrows separating each from the other. These, however, are not general, but special features of individual disorders, and are best studied in connection with the latter.

The elementary lesions of the skin are termed by Auspitz *anthemata;* groups of such lesions, *synanthemata;* and, in accordance with common usage, generalized eruptions affecting the entire surface of the body, *exanthemata*. The word *erythanthema* is used to describe groups composed of several of the elementary lesions of the skin, as, for example, of papules, vesicles, and pustules, rising from a common reddened and hyperemic base.

In addition to the names of the lesions of the skin just enumerated, certain peculiarities of cutaneous symptoms are described in qualifying terms which require definition. They relate chiefly to the color, shape, distribution, and method or period of evolution of lesions as they are observed in individual cases. The more important of these terms, as used by modern writers, are alphabetically arranged below, with a brief explanation appended to each.

Abdominalis. Located on the abdominal surface.

Acquisitus. Acquired.

Acuminatus. Having a pointed apex.

Acutus. Of acute course.

Adultorum. Occurring in adult years.

Æstivalis. Occurring in the summer season.

Aggregatus. Collected in patches.

Agrius. Acute, or angry in appearance.

Albidus. Of whitish color.

Angiectaticus. Vascularized.

Annulatus. **Annularis.** In the form of a ring.

Apyreticus. Unaccompanied by fever.

Areatus. Occurring in areas.

Artificialis. Producible artificially.

Asymmetricalis. Of different distribution on the two lateral halves of the body.

Autumnalis. Occurring in the autumn.

Brachialis. Occurring on the surface of the arm.

Cachecticorum. Occurring in debilitated subjects.

Capitis. Occurring on the head, usually the scalp.

Cavernosus. Large chambered.

Chronicus. Chronic in course.

Circinatus. Of circular outline.

Circumscriptus. Having a definite contour.

Confertus. **Confluens.** Arranged in close proximity, with coalescence of lesions.

Contagiosus. Capable of transmission by contagion.

Corporis. Occurring on the surface of the body; employed usually to designate an eruption upon the trunk, as distinguished from that on the head or the extremities.

Crustosus. Crusted.

Crystallinus. Of crystalline appearance.

Diffusus. Irregularly disposed.

Discretus. Having isolated lesions.

Disseminatus. Disseminate; without regularity of distribution.

Eruption. Is used of the totality of all patches and lesions upon the person of one individual.

Erythematosus. Having a reddish blush.

Essentialis. Idiopathic.

Exfoliativus. Having a tendency to exfoliation or shedding of scales from the surface of the body.

Exulcerans. Exhibiting lesions with a tendency to superficial ulceration.

Facialis. Located on the face, usually as distinguished from the scalp.

Favosa. Displaying crusts of favus.

Febrilis. Accompanied by a febrile process.

Femoralis. Occurring on the surface of the thigh.

Fibrosus. Composed of fibrous tissue.

Figuratus. Having a figured appearance.

Flavescens. Of yellowish hue.

Foliaceus. Resembling a leaf or leaves.

Follicularis. Concerning the cutaneous follicles.

Fungoides. Resembling a fungus.

Furfuraceus. Exhibiting numerous fine, bran-like scales.

Guttatus. Of the size of a drop of water.

Gyratus. Having a serpiginous or gyrate outline, which is usually the result of a coalescence of imperfect circles or semicircles.

Herpetiformis. Vesicular or herpetic in type.

Hiemalis. Occurring in the winter season.

Humidus. Accompanied by moisture.

Hypertrophicus. Characterized by hypertrophy.

Hystrix. Having lesions projected or erected like quills.

Imbricatus. With crusts or scales overlaid like tiles.

Impetiginodes. Pustular.

Infantilis. Occurring in infancy.

Intertinctus. Distinguished by color.

Iris. Occurring in more or less distinctly defined concentric rings.

Labialis. Occurring upon the surface of the lip.

Lenticularis. Of the size of a small bean.

Lividus. Deeply colored.

Maculosus. Discolored.

Madidans. Characterized by moisture.

Marginatus. Having a defined margin.

Medicamentosus. Produced by external or (more commonly) internal medication.

Melanodes. Of blackish color.

Miliaris. Of the size of a millet-seed.

Mitis. Of mild, benignant type—the reverse of agrius.

Multiformis. Exhibiting simultaneously several types of elementary lesions.

Neonatorum. Occurring in the newborn.

Neuriticus. Having nervous association.

Nigricans. Of a black or blackish color.

Nodosus. With development of nodes or tuberosities of the surface.

Nummularis. Of the size of small coins.

Oleosus. Accompanied by an oily secretion.

Palmaris. Occurring on the palms.

Parasitarius.
Parasiticus. } Produced by an animal or a vegetable parasite.

Patch. The aggregation of several isolated or confluent lesions.

Phlegmonosus. Accompanied by deep-seated inflammation.

Phlyctænoides. Characterized by groups of small vesicles.

Pigmentosus. Accompanied by pigmentation.

Pilaris. Related to the hair.

Plantaris. Situated on the soles of the feet.

Planus. Flat.

Polymorphous. The Greek equivalent of the Latin *multiform.*

Præputialis. Situated upon the prepuce.

Progenitalis. Situated upon the exposed mucous surfaces of the genitalia.

Pruriginosus. Accompanied by itching.

Pubis. Located upon the skin or hairs of the pubes.

Punctatus. Occurring in dots or points.

Rhagadiformis. Fissured, or tending to produce fissures.

Rosaceus. Having a rosy or pinkish hue.

Ruber. Red; usually dark red in color.

Scutiformis. Having the shape of a shield.

Sebaceus. Concerning the sebaceous glands or their secretion.

Senilis. Occurring in advanced years.

Serpiginosus. Literally, creeping; advancing in irregular gyrations.

Siccus. Dry; unaccompanied by moisture.

Solitarius. Exhibiting an isolated lesion, or with isolated lesions.

Symmetricalis. Similarly distributed on the lateral halves of the body.

Toxicus. Poisonous.

Uniformis. Exhibiting lesions all of one type.

Universalis. Affecting the entire surface of the body.

Urticatus. Accompanied by wheals.

Uterinus. With association of uterine disorder.

Variegatus. Exhibiting several distinct colors.

Vasculosus. Accompanied by vascular development.

Vernalis. Occurring chiefly in the spring of the year.

Versicolor. Exhibiting several shades of the same color.

Vulgaris. Of the usual or commonly observed type.

III. GENERAL ETIOLOGY.

SEVERAL features must be considered in the study of the causation of cutaneous diseases. A large number of the latter are simply expressions on the skin of a constitutional disease (the exanthemata: syphilis, pellagra, the xanthomata, and other diseases of this type). Others are purely cutaneous diseases, the entire process, including the cause, being limited to the skin (scabies, pediculosis, impetigo, and other parasitic diseases whose causative factor has been demonstrated). Still others, while purely skin diseases, have general conditions which contribute either to their cause or continuance (acne rosacea, seborrhea, eczema). Finally, many are cutaneous expressions of constitutional disturbances due to toxins other than those produced by a specific infection (acne, erythema multiforme, the purpuras, scorbutus, etc.).

A very important matter in relation to the general cause of cutaneous disease is the question of individual susceptibility. Of the many persons exposed to poison ivy, only a few develop a dermatitis venenata from such exposure; and a small proportion of these have their skins so sensitized that many and varied external and internal irritations will induce a subsequent dermatitis or eczema, which before the attack of the original trouble would have been inoperative. Idiosyncrasy has been the term employed to indicate this susceptibility from ingestion of certain drugs. The workers in anaphylaxis have thrown some light on this subject, but much more is needed. The fact remains that such susceptibility exists and must be taken into consideration.

Environment.—Environment is an important factor in the general etiology of cutaneous disease. Under this topic is immediately suggested the care or lack of care of the skin. Personal hygiene is very important, both directly and indirectly, as a factor in the production of skin diseases. Bathing, when overdone or employed insufficiently, often produces skin disease. In infants that are over-bathed intertrigo, eczema, and the like are often caused. The temperature of the water is important, as is demonstrated frequently in the excessive use of hot baths by young women, which is a factor in the production or aggravation of acne and seborrhea. The lack of bathing, as exhibited in public practice, predisposes to all parasitic diseases. It is a matter of common experience that pus-infection, some types of eczema, impetigo, and other diseases of this class occur much more frequently under these conditions. Poor air is indirectly a factor. This probably produces its effect through the lowering of the general vitality, rather than by direct local action. In the crowded apartments of the lower classes, the lack of fresh air plays a part in all diseases, including those peculiar to the skin. Clothing plays its part through local irritation, when the material used next to the skin is either rough or poorly dyed. Again, when too warmly clad, the skin becomes tender, and therefore more liable to external and internal irritation. As a general rule, woolen clothing should not be worn next to the skin; cotton, linen or silk is preferable.

Occupation.—Many dermatoses are due exclusively to the occupations of men and women. The workers in dyes, in chemicals, and in drugs suffer in one way; the men who handle tiles, bricks, mortar, or clay in another; the baker, the confectioner, the cook, the laundress, the green-grocer, the seamstress, the shoemaker, the carpenter, and the machinist each have their forms of erythema, dermatitis, keratosis, or induration. Similarly, those whose faces are much exposed, as the wheelmen of vessels, tramcar-drivers, locomotive-engineers, and day laborers, exhibit symptoms in that region. Butchers, wool-workers, cattlemen, and sheep-shearers are liable to contract glanders, ringworm, or malignant pustule. Those who handle the bodies of the dead are prone to tuberculosis of the hands (anatomical tubercle), and those compelled to stand much of the time are exposed to the consequences of varicose veins of the legs and resulting eczema of that region. Finally, the dermatitis of the hands of laboratory workers produced by formalin, and of surgeons and nurses produced by the irritating substances used to cleanse the hands preliminary to surgical work, are of comparatively frequent occurrence.

Scratching is a potent factor in inducing and aggravating cutaneous disease. In pruritus, scratching frequently produces a traumatic dermatitis of varying grades. The aggravation of an eczema by scratching is a matter of daily observation. In this connection, other agents producing trauma with deleterious effects upon the skin should be mentioned, as the bites of insects, such as lice, fleas, bed-bugs; of animals, such as horses, dogs, and cats; and of serpents. In addition to the purely traumatic effect of all of these may be added the introduction of a toxic agent; and, finally, self-inflicted injuries must be recognized.

Food.[1]—Food is another important factor, as many toxic erythemas, urticarias, and other angioneurotic disturbances, some forms of eczema and possibly pellagra are produced through errors in diet. These disorders are produced in several ways. Toxic substances forming in the food itself may be absorbed and be the factor. The reflex irritation from indigestible articles is the active factor in a few instances. Idiosyncracy is an important factor in many others in which neither of the above plays any part. Recent work appears to account for this hypersensitiveness by anaphylaxis.[2]

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[1] Chipman: Trans. Sec. on Derm., Am. Med. Assn., 1916, p. 49; "Dermatologic Dietetics."

[2] *Cf.*, McBride and Schorer, Jour. Cut. Dis., 1916, xxxiv, p. 70; "Erythematous and Urticarial Eruptions Resulting from Sensitization to Certain Foods." Strickler, New York Med. Jour., 1916, July 29; "Anaphylactic Food Reactions in Skin Diseases, with Special Reference to Eczema."

Sensitization to certain protein substances is an important factor in the production of cutaneous disease.[1] Goldberger[2] has demonstrated the importance of proper food in the prevention of pellagra. Drugs of various kinds very commonly produce cutaneous eruptions. A reference to the chapter on dermatitis medicamentosa will demonstrate this fact. Most of the drugs, while capable of producing eruptions, do not produce characteristic ones. The iodids, bromids, and arsenic, however, are exceptions.

Climate and Seasons.—Certain diseases exist only or mainly in tropical countries. The list of exclusively tropical diseases is decreasing, however, and particularly during the last few years. Pellagra, hitherto practically limited to the so-called warm countries, is now fairly prevalent in portions of the United States. Certain other diseases occur largely in or are made worse by cold weather.[3] In this connection, the seasons also play an important part. In winter, psoriasis, pruritus hiemalis, and certain types of eczema flourish; in the spring and autumn, erythema multiforme and pityriasis rosea are prevalent; while during the summer, dermatitis venenata, hydroa æstivale, certain types of eczema, dysidrosis, and sudamen are of frequent occurrence.

Heredity.—In certain diseases, such as ichthyosis, angioneurotic edema, xeroderma pigmentosum, congenital lymphodermia, keratodermia, and others, heredity is a factor. It is difficult to state whether some of these are hereditary disorders or are family diseases. It is uncommon to find xeroderma pigmentosum in different generations, but it is very common for several members of one family to be affected. On the other hand, ichthyosis apparently occurs in many generations, and in addition in several members of one generation. Keratodermia, on the contrary, may be limited to one member of a family, and yet examples occur where heredity seems to be important. In tuberculosis of the skin, a predisposition in certain families is assumed, but infection with the bacillus of tuberculosis must occur, though the soil may have been prepared by heredity. In very many other cutaneous disorders the influence of heredity remains a matter of opinion, which at present cannot be proven in either direction.

Race.—Very few diseases are prone to attack a single race. It is well known, however, that keloids occur often and grow to immense proportions in colored people, and that among these leucoderma is also common. Again, idiopathic multiple pigmented sarcoma is largely limited to the Jewish race. Most other disorders appear to occur without much selection as to race.

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[1] For recent views on protein sensitization see papers by Harvey P. Towle, "Protein Sensitization in the Production of Skin Diseases," Archives of Derm. and Syph., 1920, ii, p. 531; and Walter J. Highman and Jeffrey C. Michael, "Protein Sensitization in Skin Diseases: Urticaria and its Allies," Ibid., p. 544. These papers opened a discussion on this subject before the American Dermatological Association in 1920, and A. J. Markley, "Anaphylactoid Dermatitis Due to Direct Contact with Animal Epidermal Structures," Ibid., p. 722.

[2] Jour. Am. Med. Assn., 1918, lxxi, p. 944.

[3] *Cf.* Hyde: "On Affections of the Skin Induced by Temperature Variations in Cold Weather," Chicago Med. Jour. and Exam., 1885, l, p. 187, and 1886, lii, p. 116; Corlett, Jour. Cut. Dis., 1894, xii, p. 457, and Jour. Amer. Med. Assoc., 1902, xxxix, p. 1583.

Age.—Infancy is characterized by the occurrence of congenital disorders, in addition to which several other cutaneous diseases are especially liable to occur. Among the commonest diseases may be mentioned the following: nevi, xeroderma pigmentosum, xanthomata, ichthyosis, epidermolysis bullosa, sclerema neonatorum, urticaria pigmentosa, ringworm (the small-spored variety), favus, lupus vulgaris, certain types of eczema, particularly pustular, and pus infections. At puberty, acne, seborrheic dermatitis, and other sebaceous-gland disorders occur; while at a more advanced age epitheliomata are common.

Physiological State.—At the menstrual epoch, certain diseases are made worse, while others apparently occur only at that time. Eczema recurring regularly at this period is occasionally noted. Acne and rosacea may be aggravated. In pregnancy, herpes gestationis may occur, and if so recurs with each subsequent pregnancy. Pruritus, particularly localized in the genital region, urticaria, and chloasma are all apparently induced by this state. Other cutaneous disorders, such as acne and eczema, may be either aggravated or improved. At the menopause, pruritus and hypertrichosis not infrequently occur.

The relationship of cutaneous diseases to the so-called general disorders is a matter that has been studied much of late. Many generalizations are made. Specific connection between a general disorder or condition and cutaneous disease is rarely demonstrated. Xanthoma occurs with diabetes, as do also pruritus, both generalized and local, and genital eczema. That there is some connection between the gouty state and eczema there can be little doubt in certain cases. Again, urticaria is a fairly common accompaniment of malaria, and in rheumatism purpura is occasionally found. The extreme pruritus accompanying jaundice induced by hepatic disease is a matter of common observation.

Internal Secretions.[1]—Various disorder and disturbances of the glands of internal secretion are connected etiologically with cutaneous disease. In connection with the thyroid, myxedema is the most striking example. Changes in the thyroid also occur in Graves's disease and scleroderma. The peculiar pigmentation seen in Addison's disease is an example of the derangement of the suprarenal glands.

Transmission of Infectious Diseases.—Infectious diseases with cutaneous manifestations are communicable from man to man, or from animal to man, in a variety of ways. Thus, transmission may occur by actual contact, either with the infected person or animal, or through an intermediate object, such as a drinking cup, roller-towel, or article of clothing. This mode of infection is an important factor in the spread of the tineas, a variety of tuberculosis of the skin known as the "anatomical wart," the acute eruptive fevers, lepra, and, most important of all, syphilis. Or it may take place through inspired air which has become infected by droplets of sputum from the respiratory passages of tuberculous patients, or by desquamation from scarlatina. In these last-named diseases ingestion of contaminated food, particularly milk, is a well-recognized mode of transmission.

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[1] Foerster: Jour. Cut. Dis., 1916, xxxiv, p. 1, "The Relation of Internal Secretions to Cutaneous Disease," and McEwen, ibid., p. 15. These papers opened the discussion on this subject before the American Dermatological Association in 1916. Also see Engman, Jour. Amer. Med. Assoc., 1919, lxxiii, p. 1565; "The Skin: A Mirror to the System."

Many diseases are transmitted by the medium of insects (particularly the fly, the bed-bug, the louse, the flea, and the mosquito), which attack the skin and deposit in the solutions of continuity which they produce bacteria or other noxious germs derived from foreign bodies on which they previously have alighted. In this connection, relative to diseases with cutaneous manifestations, may be mentioned Rocky Mountain spotted fever, transmitted by a tick; typhus, by the louse; and lepra, by the bed-bug.

Parasitic Diseases.—Among other important animal parasitic diseases may be mentioned scabies and pediculosis, and as representative vegetable parasitic disease may be mentioned ringworm of the scalp and other areas. In addition to these several cutaneous disorders are due to a variety of microörganisms, among which are the bacilli which induce cutaneous tuberculosis and lepra, pus cocci responsible for several forms of impetigo, ecthyma, eczematoid dermatitis, and others. The number of parasitic diseases is comparatively small and the danger of their dissemination is exaggerated by the layman. The possibility of transmitting scabies or syphilis or other affections by handshaking is remote. On the other hand, there is but little doubt that the public roller-towel, public drinking-cups and glasses have been responsible for the dissemination of contagious diseases. Promiscuous kissing is a not uncommon factor in the dissemination of syphilis.

Focal Infections.[1]—The connection between various focal infections of the body and rheumatism has been emphasized within the last few years by Billings and others. The close association of erythema nodosum and rheumatism has been recognized for years. Other cutaneous affections such as recurring erythema both simple and scarlatiniform, multiform erythema and purpura have also been associated with rheumatism. The possibility, therefore, of a connection between these disorders and an infection at some point in the body is evident. A large number of cutaneous diseases have been demonstrated to be associated with localized infection such as that occurring in tonsils, in pyorrhea, in appendicular abscess and infection in the genito-urinary tract. The connection existing between the cutaneous disorder and the focal infection is difficult to prove. In many instances remarkable recoveries have been reported following the removal of the focus or foci of infection. That focal infections play a part in the causation of cutaneous disease is certain, but the number of such diseases and the extent of their role is as yet undetermined. Careful study by a large number of observers over a considerable time will be required to demonstrate the etiological position of focal infections.

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[1] Mackay, Dominion Dental Journal, 1917, v, 29, p. 211, Infection in Cutaneous Disorders; Ravitch, Jour. Amer. Med. Assoc., 1916, 67, p. 430, Focal Infection in Relation to Certain Dermatoses; Ravitch and Steinberg, Ibid., 1918, 71, p. 1273, Focal Infection; Chipman, Trans. Sec. on Derm., Amer. Med. Assoc., 1918, p. 128, The Etiology of Lichen Planus; Galloway, Practitioner, London, 1912, 88, p. 67, Cutaneous Infections in Rheumatism; Chipman, Jour. Cut. Dis., 1917, xxxv, p. 646, Focal Infection in the Etiology of Skin Disease; Geber, Derm. Zeitschr., 1912, xix, p. 782, Ueber das Wesen der sogenannte idiopathen Erythem.

IV. GENERAL PATHOLOGY.[1]

THE pathological processes occurring in the skin are similar in many diseases to those occurring in other organs; but, owing to complicated structure and functions, the integument has a pathology peculiar to itself. Various pathological conditions, such as inflammation, hyperemia, anemia, hypertrophy, atrophy, degeneration, and neoplasms, are found in the skin, as in other organs of the body. Some diseases, such as the toxic erythemas, are merely cutaneous manifestations of an internal disorder which often exhibits no demonstrable internal lesions; in others, such as lupus vulgaris, the pathological and clinical manifestations are for the most part limited to the skin. Again, in diseases such as syphilis, similar pathological changes may be noted both in the internal organs and in the skin.

Bacteria.—The skin furnishes a habitat for a large number of bacteria, both pathogenic and non-pathogenic. From the normal skin may be collected a number of varieties of cocci, bacilli, and yeasts. Many diseases of the skin are demonstrably of bacterial origin, while others are probably due to specific microörganisms not yet recognized. Schizomycetes (tuberculosis, leprosy), streptotricheæ (actinomycosis), blastomycetes (blastomycosis cutanea), hyphomycetes (favus, "ringworm") are all concerned in the production of diseases in the skin or its appendages. Animal parasites are responsible for several disorders (scabies, pediculosis).

Hyperemia.—Hyperemia in the skin may be active or passive, local or general, transient or persistent. On account of the conditions which may be associated with hyperemia, it plays an important part both in cutaneous and general pathology. Galloway[2] has emphasized the importance of erythema as an indicator of disease.

Anemia.—Anemia may be general or local. It is not a frequent factor in the production of cutaneous disease. Generalized anemia is a symptom of several diseases of the blood. Local, transient anemia occurs in urticaria and when cold is applied to the integument.

Inflammation.—Some of the many phases and pathological changes of the process recognized as inflammation are present in the majority of cutaneous diseases. Primarily, there occurs vascular dilatation, with leukocytic infiltration and exudation of plasma. The leukocytes, attracted by positive chemotaxis to the point of irritation, either remove the offending material (microörganisms, etc.) by phagocytic action, or themselves are overcome, undergo fatty degeneration, and become converted into pus-cells. The chemotactic agent may be a mechanical, chemical, or thermic irritant, or its cellular products. The toxins of microörganisms may be effective. The plasma dilutes the toxins, and by depositing fibrin through the action of a ferment helps limit the process. Varying with the degree of the reaction and its attendant conditions, numerous secondary epidermal changes occur.

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[1] For a more complete discussion of the pathology of the skin, see MacLeod, Pathology of the Skin; Unna, Histopathology; Darier, La Pratique Dermatologique. pp. 67-136.

[2] Brit. Jour. Derm., 1903, xv, p. 235.

Histology.—The epidermis and corium, being unlike in development and structure, undergo different pathological changes.

The epidermis is composed of epithelial cells in various stages of evolution, from the columnar, nucleated, and comparatively highly differentiated cell of the basal layer of the rete mucosum, to the flat and lifeless external cells of the stratum corneum. A knowledge of the normal process of evolution of these cells is necessary to an understanding of the changes which necessarily must occur in morbid conditions when the normal course of evolution is interrupted by some mechanical, chemical, or other agency. Each cell progresses from the basal layer of the rete through the several strata above until it reaches the superficial part of the stratum corneum, having on its way passed through various stages and performed different functions. After completing its cycle of existence, it is finally cast off.

In the basal layer are situated the mother-cells of the epidermis. They are columnar in shape, contain nuclei and pigment, receive the termination of non-medullated nerve-fibrils, and have extending from them prolongations of protoplasm called prickles. As they progress upward through the rete, they become gradually flattened, no longer contain pigment (in the white races), and on reaching the granular layer are filled with granules of keratohyalin, upon the perfect formation of which depends the normal process of cornification. Farther up, the cells become homogeneous and lose their keratohyalin, but acquire eleidin in the stratum lucidum. In the lower part of the stratum corneum their nuclei disappear and a horny substance, termed keratin, is formed, to which substance this layer owes its hardness. Here also some fat appears. Still more externally, the cells become entirely flat and lifeless, and eventually are shed.

Hyperkeratosis—Acanthosis.—One or all of the layers of the epidermis may be involved in pathological processes, depending upon the character of the change and its cause. When there is overgrowth (hypertrophy), either local or general, of the stratum corneum, it is designated as a hyperkeratosis, examples of which are seen in keratodermia and ichthyosis.

By acanthosis (Unna) is meant a benign hypertrophy of the rete, in which the fibrillary structure of the cell is retained. Acanthosis occurs in all the infective granulomata, including syphilis and tuberculosis. Malignant hypertrophy of the rete occurs in epithelioma, in which affection the normal rete-pegs are not only enlarged and elongated (acanthosis), but there are also rupture of the basal layer and irregular infiltration into the corium of epithelial cells, which lose their fibrillary structure and often become so changed as to resemble cells of mesoblastic origin.

Atrophy.—Atrophy of the cells of the epidermis occurs under various conditions. It may be caused by pressure, either external (as from a truss) or internal (neoplasm beneath the skin). It is found commonly in the senile skin, and is marked in cases of diffuse idiopathic atrophy of the skin.

Parakeratosis, Production of Vesicles, Bullæ, and Pustules.—Edema occurring in and between the rete-cells interferes with the formation of keratohyalin in the granular layer, causes the cells of the stratum corneum to appear swollen and moist and to retain their nuclei, and prevents the formation of keratin. This condition is termed "parakeratosis" (Unna), and is found in typical development in eczema and psoriasis. When the edema becomes greater, collections of fluid form, usually in the rete, and thus vesicles are produced. They are called "parenchymatous" when the early edema is intracellular, or "interstitial" if it be intercellular. The balloon cell of Unna is found in the vesicles of herpes zoster, varicella and variola. It is a large epithelial cell whose protoplasm has been transformed into a homogeneous mass that strongly attracts acid dyes. It frequently contains two to twenty or more nuclei. Unna considers the balloon cell the result of colliquation degeneration of the prickle cell. (See Pathology of Herpes Zoster, also see Pathology of Eczema for more complete description of vesicle formation.) Vesicles may be located superficially in the rete, as they usually are in eczema; or deeper, as in dermatitis herpetiformis; or beneath the epidermis, as occasionally happens in herpes zoster. Vesicle-formation is dependent not only on the mechanical separation of the cells by edema, but also upon the presence of toxic and other substances in the lymph, which may produce separation and disintegration of the epithelial cells, and thus leave spaces. Bullæ similarly are formed and located, and differ from vesicles chiefly in being larger. A typical bullous disease is pemphigus. When a large number of leukocytes collected in a chamber by chemotactic or other action have undergone fatty and other degenerative changes, the lesion becomes a pustule. When edema is long persistent, such as occurs when the leg is the seat of varicose veins, the epidermis is destroyed entirely and ulceration results.

Epithelial Degeneration.—The cells of the epidermis are subject to degenerative processes, the one most studied being of the "hyaline" type. This occurs in carcinoma and also in several other diseases, but is not, as once was believed, pathognomonic (see cellular degenerations of the corium). Degeneration occurring in epithelial cells exposed to *x*-rays, though not definitely classified, is pronounced and important. The nucleus as well as the cellular protoplasm is affected. The cell is swollen, stains poorly, becomes vacuolated, and eventually completely disintegrates and is carried away by leukocytic action during the period of reaction.[1]

Fibrous and Cellular Structure of the Corium.—The corium is mesoblastic in origin, and is composed of fibrous tissue and cellular elements. The white fibrous bundles are called collagen, while the yellow elastic fibers are termed elastin. The cells found normally in the corium are connective-tissue, mast-, and vacuolated cells. As cellular pathology is so important in cutaneous disease, some knowledge of the minute structure of normal and pathological cells is important.

The common types of connective-tissue cells are large, spindle-shaped cells, which vary both as to size and shape. They have extending processes, which connect with those of neighboring cells. The nucleus is surrounded by a membrane, is usually either oval or round in shape, and is said to be *vesicular* on account of its open appearance, which is due to large spaces found between the chromatin threads. This open structure causes it to stain less deeply than the more compact nucleus of the mononuclear leukocyte, with which it is often confounded. In young connective tissue the cells are small and more or less oval, have a nucleus as above described, are surrounded by cell-protoplasm, and are termed *fibroblasts*. Other and less common varieties of connective-tissue cells are described by Unna as *plate-cells*.

Vacuolated cells of the corium have nuclei similar to those of ordinary connective-tissue cells. The cell-protoplasm presents spaces or vacuoles, but has no processes extending from it. On account of mitoses occurring in these cells, and because their apparent function is that of reproduction and not of evolution into connective tissue, MacLeod suggests that these may be the mother-cells of the corium, being thus analogous to the cells of the basal layer of the epidermis.

Mast-cells in the corium resemble other connective-tissue cells, but differ from them in that they contain a number of basophilic granules. They are discussed more fully in connection with the pathological cells of the corium.

Pathological Cells of the Corium.—**Plasma-cells.**—Before Unna described the cell now generally recognized as the plasma-cell, at least two classes of cells were so denominated. The term is now restricted to cells which vary in size from that of a leukocyte to that of a cell two or three times as large. They are rounded or oval in shape and contain a large amount of protoplasm. The nucleus is usually eccentrically placed and corresponds in shape to that of the cell. It may be vesicular in appearance, or again several deeply stained masses of chromatin may be arranged about its border. Two nuclei are occasionally present. A cell having a similar nucleus, but containing a small amount of protoplasm, is found abundantly in tuberculosis, but is considered by many to be a lymphocyte. Plasma-cells are found abundantly in the infective granulomata, and to these cellular infiltrations Unna applied the term *granuloma*. Unna maintains that plasma-cells originate from connective-tissue cells, while Jadassohn, Councilman, Krompecher, Schottlander-Vmarschalko, and others believe that they arise from leukocytes. Krompecher, Vmarschalko, and others agree that these cells evolve into connective tissue, thus admitting the formation of connective tissue from leukocytes.[1] Plasma-cells are studied best when stained with polychrome-methylene-blue (Unna), or Pappenheim's compound stain of pyronin-methyl-green. In the former, metachromatism is shown by the nucleus taking a blue color, while the protoplasm is stained a blue violet.

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[1] Scholtz, Archiv, 1902, lix, pp. 87 and 241. Abstr. Brit. Jour. Derm., 1902, xiv, p. 397.

Giant-Cells occur in typical development in tuberculosis, but are found to a degree in syphilis, and cells resembling them may be noted in several chronic inflammatory diseases of the skin. The tubercular giant-cell may be round, oval, or irregular in shape, depending somewhat on its surroundings; as, for example, the presence of collagen, elastin, etc. They vary in size from two to three to many times the dimensions of a leukocyte. They contain nuclei which are similar to those of plasma-cells; and which may be arranged at one or both ends or sides, or completely around the periphery of the cell, and may number from a dozen or less to more than a hundred in a single cell. They stain deeply, thus making a contrast with the poorly stained center of the cell, which presents a homogeneous protoplasm, As to their origin, several theories are advanced. One is that they are formed by the rapid proliferation of the nuclei in a single cell without corresponding division of the protoplasm. A second is that a number of cells surround some irritant, such as tubercle-bacilli, and coalesce, thus producing the multinucleated giant-cell. The question whether the giant-cell originally comes from connective-tissue cells or from leukocytes cannot be answered until the origin of the plasma-cell has been determined.

Mast-Cells occur to some extent in the normal corium, and are found in increased numbers in some diseases, including the infective granulomata, in which they are not specially significant. In urticaria pigmentosa, however, their increase is so marked as to be pathognomonic. They may be produced rapidly, as was demonstrated by Gilchrist,[2] who noted that they formed synchronously with an urticarial wheal. They may assume the shape of a connective-tissue cell, plasma-cell, or lymphocyte, and may originate apparently from any cell found in the corium. Their chief characteristic is the presence of basophilic granules in the protoplasm. Mast-cells of the corium correspond in staining reactions to Ehrlich's mast-cells of the blood, but it does not follow that those present in the cutis come from the blood. They are demonstrated best by stains having metachromatic properties, such as polychrome-methylene-blue (Unna), which stains the nucleus blue and the granules red.

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[1] For full consideration of the cells of chronic inflammation, including plasma-cells and mast-cells, the reader is referred to a critical review of the literature by Williams, Amer. Jour. Med. Sci., 1900, cxix, p. 702; a series of papers by Pappenheim, and by Almkvist, Monatshefte, 1901–2; Maximow's monograph, Ziegler's Beiträge, Suppl. v, 1902; and a review of the subject by Whitfield, Brit. Jour. Derm., 1904, xvi, pp. 7 and 63.

[2] Johns Hopkins Hosp. Bull., 1896, vii, p. 140.

Degenerations Occurring in the Corium.—Hyaline degeneration similar to that occurring in epithelial cells in carcinoma is found also in the corium in sarcoma, in rhinoscleroma, in syphilis, and in other affections. It produces a homogeneous material in the cellular protoplasm, which is acidophilic in reaction, and, owing to its semifluid character, forms round globules. Hyalin is stained orange-red by Van Gieson's method.

Fatty degeneration occurs in several conditions in the skin, and is well represented in xanthoma. Here are found variously sized granules within a large cell, known as the xanthoma cell, which is characteristic histologically of the disease. This cell is the product of a connective-tissue cell in the multiplex varieties, while, according to Pollitzer,[1] in xanthelasma it results from degeneration of muscular tissue.

Mucoid degeneration is found in the "Mikulicz cells" of rhinoscleroma and in the lepra-cells of lepra. In both it occurs as a homogeneous mass, within which the specific bacilli are found.

Edematous degeneration occurs in the cells of the corium, which is the seat of marked edema. They appear swollen, stain poorly, and contain fluid. This form of degeneration is seen in tissue reacting after exposure to actinic and Röntgen rays.

Crenation degeneration is found in granuloma fungoides, and is evidenced by the cell becoming irregular and toothed. Eventually, the cell entirely disintegrates.

In addition to the cellular degenerations described above, several degenerative processes occur which affect the collagen and elastin.

Myxomatous degeneration, in which a peculiar jelly-like substance containing mucin results from collagenous degeneration, is found in sarcoma and myxedema. This substance is basophilic in reaction and is stained by any of the metachromatic dyes.

Colloid degeneration in the skin is comparatively rare. It occurs in the disease termed colloid milium. It consists of a homogeneous degeneration of the fibrous elements of the corium. The exact chemical composition of the colloid material is not known. It is stained yellowish-red by Van Gieson's method.

Other degenerations occur in the corium, in which collagen and elastin are concerned, and these are demonstrated chiefly by the staining methods described by Unna,[2] and are termed basophilic collagen, collastin, collacin, and elascin.

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[1] Jour. Cut. Dis., 1897, xv, p. 367; N. Y. Med. Jour., 1897, lxv, p. 679.

[2] Monatshefte, 1894, xix, p. 465.

V. GENERAL DIAGNOSIS.

The successful management of cutaneous disease necessarily demands a correct diagnosis. Many such diseases may be recognized by a comparatively superficial examination, but experience demonstrates that even apparently simple disorders may be complicated by conditions that would be overlooked without careful, systematic examination. Many disorders are of unknown etiology, and though their nature may be recognized, their successful management depends much upon facts gained by thorough examination of the individual. The past personal history in general, with the family history in many instances, should be taken, together with the history of previous cutaneous disorders, including that of the one under consideration.

History.—While the history in many instances might be misleading, facts are frequently obtained which are of great value. The discovery of past digestive, respiratory, circulatory, genito-urinary or nervous system disturbances, or defects in elimination, assimilation and nutrition, any of which might be causative or at least contributing factors in the disorder would be an aid in diagnosis.

Inquiry should be made concerning the occurrence of disorders such as rheumatism, tuberculosis, diabetes, the acute exanthemata, tonsillar and other focal infections, any of which might be concerned in the production of the present cutaneous disorder and, therefore, would be of assistance in arriving at proper conclusions. In the case of possible syphilitic infection the past history relative to cutaneous eruptions and other manifestations of the disease, including in women the history of miscarriages, are naturally of importance. The personal habits and surroundings of the individual may have a bearing on the production or continuation of the disease. The history of former skin diseases is important, and also the past history of the disorder under consideration. It should be ascertained whether this is the first attack and, if not, a description of previous attacks is to be elicited. It is well to ascertain from the patient what, in his opinion, was the cause of the disorder and whether any of his associates are similarly affected, the appearance when first seen, and any changes in character and type since its appearance, its duration, the rapidity and regularity or the reverse of the progress of the disease, the effect of outside influence upon the disorder, and the subjective symptoms.

Certain diseases occur only in childhood, others are more prevalent then, while many others never occur at this time. For example, eczema and urticaria are the common diseases, while such disorders as ichthyosis, urticaria pigmentosa and xeroderma pigmentosum begin at this time. At the age of puberty disorders of the sebaceous glands, such as acne, are prevalent, while in after years epithelioma and changes induced by age characterize this period.

Only a few disorders occur particularly in the different sexes, though many occur in the majority of instances in one or the other. As stated in the section devoted to etiology, race or nationality might be of some assistance. Diseases such as pemphigus, multiple and pigmented hemorrhagic sarcoma (Kaposi) occur more frequently in the Jewish race and keloids in the colored. The occupation may aid, as many cutaneous disorders, particularly the trade or professional eczemas are induced primarily by the occupation.

The Eruption.—Concerning the eruption various features are important: the distribution, configuration, color and the lesions.

Distribution.—Certain diseases select particular areas and not infrequently the nature of the disease is intimated by the region in which it occurs. Only a few of the more important of these factors can be mentioned. A more or less generalized eruption occurring suddenly suggests, aside from the exanthematous diseases, a drug eruption, or a toxic eruption from some other cause. A similar distribution of long duration might be induced by several disorders, such as general exfoliative dermatitis, generalized lichen planus, eczema and some others. An eruption limited to a certain area, particularly on one side of the body, suggests a local irritant in most instances. Occasionally a localized disorder is limited in its distribution not so induced (herpes zoster). An acute eruption limited to the forehead or neck, or a recurrent eruption in these areas suggests a chemical irritant such as is found in hair dyes and died furs. A disorder limited to the scalp, other than the various alopecias, is frequently seborrheic dermatitis or eczema or, if in a child, some type of ringworm or staphylococcic infection. If accompanied by scar formation and particularly in adults, lupus erythematosus, syphilis, pseudo pelade and folliculitis decalvans are to be considered.

The most common disorders largely limited to the neck are eczema, lichen simplex chronica, the latter occurring on the back of the neck below the hair line and extending into the scalp, and dermatitis papillaris capillitii (Kaposi), also at the back of the neck. The pigmented syphiloderm is found largely at the sides of the neck and in the same situation, extending on to the shoulders, the atrophic form of lichen planus is most frequently seen.

Among the disorders limited to the axillary and genito-crural regions epidermophyton infection, pediculosis and eczema are most common. Furunculosis in the axillæ and intertrigo in the genito-crural region in fleshy subjects are quite common.

On the trunk, particularly the upper portion, pityriasis rosea, psoriasis, tinea versicolor, the macular and other syphilodermata, and seborrheic dermatitis, are the chief affections seen. About the lower portion, eczema of the umbilicus is to be added.

The ano-genital region is most often attacked with eczema, essential pruritus, or syphilis.

About the flexor surfaces of all the joints eczema is apt to occur, while psoriasis attacks the extensor surfaces. About the ankles and wrists lichen planus frequently begins. The hands are prone to be attacked with dermatitis or chronic eczema induced by the work of the individual. Dysidrosis, ringworm and syphilis also frequently occur in this region. On the feet and between the toes ringworm, usually produced by the epidermophyton fungus, eczema and occasionally dysidrosis occur. On the soles keratoses and plantar warts are common.

Configuration.—Configuration is an important diagnostic feature. In many disorders the lesions are irregularly disseminated. On the other hand, in many others a conspicuous configuration is present. An annular lesion with a clear center is not infrequently seen in psoriasis, ringworm and syphilis. Gyrate figures produced by peripheral enlargement of early lesions with clear or other centers and coalescence with others are seen in psoriasis, multiform erythema, occasionally in mycosis fungoides, and some other disorders. A peculiar linear arrangement[1] is frequently observed in nevi, lichen planus, verruca planum juvenilis, and occasionally in psoriasis.

Color.—The color of lesions is also an important factor. A dull red is characteristic of most of the lesions of syphilis, but this is not infrequently mixed with brown. A purplish or violaceous tint occurs in lichen planus and different shades of yellow in the xanthomata. In acute eruptive disorders and in the early stages of more chronic ones the shade of red varies.

Lesions.—The diagnosis is made from the lesions presented. A thorough discussion of this is precluded on account of its great scope. Only a few important points can be mentioned. It is well to determine whether the lesions presented have been changed by local applications or internal medication. Very frequently the character of the eruption is so altered that a diagnosis is impossible until the added changes have been removed. It is well to determine, if possible, what constitutes the primary lesion. This, as a rule, can be accomplished by examining the eruption as a whole, as new lesions in the initial stage are very apt to be present among the older ones. The evolution of the disease can frequently be ascertained through a study of lesions on various parts of the body in different stages of development. Most cutaneous disorders have one or more characteristic lesion, and by recognizing the important lesions in an eruption the disease can usually be placed within a small group. For example, if the lesions are bullous, only a few diseases are characterized by these lesions and a consideration of them is soon accomplished. Again, if the lesions are ulcers, or should scar formation be present, the number of diseases producing these lesions are limited. A discussion of the disorders characterized by the various lesions of the skin is impracticable, although a knowledge of this is essential in order to properly classify cutaneous disorders. By referring to the Section on General Symptomatology several diseases illustrative of the various types of lesions may be seen.

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[1] Wise, New York Med. Jour., March 1, 1919, p. 367, The Linear Dermatoses.

In the examination of any eruption daylight is essential, as artificial light changes the appearance of the lesions and renders them less conspicuous. It is, furthermore, necessary to make a fairly general examination of the skin, as points of importance are frequently overlooked by the patient and these might also be missed by the examiner. For example, the nature of a small nodular eruption occuring about the angle of the mouth, simulating sycosis, is suggested when upon examination of the leg large ulcerating gummatous lesions are found which the patient considered of no importance. Careful examination of the mucous membranes of the mouth and throat frequently reveals signs of past or present disease, and these should never be overlooked.

A microscopic examination of hairs, scales, crusts and tissue is often necessary. In all cases of scabies and ringworm of its various types confirmation of the clinical diagnosis with the microscope is of much value.

An outline is given at the end of this section which might be followed in the study of the special features of certain lesions.

Tuberculin.—Three methods of administering tuberculin for purposes of diagnosis are now available: first, by giving hypodermatically Koch's old tuberculin; second, the "von Pirquet" tuberculin test; and third, the ophthalmo-tuberculin test.

Hypodermic Test.—Koch's old tuberculin is given preferably at midnight, beginning with one-quarter of a milligram (0.00025). The patient should have been prepared by being kept quiet for two days preceding the test and his temperature taken every two hours to determine the normal. If this be found to amount to 100 or more degrees the test should not be used. The reaction begins in from eight to twenty hours after the injection, usually in eighteen hours, and is indicated by a rise in temperature to 100, 101, or even 104 or 105 degrees. This is accompanied by severe headache, a feeling of general malaise, pain in the back and limbs, loss of appetite, at times nausea and vomiting, and, if severe, by grave prostration. As a rule, the symptoms subside in twenty-four hours, but may require two or three days to disappear. In addition to the general symptoms above recorded a local reaction is evident in the cutaneous lesion, exhibited by redness and other inflammatory phenomena. If no reaction occurs, a second dose of one milligram (0.001) is given in three days. If still no reaction, a third dose of three milligrams (0.003) is given in another three days. If after this no reaction is evident, the diagnosis may be considered negative as to tuberculosis.

"Von Pirquet" Test.—This is practically a local vaccination method. Two solutions are necessary: first, a 25 per cent. solution of Koch's old tuberculin; second, a blank solution. Dr. Lincoln[1] suggests for the first solution one part tuberculin, one part 5 per cent. phenol in glycerin, and two parts sterile 0.85 per cent. salt solution. The blank solution represents one part 5 per cent. phenol in glycerin and three parts sterile 0.85 per cent. salt solution.

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[1] Lincoln, May C., Jour. Amer. Med. Assoc., 1908, vol. li, 21, 1756-1761.

The arm is cleansed as in ordinary vaccination and one drop of each of the above solutions is placed on the cleansed area about two inches apart. Each is then scarified into the skin, with care not to make the surface bleed. Each drop is allowed to dry and is protected by a shield. In twenty to twenty-four hours the reaction, if it occur, is at its maximum and is exhibited as a hyperemic, sharply circumscribed, infiltrated lesion. There may be vesicle-formation, followed by crusting. In the area treated by the blank solution no significant change should occur. The reaction subsides in one to three weeks and is unaccompanied by constitutional symptoms.

Ophthalmo-tuberculin Test.—A 1 per cent. solution of tuberculin is used. This may be prepared by adding one tablet of prepared tuberculin (to be had in the market, prepared for this purpose) to 1 c.c. of sterile 0.85 per cent. salt solution.

The eyes should be free from all evidence of inflammatory changes when the test is made. If found normal, one drop of the solution named is instilled into one eye. The liquid should be moderately warm and then evenly diffused over the conjunctiva by gentle manipulation of the lower lid. The reaction reaches its maximum in twenty-four to thirty-six hours and subsides in two days to one week, as a rule, and is exhibited as a catarrhal conjunctivitis. Usually no subjective sensations are present, though mild burning and smarting with photophobia may occur.

A positive reaction is indicative of tuberculosis in some region of the body, provided the eye has not been previously tested. It is important to note that tests repeated after five or eight days are valueless.

Serum Diagnosis of Syphilis (Wassermann Reaction).[1]—The complement-fixation test, as employed in syphilis, was first used by Wassermann, Neisser and Bruck.[2] Its elaboration was made possible by the principles involved in the Bordet and Gengou reaction.[3] This reaction, with the principles of hemolysis, must be understood to appreciate the phenomena occurring in the test as applied to syphilis. Special laboratory facilities are required and much experience is necessary to make the test reliable. Many factors are concerned and much time is required, which make its performance purely a laboratory function. To obviate some of the disadvantages, many modifications have been used,[1] but the major portion of workers employ the so-called original test rather than its modifications. Boas[2] uses a quantitative method, by which more accurate results are obtained. Five amounts of serum, ranging from 0.2 c.c. to 0.01 c.c., are used, and in addition practically all workers agree that more than one antigen should be employed.[3] The principles and application of the test were early brought out in America by Fleischmann, Butler, Howard Fox, Noguchi, and others. The technique as employed by Dr. J. Frank Waugh is briefly outlined by him as follows (*Cf.* Section on Diagnosis of Syphilis for discussion of practical value of test).

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[1] Fleischmann and Butler, Jour. Amer. Med. Assoc., 1907, xlix, p. 924; Butler, New York Med. Jour., November 30, 1907; idem., Jour. Amer. Med. Assoc., 1910, liv, p. 1114; Howard Fox, Med. Record, New York, March 13, 1909; Swift, Archives of Internal Med., 1909, iv, p. 376 (A Comparative Study of Serum Diagnosis in Syphilis); Kolmer and Schamberg, Amer. Jour. Med. Sc., March. 1915, Discussion of Cholesterinized Antigens. For complete discussion of standardization of the Wassermann reaction see the following series of papers on studies in the standardization of the Wassermann reaction by Kolmer, Amer. Jour. Syph., 1919, 111, p. i and Brown and Kolmer, Ibid., p. 8 and 169, and Kolmer, Matsunami and Trist, pp. 407 and 513, and Kolmer and Flick, p. 541, and Kolmer, Trist and Flick, Ibid., 1920, iv, p. 111 and Kolmer and Riele, p. 135.

[2] Deutsch. med. Wochenschr., 1906, xix, p. 745.

[3] Annales de l'Institute Pasteur, 1901, p. 289.

Five factors are used in performing the test: sheep's corpuscles, amboceptor, complement, antigen, and patient's serum.

The first known factor to be prepared is a 5 per cent. suspension of washed sheep's corpuscles. The sheep's blood is defibrinated as soon as withdrawn from the animal and is then stored in an icebox until the test is to be made. Corpuscles over forty-eight hours old should not be used. The amount of defibrinated sheep's blood used depends on the number of sera to be tested. One c.c., after being washed at least twice in normal-salt solution, added to 19 c.c. normal-salt solution, will make 20 c.c. of a 5 per cent. suspension, as the normal defibrinated sheep's blood is considered a 100 per cent. suspension.

The next step is to determine the titer of the amboceptor contained in the inactivated serum of a rabbit immunized against sheep corpuscles by the successive intraperitoneal injections of washed sheep's corpuscles. Usually five injections of 2 c.c., 5 c.c., 8 c.c., 12 c.c., and 15 c.c., respectively, at five-day intervals, will suffice, the animal being bled nine days after the last injection.

Into each of a series of test-tubes is placed 1 c.c. of the 5 per cent. suspension of sheep's corpuscles and variable amounts of the inactivated rabbit serum; an equal and sufficient amount of complement or fresh guinea-pig serum being added to each tube, in order that all the amboceptor present be utilized in the process of hemolysis. From 0.10 to 0.12 c.c. of guinea-pig serum is used in each tube in titrating the amboceptor. The tube in which hemolysis is just complete contains 1 unit amboceptor. By reversing the process, or placing 1 unit amboceptor in each tube and variable amounts of complement, the titer of the complement is determined. Two units of each are used in the test. One c.c. of sheep's corpuscles, 1 unit amboceptor, and 1 unit complement constitute the hemolytic circle. When all are added to one tube, complete hemolysis results.

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[1] Noguchi, Jour. Exper. Med., 1909, ii, p. 392 (A New and Simple Method for the Serum Diagnosis of Syphilis); Waugh, Trans. Amer. Med. Assoc., Sec. on Derm., 1910, p. 193 (Results of Experience with Noguchi Modification of the Wassermann Serodiagnosis Test for Syphilis); Howard Fox, Jour. Cut. Dis., 1909, xxvii, p. 338.

[2] Die Wassermannsche Reaktion mit besonderer Berücksichtigung ihrer klinischen Verwertbarkeit (Harald Boas, Berlin, 1911, German translation); discussed by Fildes, Brit. Jour. Derm., 1911, xxiii, p. 13 (The Wassermann Reaction).

[3] Stillians, Jour. Cut. Dis., 1913, xxxi, p. 316.

A number of antigens have been recommended by different serologists. The following preparations have proven to be the most satisfactory: an alcoholic extract of a luetic fetal liver, using Porges and Meier's[1] method of preparation; alcoholic extracts of normal organs, a human or beef heart or liver usually being used and prepared by Michaelis and Lesser's[2] method; a cholesterinized alcoholic heart or liver extract, as recommended by Sachs;[3] and an acetone, insoluble antigen composed of lipoids from a beef liver, as advocated by Noguchi.[4] After having been prepared, the antigenic properties of the different antigens are determined by careful titration, using both luetic and normal serum.

It should be the rule that the amount of antigen selected as a unit should be the amount which does not bind complement, even when the antigen is used in double quantity, which does not cause hemolysis, and which does not inhibit hemolysis when normal serum is used. One unit of antigen is used in the test.

The patient's serum is secured by withdrawing blood from a vein, permitting it to clot, and inactivating the serum at 56° C. for half an hour in a water-bath. Washed sheep's corpuscles as recommended by Jacobeaus are then added to the serum, which is placed in an incubator for thirty minutes, during which period the normal anti-sheep amboceptor, if present, will be taken up by the corpuscles. The serum is then centrifuged, pipetted off, and placed in the icebox. From 0.1 c.c. to 0.2 c.c. is used in the test.

All the material for the test having been prepared, test-tubes are numbered and placed in a suitable rack or holder. There should be at least five for each serum. Others can be added if desired, in which may be placed additional antigens, if more than two are used, or variable amounts of the serum to be tested, if more than two different quantities are desired.

In the first five test-tubes are placed 2 units of complement; in tubes one and three, 0.1 c.c. of the serum to be tested; in tubes two, four, and five, 0.2 c.c. of the same serum. In tubes one and two, 1 unit of antigen No. 1, and in tubes three and four 1 unit of antigen No. 2 is added. In the second group of five tubes a similar distribution of the ingredients is made, using positive luetic serum in place of the unknown. In the third group normal serum is placed as above described, the antigens and the complement being the same as in the first two groups. Additional control tubes contain double the amount of antigens used in the test, with 2 units of complement.

The tubes are shaken to insure thorough mixing, then incubated for one hour. One c.c. of the 5 per cent. corpuscle suspension, 1 c.c. of normal-salt solution, and 2 units of amboceptor are then added to all tubes and the tubes again placed in the incubator. Hemolysis is usually complete within an hour in all tubes except those containing luetic serum and to which antigen was added; in these the corpuscles remain in suspension, which indicates a positive reaction. The final reading is taken several hours after the second incubation. The test is one of degree, depending upon complete or partial inhibition of hemolysis.

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[1] Berlin. klin. Wochenschr., 1908, xlv, p. 731.

[2] Ibid., 1908, xlv, p. 301.

[3] Ibid., 1911, xlviii, p. 2066.

[4] Serum Diagnosis of Syphilis. H. Noguchi, 3d edition.

Luetin Test.[1]—In 1911, Noguchi introduced another diagnostic test for syphilis, consisting of a cutaneous reaction similar to that devised by von Pirquet for tuberculosis. The material used in the test is termed "luetin," and is prepared by Noguchi as follows:

"Pure cultures of several strains of the pallidum are allowed to grow for periods of six, twelve, twenty-four, and fifty days at 37° C., under anaërobic conditions. One set is cultivated in ascitic fluid containing a piece of sterile placenta, and the other in ascitic-fluid agar also containing placenta. The lower portion of each solid culture, in which a dense growth has occurred, is cut out and the tissue removed. The agar columns, which contain innumerable spirochetæ, are then carefully ground in a sterile mortar. The resulting thick paste is gradually diluted by adding, little by little, the fluid culture, which also contains an enormous mass of the pure organisms.

"The dilution is continued until the emulsion becomes perfectly liquid. The preparation is next heated to 60° C. for thirty minutes in a water-bath and then 0.5 per cent. tricresol is added. When examined under the dark-field microscope, numerous dead pallida per field may be seen. Cultures made from this suspension remain sterile and with it no infection can be produced in the testicles of rabbits. The suspension is kept in a refrigerator when not in use.

"In order to ascertain whether the reaction with this suspension may not be due to the introduction of antiseptic culture medium alone, it is necessary to prepare a similar emulsion, with uninoculated media, to be used for control purposes."

Technique of Application.—Noguchi recommends the injection of luetin in one arm, with the control material in the opposite arm. Several others have used both injections in the same arm. In either case, the proposed sites for inoculation are thoroughly cleansed in the manner ordinarily used in the preparation for a surgical operation. The luetin and the control emulsions are thoroughly shaken and the desired amounts of each are removed from the containers with sterile pipettes and diluted with an equal quantity of sterile salt solution. They are then placed in sterile graduated tuberculin syringes fitted with fine needles. An intradermic injection, consisting of 0.07 c.c. of luetin, is made in one area and a similar quantity of control material injected in the other. It is recommended always to make the injection as superficial as possible.

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[1] Noguchi, Jour. Exper. Med., 1911, xiv, p. 557: "A Cutaneous Reaction in Syphilis." Idem., Jour. Amer. Med. Assoc., 1912, lviii, p. 1163.

The reactions which follow are described by Noguchi as follows:

Normal or Negative Reactions.—In the majority of normal persons, there appears after twenty-four hours a small erythematous area at and around the point of injection, unaccompanied by subjective sensations. The reaction gradually recedes within forty-eight hours and leaves no induration. In occasional instances a slight yellowish pigmentation results from mild ecchymosis.

Positive Reactions.—Three forms are described: (1) A papular form, in which a large, raised, reddish, indurated papule, usually from 5 to 10 mm. in diameter, makes its appearance in twenty-four to forty-eight hours. The papule may be surrounded by a diffuse zone of redness and show marked telangiectasis. The dimensions and the degree of induration slowly increase during the following three or four days, after which the inflammatory processes begin to recede, and the color of the papule gradually becomes dark bluish-red. The induration disappears within one week, except in certain instances, in which a trace of reaction may persist for a longer period. (2) A pustular form, the beginning and course of which resemble those of the papular form until about the fourth day, when the inflammatory processes commence to progress. The surface of the indurated, round papule becomes mildly edematous, and multiple miliary vesicles occasionally form. At the same time a beginning central softening of the papule can be seen. Within the next twenty-four hours the papule changes into a vesicle, filled at first with a semiopaque serum, which later becomes definitely purulent. Shortly afterward the pustule ruptures; its margin remains indurated, and its surface becomes covered with a crust, which falls within a few days. With the fall of the crust the induration disappears, leaving no sequels. A wide range of variation in the degree of intensity of the reaction described in different cases has been noted. (3) A torpid form. In rare instances the injection sites fade away to almost invisible points within three or four days, so that they may be passed over as negative reactions. These spots suddenly light up again after ten days or even longer and progress to small pustular formations, the subsequent course of which is similar to that described above.

The papule above described is in most instances a comparatively deep-seated, inflammatory nodule, and is frequently surrounded by a bright-red areola, varying from half an inch or less to two or more inches in diameter. Constitutional symptoms vary. As a rule, a slight rise of temperature is present for one day. With a marked reaction, however, general malaise, loss of appetite, and diarrhea may occur.

The test is chiefly of value in chronic cases, being rarely positive during the early and more active stages of the disorder. The reaction depends upon the hypersensitiveness induced in the system by the long-continued action of the Spirocheta pallida. In the early and active stages, where the Wassermann test is of most value, the luetin test is of little use; in the later and latent stages of the disease, where the Wassermann is liable to be negative and the disease still present, the luetin test becomes of value. Noguchi states that when syphilis has entered upon its chronic course, the direct demonstration of the Spirocheta pallida becomes difficult, if not impossible, the Wassermann reaction less frequently positive, and the clinical aspect less decisive. Here the detection of the allergic condition by the luetin test will be an aid in deciding the diagnosis in doubtful cases. As summed up by Foster,[1] a positive Wassermann test indicates the presence of metabolic substances in the blood serum, due to present or recent activity of numbers of spirochetæ on the tissues; while a positive luetin reaction is indicative of a state of hypersensitiveness to the specific proteins of the spirochetæ, induced by a period of cessation of the introduction of these proteins prior to the injection of the luetin.[2]

Method of Examination.—The following outline for the methodical examination of a patient affected with skin disease is based on the subjects considered in the preceding pages, and is given in such detail that a careful investigation of the questions suggested should furnish material for all but exceptional cases. For the average case much may be omitted.

The first attempts to follow such a scheme are necessarily tedious, and therefore often discouraging; but one patient thus carefully examined is of greater educational value than an aimless and indefinite examination of a dozen cases. There is no greater economy of time than is found in methodical and systematic habits of work.

HISTORY.

I. Name and Residence.
II. Age.
III. Sex.
IV. Married or Unmarried.
  1. Children.
    a. Living.
    b. Dead.
  2. Abortions or Miscarriages.
V. Family History.
VI. Individual History, *including that of previous skin diseases.*
VII. Occupation.
VIII. Habits *of eating, drinking, bathing, tobacco-usage, etc.*
IX. Present State of Health.
  (*Note the condition of the digestive, respiratory, circulatory, genito-urinary, and nervous systems; also, defects in assimilation, elimination, and nutrition.*)
X. History of Present Skin Diseases.
  1. Cause—if known.
  2. Character at first.
  3. Sites affected in order.
  4. Manner of progressing.
    a. Slow or rapid.
    b. Steady or irregular.
    c. With exacerbations and remissions.
    d. With periods of entire freedom from symptoms.
  5. Changes in character.
  6. Subjective sensations.
  7. Duration.
  8. Effect of temperature and seasons.
  9. Treatment to date.

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[1] Amer. Jour. Med. Sci., 1913, cxlvi, p. 645.

[2] For further discussion relative to the clinical application of the test, see chapter on Syphilis devoted to diagnosis.

OBJECTIVE SYMPTOMS.

- A. Accidental Complications *due to scratching, treatment, etc.*
- B. Site.
  - 1. Universal.
  - 2. Generalized.
  - 3. Diffuse.
  - 4. Local. (*Note influence of clothing, occupation, etc.*)
- I. Uniformity, *or multiformity.*
- II. Arrangement.
  - 1. Isolated.
  - 2. Grouped. (*Circinate, linear, etc.*)
  - 3. Discrete.
  - 4. Coalescing.
  - 5. Irregular.
- III. Definition. (*Sharp, fair, poor, or none.*)
- IV. Elevation, *or depression.*
- V. Color.
  - 1. Persistent.
  - 2. Changing or disappearing under pressure.
- VI. Shape.
  - 1. Apex.
  - 2. Base.
- VII. Size.
  - 1. Superficial.
  - 2. Deep.
- VIII. Anatomical site.
- C. Symmetry, *or asymmetry.*
- D. Acuteness, *or chronicity.*
- E. Moisture, *or absence of.*
- F. Individual Lesions.
  - 1. Elementary (*macule, papule, wheal, tubercle, tumor, vesicle, pustule, or bleb*).
  - 2. Consecutive (*scale, crust, excoriation, fissure, ulcer, or scar*)
- IX. Consistence.
  - 1. Firm.
  - 2. Soft.
- X. Base.
  - 1. Color.
  - 2. Infiltration.
- XI. Evolution.
  - 1. From sound skin.
  - 2. From other lesions.
- XII. Career.
  - 1. Transitory.
  - 2. Persistent.
  - 3. Type.
    - *a.* Simple.
    - *b.* Changing.
    - *c.* Modified.
- XIII. Involution.
  - 1. Resorption.
  - 2. Exfoliation.
  - 3. Ulceration.
  - 4. Atrophy, etc.
- XIV. Sequelæ.
  - 1. Stains.
  - 2. Scars.

SPECIAL FEATURES TO BE OBSERVED IN CERTAIN LESIONS

- A. Vesicles, Pustules, or Blebs.
  - I. Roof.
    - 1. Tense.
    - 2. Flaccid.
    - 3. Easily ruptured.
  - II. Contents.
    - 1. Translucent, *or opaque.*
    - 2. Serous.
    - 3. Purulent.
    - 4. Hemorrhagic.
  - III. Surface beneath.
  - IV. Areola.
  - V. Involution.
    - 1. Desiccation.
    - 2. Rupture.
    - 3. Crusts.
- B. Scales.
  - I. Size.
  - II. Color.
  - III. Quantity.
  - IV. Consistence.
    - 1. Dry.
    - 2. Fatty.
    - 3. Friable.
    - 4. Tough.
  - V. Attachment.
    - 1. Firm.
    - 2. Slight.
  - VI. Surface beneath.
    - 1. Color.
    - 2. Dry.
    - 3. Greasy.
    - 4. Hemorrhagic.
- C. Crusts.
  - I. Size.
  - II. Shape.
  - III. Color.
  - IV. Composition.
    - 1. Serum.
    - 2. Pus.
    - 3. Blood.
  - V. Attachment.
  - VI. Thickness.
  - VII. Consistence.
  - VIII. Surface beneath.
- D. Excoriations.
  - I. Distribution.
  - II. Shape.
  - III. Arrangement.
  - IV. Relation to other lesions.
  - V. Exudation.
- E. Fissures.
  - I. Distribution.
  - II. Size.
    - 1. Length.
    - 2. Depth.

- III. Pain.
- IV. Moisture.

F. Ulcers
- I. Size.
- II. Depth.
- III. Contour.
- IV. Base.
    1. Soft.
    2. Infiltrated.
    3. Indurated.
- V. Edges.
    1. Sloping.
    2. Perpendicular.
    3. Punched.
    4. Ragged.
    5. Everted.
    6. Undermined.
    7. Soft.
    8. Indurated.
- VI. Floor.
    1. Smooth.
    2. Uneven.
    3. Clean.
    4. Pus-covered.
    5. Granular.
    6. Sloughing.
    7. Hemorrhagic
    8. Glazed.
- VII. Secretion.
    1. Scanty.
    2. Profuse.
    3. Serous.
    4. Purulent.
    5. Hemorrhagic.
    6. Odor.
- VIII. Pain.
- IX. Crust.
- X. Evolution.
- XI. Duration.
- XII. Involution.

(*Note carefully the number and location of ulcers, the age of the patient, and the character of scars if present.*)

G. Scars.
- I. Size.
- II. Shape.
- III. Color.
- IV. Depression, *or elevation.*
- V. Texture.
    1. Soft, pliable.
    2. Hard, indurated.
    3. Thin.
    4. Thick.
    5. Smooth.
    6. Rough, corded.
- VI. Attachment.
- VII. Deformity.
- VIII. Subjective sensation.
- IX. Absence or presence of hairs, glands, and papillæ.

VI. GENERAL PROGNOSIS.

The ability accurately to outline the progress, duration and termination of the various diseases of the skin presupposes a knowledge of the subject. Some disorders will respond to treatment soon with their complete eradication. Others can only be benefited temporarily, as recurrences will happen in spite of any known method of treatment. A few are grave and irremediable and in these only temporary alleviative measures can be employed. By recognizing these facts and outlining them accurately, the patient, as a rule, accepts the situation and coöperates with the physician in the treatment outlined. Those individuals suffering with psoriasis, dermatitis herpetiformis, some forms of eczema, and other disorders accept recurrences without complaint and cheerfully do their part in combating the disease and keeping it under control so far as it is possible. Even in such serious affections as granuloma fungoides, pityriasis rubra of Hebra and other forms of general exfoliative dermatitis, and pemphigus, complete coöperation in the management is commonly seen when the limitations of treatment are understood by the patient, or his immediate friends or relatives. The important factor in all cases is not to engage to accomplish the impossible through the exigencies of serious cases, or through lack of proper appreciation of the facts concerned.

VII. GENERAL THERAPEUTICS.

THE general principles of the treatment of cutaneous disease are similar to those of other organs of the body. As cutaneous medicine is but a branch of internal medicine its successful practice demands a knowledge and experience in the latter. A number of visceral disorders exhibit some of their symptoms on the skin, and to interpret the latter properly general information is essential.

The etiology and pathogenesis of a large number of cutaneous diseases is obscure and yet by proper care of the patient the disorders often are carried to a successful conclusion. Many internal conditions indirectly contribute to the prolongation or severity of cutaneous diseases, and the recognition of this fact facilitates eradication of the disease in question.

Some diseases are self-limited and require but little actual treatment other than preventing harmful measures being employed by the patient or others. Other disorders can only be temporarily improved and in these the coöperation of the patient is gained by giving him the facts concerning the therapeutic limitations. It is not only necessary to make a correct diagnosis of the disorder presented, but, in addition, the stage to which it has progressed together with the associated conditions which may be contributory, and the individual peculiarities of the patient must be ascertained. This requires a thorough examination and proper judgment in interpreting the findings. The mode of life, habits of eating, drinking, exercise and rest may upon examination be found to be incorrect and necessitate regulation. Everything that is possible should be done to improve the general health and thereby increase the patient's resistance to disease.

The thorough examination above suggested is obviously not required in local parasitic affections. Except in the last-named conditions both internal and external treatment are essential.

INTERNAL TREATMENT.

The internal treatment of cutaneous diseases is comparatively limited. Large numbers of medicinal agents have been and are still being employed with indifferent results. The major portion of the internal treatment is directed toward removing the contributing factors concerned in the production or continuation of the cutaneous disorder. Only a few medicinal agents, such as arsenic, quinin, mercury and iodin compounds exert a decided influence on the diseases in which they are indicated.

Arsenic has headed the list of remedies used internally for the relief of cutaneous disorders, but with the exception of a few diseases arsenic should be reserved as a remedy of last resort. It is undoubtedly of great value in many cutaneous diseases and, furthermore, has a marked effect on the skin and particularly on the epidermis. The untoward results are acute or chronic. In the acute cases a mild erythema may occur, accompanied by moderate coryza and some redness from congestion of the vessels in the eyes and eyelids. The chronic effects occur only after prolonged administration of the drug and are evidenced by a generalized pigmentation or hyperkeratosis. It frequently produces excessive local keratosis of the palms and soles, which occasionally develops into carcinoma. As the major portion of diseases in which it has its most brilliant effects recur and thus require repeated periods of treatment, it is better to use other agents, if possible to prevent the chance of producing the untoward results above mentioned.

Arsenic may be administered in the following forms: Liquor potassii arsenitis (Fowler's solution); liquor arseni et hydrargyri iodidi (Donovan's solution); liquor arseni chloridi (de Valagin's solution); arsenic trioxid dispensed in tablet form, and the Asiatic pill. Duhring's modification of this pill is obtained by making 2 grains (0.13) of arsenic trioxid and 32 grains (2.13) each of black pepper and licorice powder into thirty-two pills by the aid of a sufficient quantity of acacia and water.

For hypodermic use sodium cacodylate is the form most often employed. This may be administered by intramuscular injection in dosage varying from ½ grain (0.0324) to 10 grains (0.65) or more. Ampules containing properly measured doses, sterilized and ready for use, may be obtained.

Another much less used form is *atoxyl* (meta-arsenious-anilide). This drug is given in large doses but not without danger. The dose recommended is 3 grains (0.195) once in three days.

Arsphenamin (Salvarsan, 606, Dioxydiamido-arseno-benzene-dihydrochlorid, $C_{12}H_{12}N_2O_2As_2 2HCl + 2H_2O$) has arsenic for its base and has largely replaced other remedial agents in the treatment of syphilis. It is fully described under that disease.

Mercury is directly beneficial in the treatment of syphilis and lichen planus. Indirectly it is of value through its power of elimination by way of the intestinal canal in a large number of cutaneous diseases. Where there is congestion or engorgement of the skin in localized areas or in general distribution, such as occurs in eczema and other dermatoses, mercury is also useful.

Iodin and its compounds are used largely in the treatment of syphilis. The preparations commonly employed are the iodids of potassium, sodium, lithium and ammonium; iodo-nucleoid, iodipin, and iodoform. The last preparation is serviceable in the treatment of acute lupus erythematosus in a dosage of 1 grain (.06) three times daily. The iodids also have been recommended in some forms of tuberculosis, keloid and psoriasis.

Cod Liver Oil.—This is a remedial agent of special value in diseases of the skin when the nutrition is subnormal. It is believed that in addition to its tonic and roborant effects it possesses parasiticidal qualities. The latter must, however, be indirect. It is of special value in the treatment of skin diseases in infants and children. In adults it is valuable in certain cases of eczema, in most tuberculous affections, in some cases of syphilis, scleroderma and in other disorders of the skin accompanied by malnutrition.

Quinin is valuable in the treatment of several cutaneous disorders. That it has a specific effect upon the skin is evidenced by the changes noted in so-called quinin eaters. It produces a peculiar smoothness and softness of the skin which disappears upon the suspension of the administration of the drug. In susceptible individuals it may produce a generalized erythema and in most individuals a certain amount of dizziness and tinnitus aurium, with possibly some gastric disturbance may occur. In cutaneous diseases occurring with malaria it exhibits its most brilliant results. It is of value when given in large doses in the treatment of dermatitis exfoliativa. It is also of some value in the treatment of pemphigus and in some angioneurotic disturbances.

Salol is employed in cutaneous diseases associated with intestinal fermentation and is most frequently used in pustular acne and urticaria.

Calx Sulphurata was once considered a very valuable agent in the treatment of cutaneous diseases. It was employed largely in furunculosis and other pyodermas, eczema and acne. It was given in doses of from $\frac{1}{10}$ (0.006) to $\frac{1}{4}$ (0.016) of a grain three or four times daily. It has now been very largely replaced by other agents.

Ichthyol[1] is a preparation which has been intensively studied and about which opinions differ as to its value. Internally it is administered in a dosage of 3 grains (0.2) three times daily, after meals. It is unquestionably of value in acute lupus erythematosus and rosacea.

Jaborandi and Pilocarpin, through their effects on the function of perspiration exert an effect on a number of cutaneous disorders, particularly those of the angioneurotic group.

Sulphur, once a very popular agent in the treatment of cutaneous disease, has largely been supplanted. It is of service as a cathartic and is recommended by Crocker in some disorders of the sweat glands.

Antimony is of unquestioned value in several cutaneous disorders. It has been employed with benefit in pruritus, dermatitis herpetiformis, some forms of eczema and psoriasis. More recently it has been found useful in the form of tartar emetic in the treatment of yaws.

Tar, Phenol, Creosote, Guaiacol, Resorcin, Turpentine, Copaiba and Phosphorus.—These remedies have been employed occasionally in the treatment of psoriasis, eczema, pruritus, and tuberculous affections of the skin. They are commonly disappointing in their action and they are now largely replaced by other agents.

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[1] McMurtry, Jour. Cut. Dis., 1913, xxxi, pp. 648-664 and 765-775 inclusive; a complete discussion of the drug, historical, chemical and therapeutic.

Animal Extracts.—Preparations from the thyroid, adrenal, pituitary, and other glands have in recent years been employed largely in various diseases of the skin. Thyroid gives brilliant results in the treatment of myxedema and it is of some value in ichthyosis, psoriasis, and some forms of tuberculosis of the skin. Also in certain cases of scleroderma and alopecia areata. Pituitary gland substance, suggested by Sherwell, has been of service in psoriasis.

Iron is of value in a long list of cutaneous disorders which are associated with anemia. It has been used largely in tuberculosis, syphilis, some of the purpuras, pemphigus and malignant disease.

Analgesics are used but very little in cutaneous medicine. It is occasionally necessary to use these preparations, but as a rule local treatment should be sufficient to control subjective discomfort without the aid of internal medication.

Cathartics, Alkalies and Diuretics.—These have an important place in the list of remedies valuable in the management of skin affections. Cathartics are chiefly valuable in eliminating toxic products from the intestinal canal, but they are also efficient in reducing congestion of the cutaneous surface. The value of mercurials in this connection already has been suggested. Saline laxatives and cathartics also are of great service, especially magnesium and sodium sulphate and Rochelle, Carlsprudel, and Hunyadi János salts.

Mistura ferri acida is a useful preparation and is constituted as follows:

℞—Magnes. sulphat,	℥jss;	45	
Acid. sulph. arom. (vel. dilut.),	ʒj;	4	
Ferri sulphatis,	grs. viij;		50
Aq. menth. piper.,	℥iv;	120	M. (filtra)

Sig.—A tablespoonful in hot or cold water before breakfast daily.

The alkalies commonly employed are the carbonates of sodium, potassium and lithium, as well as liquor potassæ. These substances are particularly valuable in certain forms of eczema and of some service in the inflammations of the face, such as acne rosacea and simple erythema. Diuretics are used occasionally in certain inflammatory disorders. The best diuretic is plain water, which should be taken between meals. It is frequently valuable to order two or three glasses of water to be taken on rising in the morning and before meals. It may be cool, but should not be iced.

Natural Mineral Waters.—The chief value of many of the mineral springs and health resorts in the United States lies in the change in the manner of living that they invite and necessitate. Sunshine, pure air and recreation after the care and toil of business, change of climate, of foods and drinks, often decide the question of speedy recovery. The sulphur waters of Richfield Springs, or Sharon Springs and of Avon Springs in this country, as of those of Europe operate chiefly by an influence exerted upon the digestive tract. The springs of West Virginia are examples of calcium waters having for the most part a diuretic effect. The water of the Poland Springs in Maine is chiefly valuable by reason of its purity. The alkaline waters of Colorado Springs, Saratoga and other sources in America, are similar to those of Vichy, Carlsbad and Ems of Europe. The constituents of these various waters are prepared in the form of salts, which may be employed with good results. The following waters may thus be prepared artificially: Apenta, Hunyadi János, Hathorn, Kissingen, Congress, Friedrichshall, Rakoczy, and others.

Opsonins.[1]—During the past several years opsonotherapy has attracted wide attention. In dermatology it is applied chiefly to infections induced by the staphylococcus and tubercle-bacillus. The chief disorders so treated are furunculosis, acne vulgaris, sycosis, lupus vulgaris, and scrofuloderma. Of those named above, lupus vulgaris seems most rebellious to the treatment.

The method was largely perfected by Wright and Douglas, of London. To be properly carried out much time and good laboratory facilities are necessary. The method essentially consists in injecting hypodermatically definite quantities of sterilized cultures of bacteria isolated from the affected patient.

It is proven that the serum of the blood contains substances which render bacteria susceptible to phagocytosis by the polymorphonuclear leukocytes. These substances Wright termed opsonins. The term is derived from a Greek word meaning "to prepare food," "to cook." As phagocytosis is the important feature in overcoming these infections, the quantity of opsonins becomes important.

For comparison in the work, the quantity of opsonins in a normal individual is denoted by 1. As a rule, in an infected patient they are reduced to 0.3, 0.4, 0.6, or 0.8; in other words, are less than normal (exceptions to this occur).

The opsonic index refers to the ratio between the amount of opsonins in the serum of an individual suffering with a bacterial infection and the amount in the serum of a normal, healthy person.

Wright says: "Vaccines are any substances that on being inoculated into the body will cause the generation of a protective substance." His vaccines consist of bacterial bodies.

Two difficult problems are presented: first, the determination of the proper dosage; and second, the time-interval between the injections.

Immediately following inoculation the amount of opsonins is diminished (negative phase). This period varies according to the size of the dose and other circumstances, and is followed by an increase and by a rise in the index (positive phase). By employment of sensitized organisms, the negative phase may be eliminated. After a varying time, the index begins to fall again but does not descend to its former low level. Therefore, by properly regulating the size of the dose and repeating it at the right interval, the amount of protective substances may be kept abundant, as indicated by a high index, and clinically by improvement in the symptoms.

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[1] LITERATURE.

Hektoen, L. Phagocytosis and Opsonins. Jour. Amer. Med. Assoc., 1906; xlvi, p. 1407 (an excellent exposition of the subject with full references to earlier work).

Whitfield, A. The Opsonic Method in Skin Diseases. Translations of the Sixth International Derm. Congress, 1908, pp. 273–283.

Von Eberts, E. M. Bacterial Inoculation in the Treatment of Suppurative and Tuberculous Diseases of the Skin, after the method of Wright, 1908. Ibid., pp. 284–290.

Schamberg, Jay F., Gildersleeve, N., and Shoemaker, H. Bacterial Injections in the Treatment of Diseases of the Skin, 1908. Ibid., pp. 291–308 (with references and followed by discussion).

The Preparation and Standardization of Bacterial Suspensions for Therapeutic Injection.—The size of the dose in the therapeutic injection should always be controlled by an approximate knowledge of the actual number of bacteria.

The method of standardizing the suspensions as originally devised by Wright is to be recommended.

It consists (1) of thoroughly mixing equal parts of an even, rather dense, bacterial suspension in NaCl solution and a known blood; (2) of making and staining a thin smear; and (3) of determining the relative number of red blood-corpuscles and bacteria in five or more fields under the $\frac{1}{12}$ objective. From this the number of bacteria per c.c. can readily be determined.

Example:—Suppose that in a given case the red blood-cells are five times as numerous as the bacteria. It is previously determined that the sample of blood used contains 5,000,000 erythrocytes per c.mm. Hence, the number of bacteria is 1,000,000 per c.mm., or 1,000,000,000 per c.c. If 50,000,000 are to be injected, one may inject $\frac{1}{20}$ c.c. diluted with b. s. NaCl sol.

Essentials in the Determination of the Staphylococcus Opsonic Index.—(1) An even suspension in NaCl of a 24-hour culture upon agar of the proper density. This is best obtained by suspending some of the growth from agar slant and then centrifuging down the clumps. A 24-hour-old broth culture also answers this purpose.

(2) Washed leukocytes or washed blood for phagocytes.—Ten or more drops of blood are obtained from a prick in the ear and suspended in a 2 per cent. sodium citrate solution contained in and nearly filling an ordinary electric centrifuge tube. This is then centrifuged thoroughly until both the red and white corpuscles are thrown down. The citrate solution is now poured or pipetted off and normal NaCl solution added and the corpuscles suspended and then again sedimented. This is usually repeated once more. The salt solution is decanted and the top layer, containing a large percentage of the white corpuscles, is pipetted off and thoroughly mixed and placed in a small test-tube. This is designated in general use as the "washed blood," "washed leukocytes," or "blood cream."

(3) The Sera.—The "normal pool" of equal parts of three or more normal sera, as well as the patient's serum, is best obtained in small U-tubes from a prick of the finger or the ear. After five to ten minutes, when clotting has taken place, the clot is separated from the serum by placing the tubes in the centrifuge sockets and centrifuging for four or five minutes at high speed.

The method of obtaining the blood and mixing the three essential factors by Wright's method is complicated and requires not a little skill in preparing the necessary glassware. The method evolved in Hektoen's laboratory by the use of the simple tube and a small capillary pipette, which is bent at right angles, is simple as well as accurate. A special incubator is entirely unnecessary.

After mixing the pool of normal sera, a small amount is drawn up the capillary tube for a distance of approximately two inches. This point is marked with a glass pencil or a bit of India ink. A small bubble of air is drawn in the end. When the washed blood, and in like manner the bacterial suspension is drawn to the point marked above, in this way equal parts of the three necessary factors (the serum, the washed blood, and the bacterial suspension) are obtained and then thoroughly mixed by drawing them back into the wider portion of the pipette five or more times. The second pipette is now prepared. It contains the patient's serum, the variable factor. Both pipettes are then placed in the thermostat at 37° and incubated for fifteen minutes.

Smears are now made, after mixing thoroughly, and the average number of bacteria contained in at least fifty leukocytes determined. This indicates the relative opsonic power of the normal and patient's sera.

From this the opsonic index is determined by dividing the result obtained where the patient's serum was used by the result obtained where normal serum was used.

For example: If the count where normal serum was used shows that an average of four staphylococci was taken up per leukocyte and where the patient's serum was used shows five, the opsonic index of the former is normal or unity, that of the latter $\frac{5}{4}$ or 1.25. Any of the polychrome blood-stains may be used to stain the smears. Two per cent. carbol-thionin solution in methyl alcohol is satisfactory.

The administration of vaccines today is largely regulated by the clinical effects produced, rather than by the complicated and tedious method above described. The principle of vaccine treatment is scientifically correct, but a definite technique has not as yet been clearly evolved. While brilliant results occasionally follow their use, they have, in the main, not accomplished the results which their early use promised in cutaneous diseases.[1]

External Vaccine Treatment.—The local application of dead bacteria incorporated in ointment bases was first largely used by Gilchrist,[2] who reported his results, combined with those of several colleagues, including the author, at the XVII International Congress of Medicine. Towle[3] at the same time was doing independent work along these lines. The method consists in the incorporation of the dead microörganisms in ointment bases, such as lanolin, vaselin, and cold cream, which are then applied on the affected areas in various cutaneous diseases. Acne vulgaris, eczema, eczematoid dermatitis, tuberculosis of the skin, and other disorders have been treated with varying success by this method.

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[1] *Cf.* Gilchrist, Trans. XVII. International Congress of Medicine, London, 1913, Sec. xiii. Part II, p. 405: Vaccine Therapy as Applied to Cutaneous Diseases (with discussion); Whitfield, ibid., Part I, p. 193: The Vaccine Treatment of Skin Diseases.

[2] Loc. cit.

[3] Jour. Cut. Dis., 1914, xxxii, p. 770.

Autoserum Treatment.[1]—During the last several years the injection of the patient's serum intravenously, subcutaneously or intramuscularly has been practised with varying results. Among other disorders so treated are pemphigus, dermatitis herpetiformis, psoriasis, chronic eczema, prurigo, lichen planus, chronic ulcerations and other cutaneous disorders. The technic suggested by Gottheil and Satenstein is the one most commonly used, with slight modifications. Some prefer to inject the serum subcutaneously or intramuscularly, while others continue the intravenous method.

Technic.—From 40 to 200 c.c. of blood are withdrawn from a vein at the elbow in the usual manner by placing a constrictor on the arm above and introducing a platinum needle into the vein, allowing the blood to flow directly into a centrifuge tube. Fordyce[2] suggested the use of the Macrae needle attached to a large centrifuge tube as a means of facilitating the removal of the blood, which is done by producing a vacuum in the tube, thus causing a free flow. We have commonly used a sterile nursing bottle fitted with a cork having two perforations. One of these is connected by rubber tubing with the needle in the vein and the other is arranged to withdraw the air from the bottle by suction. After removal the blood is permitted to clot. After a few hours the serum which has separated is decanted and then centrifuged with a highspeed centrifuge for thirty to forty minutes. The serum thus prepared is then injected intravenously in a dosage ranging from 20 to 60 c.c. Surgical cleanliness is absolutely essential throughout the entire procedure. Gottheil and Satenstein originally suggested from four to six injections at intervals of from five to seven days. We have adopted a method of giving the injections every five days and give a period of from five to ten injections, which has proved to be very satisfactory. The treatment is of distinct value in psoriasis when used in connection with chrysarobin. It is also of great assistance in dermatitis herpetiformis.

EXTERNAL TREATMENT.

The external treatment of skin diseases includes the use of many substances. The most important are the following: water, soaps, oil and liniments, lotions, numerous pastes and other preparations. A moderate amount of information concerning these is essential in the treatment of skin diseases.

Water is employed for cleansing the skin, as a component part of lotions, and in the form of baths. It may be used cold, warm or hot. It may be softened by the addition of various agents, or medicated in certain ways. As a rule, water is contra-indicated in acute, inflammatory diseases and in these the skin is cleansed with oils or greases. If necessary to use water it should be made bland by the addition of oatmeal, bran, starch, barley or gelatin. In certain diseases such as ichthyosis, keratosis pilaris and psoriasis, water alone or medicated may be freely used with benefit for the removal of scales and other epidermal product as a preliminary to the employment of other indicated treatment.

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[1] Gottheil and Satenstein, Trans. Amer. Med. Assoc., Sec. on Derm., 1914, p. 124; "The Autoserum Treatment in Dermatology." "Description of Technic, Personal Results and Bibliography." Hilario, Jour. Cut. Dis., 1914, xxxii, p. 480: "A Contribution to the Autoserum Therapy in Certain Diseases of the Skin."

[2] Trans. Amer. Med. Assoc., Sec. on Derm., 1914, p. 135.

Antiseptic solutions are employed in cleansing ulcers and various cutaneous infections as adjuvents to other methods of treatment.

Baths.—Continuous warm baths in which the patient remains in the water for several days at a time are employed in pemphigus.

Antiseptic baths composed of boric acid in saturated solution or mercuric chlorid, one dram in 30 gallons of water may be used in generalized furunculosis or other pyodermas.

Sulphur baths are prepared by adding a dram of Vleminckx's solution to a gallon of water. This is used only in sluggish dermatoses, or parasitic diseases such as scabies.

Salt baths are employed for stimulating effects and are limited in their use to disorders such as acne or rosacea. Four ounces of marine salt to the gallon of water may be used. The body surface only is bathed.

Alkaline baths may be used. Sodium bicarbonate or biborate, 8 to 12 ounces in 30 gallons of water may be employed when baths are necessary in inflammatory dermatoses, and are of value in pruritus and urticaria for the softening effect produced on the epithelial cells. They are more valuable in ichthyosis, psoriasis and keratosis pilaris than ordinary baths.

Tar baths are employed for stimulating purposes by anointing the skin with the preparation selected and then immersing the skin in warm water for varying periods of time, usually fifteen minutes to one hour. The surface after being dried is dusted over with an inert powder, or covered with a bland fat.

Vapor, steam, Russian and Turkish baths are of little value in cutaneous disease. They may be employed in ichthyosis, keratosis pilaris, and in some cases of general pruritus.

Natural spring water baths are of value and their usefulness is enhanced by the change in environment, rest from business and the regularity of their employment.

Cool spray and sponge and other baths recommended for improvement of the general health can only be employed in cutaneous medicine in a few disorders, such as acne, where the general surface of the skin is normal. The purpose of the general bath is the application of heat and this is rarely required.

Local hot bathing is of great service in relieving itching, such as that occurring in pruritus ani.

Soaps.—Soft soap (*sapo viridis, sapo mollis*) made by the addition of potassium hydroxid in slight excess to linseed oil is valuable in the treatment of skin diseases. It is used for the purpose of producing either a detersive or stimulating, and at times a slightly destructive effect upon the surface of the skin, or for the removal of pathological accumulations, such as crusts and scales. It may be used as a plaster or with water. It resembles Hebra's "Spiritus saponis alkalinus," which is composed of 2 ounces of green soap and 1 ounce of alkali scented with spirit of lavender. Hard, or sodium, soaps are employed chiefly for cleansing purposes.

"Over-fatty" or "superfatted" soaps, both sodium and potassium soaps, are neither alkalin nor neutral in reaction but contain a small excess of saponified fat. They have a mildly detersive action on the skin but only produce a moderate amount of lather.

Medicated soaps containing phenol, glycerin, tar, sulphur and various oils are to be had. They usually contain such a small portion of the individual medicament from which they are named that they are practically worthless except for purposes of ablution. Under cold pressure they may be made to contain medicinal agents in therapeutic proportions, but other methods of using the medicaments are preferable.

Oils and Liniments.—Oils are applied to the skin directly by pouring or by friction, or by the use of compresses or bandages which are saturated, or are spread with the material to be applied. Oils may be used for their nutritive, soothing or stimulating effects. They are very frequently used for the removal of crusts and scales. The nutritive and soothing oils are cod liver, olive, almond, linseed, neat's foot, castor and similar oils. Among the stimulating oils are those of tar, cade, white birch, cashew-nut, and juniper.

Liniments are composed of an oil and lime water. They are valuable where large areas are involved. They may be applied directly on the surface or by means of saturated dressings. Calamine liniment is an example.

℞—Zinc oxid,		℥ss;	15
Calaminæ,		℥ss;	15
Liquor calcis,			
Olei olivæ,	āā q. s.	℥iij;	240
M. Sig.			

Another soothing liniment is made by using equal parts of olive oil and lotio nigra. This may be made antipruritic by adding ½ ounce (15) of liquor carbonis detergens to four ounces (120) each of the other preparations. A commonly used one consists of equal parts of aqua calcis and olive or linseed oil.

Lotions.—Lotions are used in many cutaneous diseases. They may be applied continuously or intermittently, the latter usually being the method of choice. Water, lime water and elder flower water variously medicated constitute most lotions. Moderate amounts of glycerin and tragacanth are added to lessen the drying effects, and alcohol in small amounts to hold various substances in solution and for its curative and antiseptic effect. Not infrequently powders, such as talcum, zinc oxid, calamine or starch are added and these remain on the cutaneous surface when the lotion dries.

In soothing and antipruritic lotions are found sodium biborate, bicarbonate, subacetate of lead, phenol, boric acid, benzoic acid, salicylic acid and others. Lotio nigra and liquor carbonis detergens also belong to this group. In the stimulating and antiseptic lotions phenol, tar, sulphur, salicylic acid, resorcin and salts of mercury are used. Many preparations used in lotions are soothing, antipruritic and mildly antiseptic when used in small quantities but become stimulating and more strongly antiseptic when employed in increased strength. In the astringent lotions are found lime, tannic acid, zinc sulphate, lead acetate and aluminum acetate.

Powders are used chiefly for their absorptive and protective properties but they may be made astringent and antipruritic. They should be impalpable and never contain gritty particles. The ones commonly used are starch, talcum, zinc oxid, calamine, bismuth subnitrate and subcarbonate, lycopodium, rice and magnesium silicate. Such active substances as salicylic acid, boric acid, menthol, camphor and calcium may be added to meet various indications. Magnesium carbonate[1] is the most effective for absorbing moisture.

Ointments are used extensively in the treatment of cutaneous disorders. They consist of animal and vegetable fats and derivatives of petroleum into which various medicinal substances are incorporated. They are applied directly to the affected surface or spread on dressings and applied. They exert their effect for a longer period of time than lotions. Ointments are particularly valuable in eczema, the parasitic dermatoses and many other cutaneous affections. The ointment bases most commonly employed are the following. Adeps (lard), an animal fat which possesses the advantage of penetration into the skin and the disadvantage of becoming rancid. It should be used freshly prepared. Benzoin is added to it forming Adeps benzoinatus (U.S.P.), a preparation which does not so quickly become rancid. Adeps lanæ hydrosus (lanolin), a purified fat from sheep's wool, was first used as an ointment base by Liebreich. It is readily absorbed by the skin, does not become rancid, but is usually mixed with other bases on account of its consistency. Eucerin,[2] introduced by Unna, is similar to lanolin. It is made by the action of some substance obtained from wool fat upon vaseline. It is readily absorbed and is capable of taking up large quantities of water. Petrolatum (U.S.P.) or vaseline is an excellent ointment base, is either white or yellow, does not become rancid, though it is irritating occasionally to the skin. Unguentum (U.S.P.) is a mixture of lard and white wax and is valuable as a base. Unguentum aquæ rosæ (cold cream) is a cooling and a commonly used ointment base and preferred by many to other bases. To increase the consistency of any of these simple bases white wax, spermaceti or cocoa butter may be added. The above are rarely used alone but are grouped in varying combinations. Soothing ointments are required where the skin is inflamed and sensitive as in certain forms of eczema. Among others may be mentioned zinc oxid ointment, cold cream, spermaceti, and cucumber. By the addition of varying amount of bismuth subnitrate, zinc oxid, calamine, salicylic acid, or boric acid to these they become astringent as well as soothing and are antipruritic and mildly antiseptic. Hebra's diachylon ointment, when well made, belongs here. Stimulating and antiseptic ointments are obtained by adding salicylic acid in greater strength, boric acid, ammoniated mercury, calomel, sulphur, resorcin, chrysarobin, various tars and other preparations. The amount of each ingredient depends upon the degree of stimulation required. As a rule soothing and astringent ointments should be applied on clean old linen or other smooth material, while stimulating ointments should be rubbed in as well as applied. Special formulæ are given throughout the text for the particular conditions in which they are employed.

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[1] Grundler, Monats., 1888, vii, p. 1029.

[2] Jackson, Jour. Cut. Dis., 1910, xxviii, p. 294.

Pastes are similar to ointments but have greater consistency, usually obtained by the addition of various powders. They were largely perfected by Lassar and Unna.[1] They are more protective and adhesive than ointments and will remain on the surface longer and may be medicated to meet individual requirements. Lassar's paste consists of 25 parts of zinc oxid, 25 parts of amylum and 50 parts of petrolatum. Usually 1 or 2 per cent. of salicylic acid is added. Duhring's modification of this is: Boric acid, 1 part; starch, 3 parts; zinc oxid, 3 parts and vaseline 12 parts. Equal parts of lanolin, vaseline, zinc oxid and talc form a stiffer and more adherent base.

Plasters are employed on small areas when a continuous effect is desired. Mercurial plasters are valuable in the treatment of syphilitic lesions of the skin. Plasters containing salicylic acid are most useful. They may be used on localized patches in chronic eczema, on keratoses and verrucous growths. Salve muslins devised by Unna are strips or bandages of muslin thoroughly impregnated and thickly spread with ointments medicated with various substances including zinc oxid, tar, salicylic acid and mercury. They are efficient when fresh but applicable only to localized surfaces. Plaster mulls also devised by Unna are plasters thinly spread on gutta percha cloth and incorporate a large number of medicinal agents.

Salve-pencils (*stilli unguentes*) and Paste-pencils (*stilli dilubiles*), the latter destitute of fat and soluble when moist, the former insoluble in water and compounded of fatty substances, are pencil-sized crayons made of wax, gum, and starch, for application to limited areas of the skin. The several mercurials, arsenic trioxid, cocain, salicylic acid, and other medicaments may be applied in this way to the surface.

Protective Dressings.—A number of preparations are employed as more or less permanent dressings which are in themselves protective, and by incorporating various medical agents in them, results are obtained that cannot be accomplished by the various methods previously described.

Glycogelatins are useful for protecting a surface and excluding the air. They are made with varying proportions of glycerin, gelatin, zinc oxid, and water. When cold they are solid, but when melted on a water-bath can be painted readily over a surface upon which on cooling they form an adherent protective coating. Before the gelatin has hardened on the skin it is well to pat it with cotton, or to lay over it a piece of thin gauze or muslin to form an additional protection and to prevent the paste sticking to the clothing. A firm but soft and flexible gelatin is made by mixing on a hot-water bath 1 part of zinc oxid, 2 of gelatin, 3 of glycerin, and 4 of water. More gelatin in the preparation makes it firmer and causes it to dry more quickly. A greater proportion of glycerin, on the other hand, interferes with the complete drying of the surface, but makes a softer preparation, more acceptable to some skins, and very useful where a bandage can be applied. Zinc oxid helps to give body to the gelatin, but if used in too large proportion interferes with the coherence of the preparation, so that it cracks when dry. To the glycogelatins may be added ammoniated mercury, sulphur, ichthyol, thiol, or chrysarobin. Some drugs, as salicylic acid, resorcin, naphthol and phenol tend to destroy the coherence of the gelatin. According to Fox this difficulty may be overcome by adding 5 to 10 per cent. of fresh lard to the paste.

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[1] Monatshefte, 1884, iii, p. 38.

Tragacanth jelly contains glycerin and tragacanth and is a colorless protective substance particularly serviceable on the face. Phenol, ichthyol and other substances may be incorporated. Pick's varnish (linimentum exsiccans) is made as follows:

℞—Tragacanth,	5 parts
Glycerin,	2 parts
Distilled water,	93 parts

The tragacanth is soaked in a portion of the water from ten to twelve hours and triturated to a perfectly smooth mass before adding the glycerin and other ingredients ordered. The jelly may be prepared without delay by triturating the tragacanth with boiling water, but the result is not so good.

The jelly is applied without heating and quickly dries on the skin. An improvement on this varnish is Elliot's bassorin paste, which keeps better than the former. The formula is as follows:

℞—Bassorin,	℥iss;	45
Dextrin.,	ʒvj;	24
Glycerin,	ʒiiss;	10
Water to make	℥iij;	90

This should be kept in a tightly closed jar, as it dries rapidly on exposure to the air. Like Pick's varnish, it not only serves as a protective coating, but also as a base for the application of other remedies.

Collodion and Traumaticin are employed for the purpose of limiting the action of the remedial agent to localized areas, for protection and contraction of the surface to which the application is made. Traumaticin is a solution of gutta percha in chloroform in the proportion of 10 per cent. Salicylic acid or chrysarobin may be incorporated in varying strengths and used in the treatment of psoriasis and ringworm over limited areas. Sulphur, ammoniated mercury, bismuth and other agents are similarly employed. Recently the contracting effect of collodion has been taken advantage of in the treatment of erysipelas with good results.

Caustics are chemical substances employed to destroy tissue. Their action varies from moderate irritation producing exfoliation of the epidermis to deep destruction of the tissue with which they come in contact. Among others having a superficial destructive effect, owing to irritative action, are salicylic acid, iodin, cantharides, mustard, mercuric chlorid and resorcin. The stronger caustics include pyrogallic acid, chromic acid, trichloracetic acid, ethylate of sodium and silver nitrate. The most active include potassium hydroxid, zinc chlorid, nitric acid, acid nitrate of mercury and arsenic.

Potassium hydroxid is powerful and has to be used with caution as the destructive effects may extend more deeply and extensively than is intended. Its application is accompanied by pain, which soon subsides. The action may be stopped at a given point by applying dilute acetic acid.

Zinc chlorid acts more slowly and the destruction is more limited than with potassium hydroxid, but is accompanied by pain of longer duration. It forms a white eschar.

Nitric acid and **acid nitrate of mercury** do not act so deeply. But the former may be followed by disfiguring scars; the latter is painful but has been used with excellent results in epithelioma by Sherwell, MacKee and others.

Arsenic has a selective action on pathological tissue. It acts slowly and is accompanied by pain and much inflammatory reaction. It should be used only on limited areas.

Pyrogallic acid also has a selective action on pathological tissue.

Chromic acid is used on warts and corns.

Salicylic acid has marked keratolytic qualities and when incorporated in plaster, collodion or ointment in a strength of 10 to 50 per cent. is valuable for removing keratoses.

Trichloracetic acid is a valuable superficial caustic in moles and other lesions.[1]

Other caustics are employed occasionally. The use of strong caustics has been greatly limited since the introduction of *x*-rays, radium and carbon dioxid snow.

Drugs.—Several drugs and chemical substances are particularly valuable in dermatology. The principle ones are briefly described below.

Salicylic Acid.[2]—Salicylic acid is one of the most valuable agents for local application. It exercises different properties, according to the strength, duration and method of application. It is used in lotions, being soluble in 308 parts of water or 2 parts of alcohol or ether. It is also used in numerous pastes, plasters, varnishes, or incorporated in collodion or traumaticin. Its chief action is keratolytic when used in a strength of 3 per cent. or more. This quality is taken advantage of in the treatment of keratoses and other epidermal hypertrophies or new growths. It is also antiseptic and is of value in pustular infections of the skin. It is keratoplastic and antipruritic when used in ½ to 2 per cent. In a strength of 25 to 30 per cent. it is mildly caustic and is selective in its action. It is antifermentative and because of that quality is used in the treatment of hyperidrosis. It has a resolvent action and may be used in chronic eczema, lichen simplex chronica and patches of lichen planus. It is also combined with other agents and adds to their effectiveness by its softening action on the epidermal cells.

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[1] *Cf.* Davis, Jour. Cut. Dis., 1915, xxxiii, p. 691; Trichloracetic acid and its uses in dermatology.

[2] McMurtry, Jour. Cut. Dis., 1913, xxxi, p. 166.

Resorcin[1] has many properties that are of value in the treatment of cutaneous diseases. It is soluble in 0.5 parts of water or in 0.4 parts of alcohol and is even more soluble in ether. It is used in lotions, ointments and plasters. It is antipruritic in solutions varying from ½ to 3 per cent. or in ointments of less than 5 per cent. It is keratoplastic in solutions of 2 to 4 per cent. and is keratolytic when used in a strength of 10 to 50 per cent. in ointment or plaster. This quality is taken advantage of in the exfoliative treatment of Unna and Darier in seborrhea, acne and rosacea. It is mildly caustic in a strength of 50 to 75 per cent. It stains both the skin and the hair and occasionally induces a severe dermatitis. On account of this propensity to stain resorcin should be used in the scalp only in dark-haired individuals. Its chief field of usefulness lies in the treatment of pityriasis steatoides, seborrhea, dermatitis seborrhoica and acne. It is also of some value in eczema, pus infections, pernio and pruritus hiemalis.

Sulphur[2] is used largely in the treatment of parasitic affections and the disorders of the sebaceous glands. Precipitated sulphur is the preparation commonly selected and it is used in varying strengths as a powder, or incorporated in an ointment base. Its action on the skin is keratoplastic, keratolytic and stimulative. It is markedly parasiticidal through the formation of hydrogen sulphid when applied on the skin. Its action is facilitated by the addition of some substance capable of removing the stratum corneum, such as sapo mollis, or an alkali such as potassium carbonate. An ointment containing 5 to 15 per cent. may be used in scabies. Much stronger applications are used in chronic eczema, for stimulating purposes alone or combined with other agents. It is also extensively used in acne, comedones, seborrhea, rosacea, pityriasis steatoides and dermatitis seborrhoica.

Chrysarobin has a marked effect on the skin, producing an inflammatory reaction of varying grades depending upon the strength, duration and method of application. As it produces changes in the color of the skin, hair and clothing, as well as irritative effects on the conjunctiva, it must be used with care. It is incorporated in numerous pastes or fixed dressings, such as collodion and traumaticin. In a strength of

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[1] McMurtry, Jour. Cut. Dis., 1913, xxxi, p. 255.

[2] Ibid., pp. 322-399.

10 to 20 per cent. it has a keratolytic action, removing the epidermis by a hardening process with exfoliation, similar to resorcin but, unlike salicylic acid, which softens and dissolves the epidermal cells. In a strength of less than 5 per cent., by means of its hardening effect on the stratum corneum, it exercises a keratoplastic influence. By producing hardening and exfoliation of the epidermis, thus removing microorganisms, it acts as an antiparasitic. Schamberg, Kolmer and Raiziss[1] found no direct parasiticidal effects on the microörganisms in the skin. Its indirect antiparasitic action makes it of value in ringworm of all varieties and pyogenic infections. It is strongly stimulating and because of this quality is used in chronic eczema, hypertrophic lichen planus, and other disorders characterized by thickening of the skin. Its action is accelerated by combining it with salicylic acid and sapo mollis. It is used largely in the treatment of psoriasis, in which a less marked inflammatory reaction occurs in the lesions than in the surrounding skin. When used in the scalp in ringworm its action must be carefully watched to prevent irritation of the eyes through transference to this region. Its ability to stain the hair is also to be recognized. Its action may be limited to the area of application by incorporating it in collodion or traumaticin.

Ichthyol[2] introduced by Unna, is a distillation product of a bituminous stone, the remains of fossillized fish found in the Austrian Tyrol. Ichthyol is a complex substance and contains a high percentage of sulphur. It is soluble in water and in equal parts of alcohol and ether. The many qualities ascribed to it have been intensely studied and at present opinion is divided as to its real value. As an antiseptic its action is slight compared with phenol and other preparations, but through its action as a reducing and exfoliating agent it is of value in parasitic affections. In pure form or diluted to a strength of 50 or 30 per cent. it produces a drying effect upon the upper layers of the epidermis, causing the cells to shrink and exfoliate, thus carrying off any bacteria present. It is astringent and acts as a vasoconstrictor. It also possesses keratoplastic and antipruritic qualities and is a resolvent. In watery solutions it may be used as a varnish to hold the various medicinal agents in contact with the skin. It is used as a lotion in pure form or diluted in varying strengths with water. Some glycerin may be added, which delays drying. It is also used in ointments and pastes in a strength varying from 2 to 10 per cent. Ichthyol is of value in erysipelas, pernio, some varieties of eczema, intertrigo and rosacea. It has also been used in acne, various pus infections, lupus erythematosus and various other disorders.

Naftalan is a distillation product from crude nafta that is found in the Caucasus. It is a thick fluid of dark green color and contains 2½ to 4 per cent. of soap. It may be mixed with powders, thus producing an ointment of any consistency. It is a most valuable preparation and is advised in inflammation of the skin accompanied by moisture.

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[1] Jour. Cut. Dis., 1915, xxxiii, pp. 1 and 98.

[2] McMurtry, Jour. Cut. Dis., 1913, xxxi, pp. 648-664 and 765-775 incl.

Boric acid is extensively used as a powder or in lotions, fomentations, ointments and pastes. It is mildly antiseptic, soothing, and possesses keratoplastic qualities.

Phenol is used largely as an antipruritic. It should be employed in a lotion combined with glycerin, or in oil in a strength of ½ to 4 per cent. Its long contact with the skin, even in high dilution in ointment or paste, or continuous wet dressing, may induce gangrene in susceptible skins. Phenol is mildly antiseptic but other preparations are commonly employed for this purpose.

Tar is one of the most valuable local agents used in cutaneous disorders. The most common preparations are pix liquida, oleum rusci, oleum cadini and crude coal tar. These preparations are used in a strength of ½ to 2 drams (2 to 8) to the ounce (30.) of ointment base. They are made more efficient by the addition of an alkali, such as potassium subcarbonate which softens or removes the stratum corneum. Tar may be used in its pure state in certain chronic sluggish conditions. The great value of crude coal tar has recently been emphasized by White.[1] In using tar moderate amounts should be first incorporated in an ointment and if well tolerated the strength may be increased. Valuable liquid preparations of tar are liquor carbonis detergens (an English proprietary preparation), liquor picis alkalinus (Bulkley) and a compound tincture of coal tar (Duhring). In moderate strength tar is soothing and antipruritic. Stronger applications are stimulating and produce inflammatory reactions of varying grade. There is no preparation that is of so much value in such a wide range of cutaneous disorders as tar. A careful study of its various effects is essential for the accomplishment of best results. In combination with sulphur it is extensively employed in the form of Wilkinson's salve.

Pyoktanin blue is employed in saturated aqueous solution and is soothing and antiseptic in its action. Its striking color precludes its use on exposed portions of the skin, but it is a useful application on ulcerating surfaces such as occur in syphilis, tuberculosis, and those due to infection with pyogenic organisms. It is disinfectant in the ulcers of carcinoma. It has keratoplastic qualities and hence is of value in some cases of weeping eczema. It is applied in several coats daily, each coat being permitted to dry before the next is superimposed.

Mercury and its salts are extensively used in various cutaneous diseases. It is applied in lotions, wet dressings, ointments and plasters. Mercuric chlorid is used as an antiseptic in lotions in the management of several affections of the scalp and as a local bath or fomentation in pyogenic infections. Mercurous chlorid (calomel) and hydrargyrum ammoniatum (white precipitate) are frequently used in ointments for their antiseptic properties in pus infections. Calomel is also applied combined with talc or starch as a dusting powder. The various preparations of mercury are of much value in the treatment of the syphilodermata. (Cf. section on this disease.)

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[1] Jour. Cut. Dis., 1916, xxxiv, p. 497.

Hyperemic Treatment (Biers).[1]—This method of treatment finds some application in cutaneous diseases. Both passive or venous and active or arterial hyperemia may be used here as well as in other branches of medicine and surgery. Passive hyperemia may be induced by an elastic bandage or by means of cupping. Active hyperemia is induced by hot air. By one or the other of the methods, such diseases as eczema, psoriasis, sycosis, keloid, alopecia areata, lupus vulgaris, and staphylocccus infections may be benefited.

Electricity.—Galvanic and Faradic electricity are used only to a moderate extent in cutaneous diseases. Their use for improvement of general nutrition may be of value in certain dermatoses. The stimulating effect on the skin may be taken advantage of in the treatment in some cases of acne, alopecia areata, and other affections. Electrictiy is employed also by means of the galvanocautery[2] in the treatment of lupus vulgaris and other affections requiring a cauterizing agent.

Electric cataphoresis is rarely used except for producing local anesthesia with cocaine. Iodin, mercury and other substances may be administered in this way.

High Frequency Current.—Many types of apparatus have been employed to produce electric currents of high potential. The one most commonly used is the Ouidan resonator, or some modification of this. Glass vacuum electrodes of different shapes and sizes are attached to one terminal of the apparatus employed. The electrode is applied directly on the skin or held at a short distance. When directly applied a sensation of heat is experienced. If held at some distance, a pricking or tingling sensation is induced, or actual pain occurs if the current is strong. Effects ranging from moderate stimulation to destruction of tissue may be obtained, depending upon the strength of the current and the duration of the application. High frequency currents have been employed in pruritus, alopecia areata, chronic eczema, acne vulgaris and other cutaneous disorders.

Fulguration.[3]—Fulguration consists in the local application of the high frequency current by means of pointed insulated metallic electrodes. A stream of fine sparks flows from the point of the electrode into the tissue treated. The action of the high frequency spark thus applied is caustic. The degree of destruction depends upon the strength of the current used and the duration of the application. The treatment is painful but is well borne on account of its short duration, a few seconds only being required to produce the desired results. The technic employed is to place the point of the electrode on the part to be treated, then turn on the current, when immediate blanching occurs. The duration of the application depends upon the strength of current used and the tissue being treated, but it is usually limited to a few seconds. A reaction follows, indicated by hyperemia, swelling, exudation and crust formation. The crust or eschar falls in a week or ten days. If the application is mild no scar formation results. Fulguration is used in the removal of moles, various types of warts, small keloids, hypertrophic scars, xanthelasma and nevi. The method must be used with careful attention to details when cosmetic results are important.

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[1] Biers' Hyperemic Treatment, 1908; Willy Meyer and Victor Schwieden.

[2] Hazen, Jour. Cut. Dis., 1917, xxxv, p. 590. The Electric Cautery in Cutaneous Surgery.

[3] MacKee, Jour. Cut. Dis., 1909, xxvii, p. 245, with references.

Electrolysis.—Electrolysis is a valuable method of treatment for many cutaneous diseases and abnormalities. It essentially is a destructive action occurring through local application of an electric current. When a needle is attached to the negative pole and inserted in living tissue active destruction occurs about the needle with the formation and liberation of hydrogen. Albumin is coagulated, a blood clot is formed and the reaction of the tissue is alkaline. If the needle is attached to the positive pole the reaction of the tissue is acid and oxygen is produced. The negative pole is more rapid in its action and is, therefore, the one selected in the treatment of most cutaneous affections. In addition, it is the only one that can be employed with safety if steel needles are used, owing to a black deposit of the oxid of the metal occurring when the positive pole is used. When platinum needles are used this danger is eliminated.

The instruments necessary for doing this work are the following: A wall plate from which a galvanic current can be obtained and to which is attached a milliampèremeter for measuring the dosage; or, what is preferable, the galvanic battery. The number of cells in this may vary from ten to forty. It should be so arranged with a switchboard that any number of cells may be brought into the circuit. The milliampèremeter is necessary to measure the amount of current employed. The strength used in most instances varies from one-half to four milliampères. The number of cells in the circuit may range from two or four to twelve, twenty or more. In addition to the source of supply of current, needle-holders and needles are necessary. The needle-holder should be simply a convenient insulated handle sufficiently long to protect all the parts of the operator's right hand from the current, and should be as light as possible. The holder suggested by Duhring[1] is the shape of a penholder and about four inches in length. The handle is of hard rubber through which passes a metallic rod which acts as a conductor for transmission of the current. The needle is inserted into the needle-holder proper which is slotted, the needle being clamped immovably by means of a screw-nut. In the other end of the stem is an insulated inserting pin attached to a cord leading to the battery. This instrument is convenient to handle and well adapted to the operation. Irido-platinum or pure platinum needles are most commonly used. Owing to the danger of tattooing, steel needles should be avoided.

The technic consists in inserting the needle, which is always attached to the negative pole, into the hair follicle or bloodvessel, mole, wart, or other structure to be treated, completing the circuit by having a wet sponge, which is attached to the opposite pole, applied to some portion of the patient's body, usually the hand, and allowing the electrolytic action to go on for the necessary time required to accomplish the desired result. (Cf. Hypertrichosis, telangiectasia and other disorders for technic in individual diseases.) Electrolysis is the best method of treatment in hypertrichosis and telangiectasia occurring in rosacea and other diseases, or independently. It is also employed in some nevi, molluscum contagiosum, various warts and other affections.

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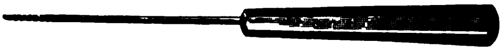
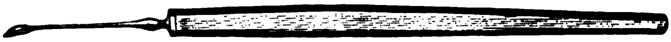
[1] Amer. Jour. of Med. Sc., 1881, lxxiii, p. 142.

FIG. 23



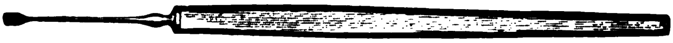
Irido-platinum needle.

FIG. 24



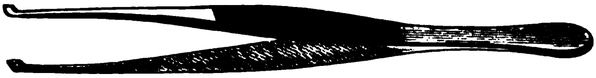
Milium-needle.

FIG. 25



Scarifying-spud.

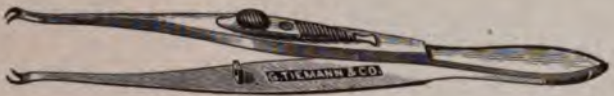
FIG. 26



Epilating-forceps.

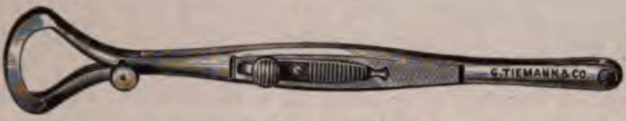
Special Instruments.—In the treatment of diseases of the skin only a few simple instruments are necessary. Among others are the following: Several dermal curettes. These are employed for erasion of the surface and each should have a fenestrum in its floor large enough to permit the escape of collected substances. The small-sized curettes which have solid bowls and sharp edges may be used particularly about the face. Epilating forceps with easy springs and smooth blades which meet in perfect apposition. Comedone extractors, provided at each extremity with different-sized, minute, spoon-shaped and perforated bowls, the convex surfaces of which are pressed over the comedo with the orifice immediately over the black head of the plaque. A set of lenses, which may vary from one-half to four inches in diameter. Needle-holders with light handles for firmly grasping needles used for opening pustules, puncturing new growths, and other purposes. Among the needles which are serviceable is the flat one with a double cutting edge. A variety of needles, probes, fine dressing forceps and

FIG. 27


G.TIEMANN & CO.

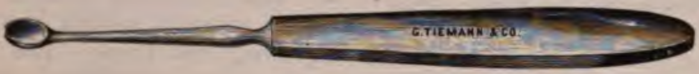
Piffard's grappling-forceps.

FIG. 28


G.TIEMANN & CO.

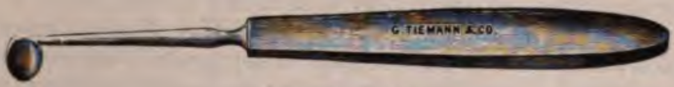
Piffard's cutisector.

FIG. 29


G.TIEMANN & CO.

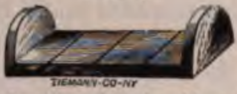
Dermal curette.

FIG. 30


G.TIEMANN & CO.

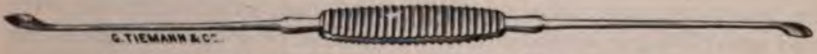
Dermal curette.

FIG. 31



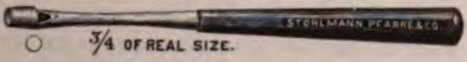
Hess's pleximeter, for observing the skin under pressure.

FIG. 32


G.TIEMANN & CO.

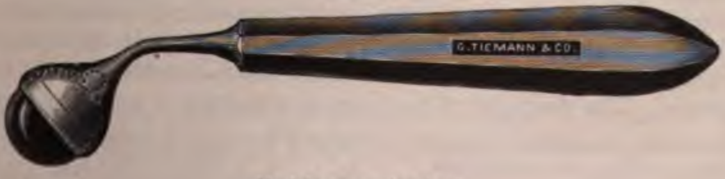
Piffard's modification of Unna's comedo-extractor.

FIG. 33


STOHLMANN PFARRE & CO.
3/4 OF REAL SIZE.

Keyes' cutaneous punch.

FIG. 34


G.TIEMANN & CO.

Hyde's massering-ball.

various shaped scissors are necessary. The equipment for administering arsphenamin is described in the section devoted to the treatment of syphilis. In addition to these a pleximeter which may be employed as a diascope, and a cutaneous punch for the removal of small growths and tissue for histological examination are essential.

Radiotherapy.[1]—*Synonym.*—Treatment by *x*-rays. Shortly after the introduction of radiotherapy it became a popular method and was extensively employed. This was followed later by a period of depression and only a moderate number continued its use. During the last few years, through improvement in apparatus, *x*-rays have become almost indispensable both as a therapeutic and diagnostic measure. Radiotherapy has been used without interruption by the author over a long period and its value has been proved. Among other cutaneous diseases in which it is of value may be mentioned epithelioma, keloid, lupus vulgaris, and other forms of tuberculosis, coccogenous and hyphogenous sycosis, acne vulgaris, rosacea, chronic eczema, lichen planus, psoriasis, ringworm, and favus. The list includes diverse morbid conditions, but these are remedied by one or the other of the therapeutic properties of the agent. *X*-rays *per se* are not germicidal, but indirectly, through tissue reaction, they may produce such effects in a high degree, as shown by the partial or complete arrest of purulent discharge from the surface of carcinomatous or other ulcers subjected to their action. They produce degeneration in cells of embryonic type without destroying the healthy stroma in which they have developed. Cells also of higher differentiation are affected early. As a consequence, hair follicles and sebaceous glands may become partially or wholly atrophied under the influence of the rays, the result depending upon the dosage employed.

Apparatus.—Early in the development of radiotherapy induction coils or the static machine were used with tubes of unstable vacuum. These have now been largely replaced by the interrupterless transformer and Coolidge tube. With the induction coil and its accompanying accessories and tubes as above mentioned, excellent results were and are still being obtained, but the question of dosage was uncertain and a perfect technic was impossible. Individual operators developed a safe technic with experience and for years employed *x*-rays safely and successfully, but owing to several unconstant factors always present, general rules could not be made which could be safely followed by all. The tube with its varying qualities always presented a problem difficult to solve, as it was impossible to measure with accuracy the amount or quality of rays emitted. With the development of the Coolidge tube, which can be regulated and kept constant, this difficulty has been overcome and with the interrupterless transformer and its accessories accurate dosage becomes possible. Even with these modern improvements a uniform method to obtain similar results has not been employed. The desire of all has been to elaborate a technic whereby a given amount of rays of a given quality could be employed and repeated, not only by a single operator but by others. With the employment of measuring devices, milliampèremeter, radiochromometer, and radiometer, more accurate dosage was possible, but much depended even here upon the individual operator.[1] This method was a great improvement on the old one of indirect electrical measurement. Recently, MacKee[2] has recorded a method employing indirect measurements (electrical) by which he states it is possible with the use of the modern apparatus accurately to duplicate results. By modern apparatus is meant the employment of an interrupterless transformer, a gasless tube (Coolidge) together with the necessary accessories. The dosage in this method is indicated by units. A unit is the amount of ray known to induce a mild erythema on the delicate skin of a blonde individual or cause epilation of the hair without erythema. The factors are milliampèrage, voltage (spark-gap), distance of the target from the skin, and time of exposure. By varying the time a fraction of a unit or several units may be secured. A unit is obtained by

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[1] For further information concerning the subject the reader is referred to any of a number of books now to be obtained.

Fig. 35

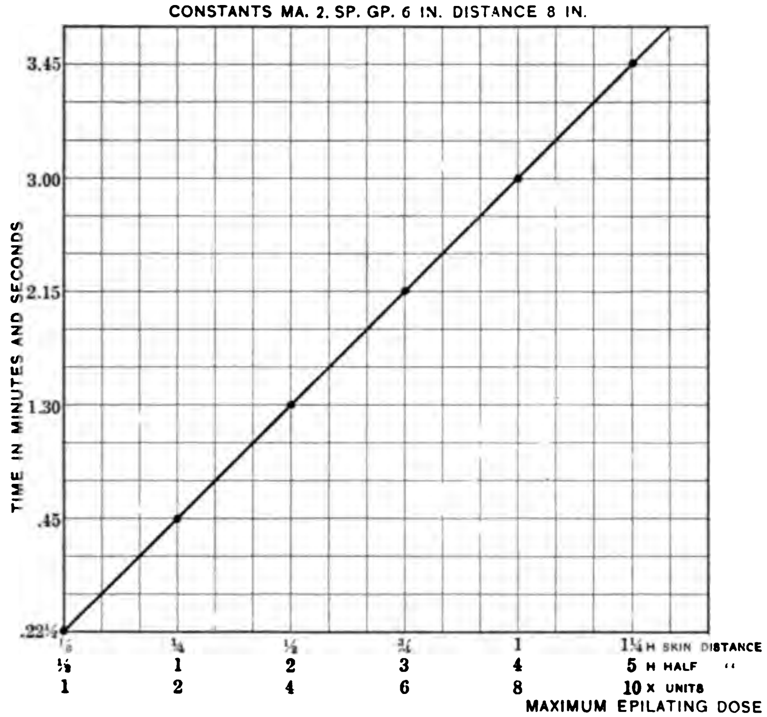

Chart for computing dosage. (MacKee.)

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[1] For more complete description of these factors see papers by MacKee and Remer, Jour. Cut. Dis., 1912, xxx, p. 528; "The Single Dose X-ray Method." Idem., ibid., 1916, xxxiv, p. 296; "The Coolidge Tube and the Corbett Radiometer."

[2] Jour. Cut. Dis., 1919, xxxvii, p. 783; "Aritemetical Computation of Roentgen Dosages."

employing the above factors in the following amounts: Milliampèrage 2, spark-gap (voltage) 6, distance of the target from the skin 8 inches, time of exposure, 3 minutes. Fig. 35 indicates how the dosage may be varied by varying the time. MacKee says that arithmetical computation based on pastile and biologic standardization has been worked out only for the skin unit and for the maximum epilating dose.

Phototherapy.—Since 1896, when Finsen published his first report on the treatment of lupus vulgaris with concentrated rays of light, the therapeutic value of light has been studied both clinically and experimentally in the laboratory by many observers, and the literature of the subject has become extensive.

Phototherapy as employed by Finsen and his followers is based on the principle of concentrating a large number of rays of light on a small area, at the same time excluding the heat-rays so far as possible. A few seconds' exposure to such concentrated light may produce a superficial erythema, but exsanguination of the area to be treated and long exposures (usually one hour) are necessary to secure deep penetration of the light and to produce an acute inflammatory reaction of the tissues. Sunlight, which Finsen employed at first, and which still is used to some extent by his followers, in summer, is too uncertain in its availability for general use, and is apparently less effective than a strong electric light.

The light from a powerful electric arc is condensed by means of a series of lenses so enclosed in a metal tube as to form chambers, which are filled with distilled water to absorb the heat rays. The lenses are made of rock crystal, as glass absorbs too large a proportion of the ultraviolet rays. The collecting lenses are 7 cm. in diameter (larger sizes being difficult to obtain and very expensive), and the rays are brought to a focus about six or seven inches from the lower end of the tube. Surrounding one of the divisions containing water is an outer jacket, through which ordinary cold water circulates, thus preventing overheating of the apparatus. In Finsen's original apparatus he employed an arc light of from 60 to 80 ampères and about 70 volts. In each quadrant of the circle around the lamp was placed a system of condensers, thus permitting the treatment of four patients with one light. This apparatus is suitable for institutions where numbers of patients are to be treated daily. A smaller lamp has been devised by Finsen and Reyn, in which they use practically the same system of condensers, but by employing one lens of shorter focal distance and by so directing the arc that the strongest rays fall directly on the first lens, 20 ampères and 55 volts give results equal in every way to those obtained by the larger apparatus. The lamp is mounted on an adjustable stand, and is much cheaper to install and maintain than the original apparatus, and more suitable for use outside of large institutions.

It is necessary that the light shall fall perpendicularly on the surface and that the surface treated be exsanguinated. This is accomplished by means of pressure with rock crystal lenses kept cool by flowing water and held in position by an attendant or elastic bands.

The frequency of the applications and the duration of each vary for different conditions and for different individuals. For superficial lesions which can be perfectly exsanguinated, one-half hour exposures are often sufficient. For deep-seated lesions from one to two hour seances may be necessary. On each area the treatment is repeated, when necessary, as soon as the reaction has subsided, which it does usually in from one to two weeks.

Following each treatment an inflammatory reaction occurs in from six to twenty-four hours, varying in degree, according to the intensity and duration of the treatment, from a simple erythema to a vesicular or bullous dermatitis, which is sharply limited to the area to which the light was applied, though when the reaction extends at all below the surface there is a surrounding narrow zone of edema. The vesicles and bullæ dry and form crusts, which ultimately fall, leaving only the new-forming epidermis. The process requires, as a rule, from eight to twelve days. The inflammation produced by the light causes no necrosis and no destruction of normal tissue, all of which is conserved. Hence the inconspicuous scars produced and the value of the treatment from a cosmetic point of view. Moreover, the light may be applied freely not only to the morbid area, but also to the apparently normal tissue surrounding it, thus insuring destruction of advancing pathological processes which cannot be recognized clinically. In the normal skin, the reaction on subsiding is followed usually by more or less pigmentation, which usually disappears in ten days or two weeks.

Though the light treatment has been used most successfully in the treatment of lupus vulgaris and other forms of cutaneous tuberculosis, it is of value also in the treatment of lupus erythematosus, alopecia areata, rosacea, vascular nevi, and some chronic inflammatory cutaneous diseases of circumscribed areas. The special technic appropriate for each of these conditions is considered with the general treatment of each. Phototherapy is limited in its applicability by the fact that the rays can penetrate exsanguinated tissue only, and this but to a limited depth. The area treated at one time is small, averaging less than an inch in diameter.

The Kromayer quartz lamp is now used much more extensively in this country as it is compact and can be a part of an office equipment. In this lamp actinic rays are obtained through mercury vapor. The rays from this may be thrown on the skin from a distance without using compression, and may be thus employed in acne vulgaris, alopecia pityroides, chronic eczema and other affections.

Radium.[1]—The first radio-active substances to be isolated were Becquerel-rays, from uranium salts, in 1896. In 1898, Madame Curie and M. Schmidt independently discovered similar rays in thorium. In 1900, Professor and Madame Curie discovered two new bodies, radium and polonium, the latter being much weaker than the former. In the same year actinium was discovered by Debierne, and in 1904 radio-thorium was discovered by Ramsay and Hahn.

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[1] Wickham and Degrais: Radiotherapy. Translation by Dore, 1912. (To this work the author is much indebted.)

Of these various radio-active substances, radium is the one chiefly employed in a therapeutic way. The strength of radium salt is determined by its power of ionization. Taking uranium as the unit of measurement, radium possesses an activity of about 2,000,000. Radium-bromid is a well-defined salt from which all other salts—carbonate, sulphate, nitrate, stearate—are prepared. The bromid, chlorid, and nitrate are soluble, and the sulphate and carbonate insoluble. The sulphates are those employed with varnish, either on metal or cloth, for therapeutic purposes. . As a rule, the radium salt is mixed with a barium salt in the proportion of one to four, which results in a preparation having a radio-activity of 500,000.

From radium at least three distinct kinds of rays are evolved, the principal ones being the Alpha, Beta, and Gamma. The Alpha rays constitute the major portion, about 90 per cent. They are readily absorbed, may be deflected by a magnet, and have only slight penetrating power, a thin layer of metal or rubber being sufficient to intercept them; and therapeutically they induce a dermatitis.

In the Beta group several varieties relative to penetrating power occur, some of which are comparatively soft, others hard. Some of these rays have great penetrating qualities and may be detected through 5 mms. of lead. It is possible also to influence these rays with a magnet.

The Gamma rays closely resemble *x*-rays. They cannot be deflected and they have marked penetrating power. They are able to traverse as much as 10 cms. of lead, which is much greater than the penetrating power of *x*-rays. They pass through the bony skeleton without producing a trace on the fluoroscopic screen.

The varying penetrating qualities of the rays are taken advantage of therapeutically, and filters may be used to eliminate the more superficial rays when desired. A screen of cotton-wool or of aluminum will intercept the Alpha rays. Screens of greater density are required for the others. In the free state the Alpha rays constitute 90 per cent. of the total radiation, the Beta 9 and the Gamma 1. This proportion is changed when the salt is mixed with varnish for therapeutic purposes, as the varnish acts as a screen. From such a source the Alpha rays are reduced to from 1 to 10 per cent.; the Beta rays predominate, in the proportion of from 80 to 90 per cent.; and the Gamma have a percentage of from 1 to 10; so that in treatment the activity emitted is largely in the form of Beta rays.

The method of employment is either to have the radium salt in tubes or spread on metallic plates or pieces of linen and held in position by a special varnish. The tube is made of glass and sealed, and is then enclosed in a second tube of gold, silver, or platinum, of a thickness depending upon the intensity of the radio-activity desired, the tube thus acting as a filter. It is possible by this form of apparatus to treat *in cavitis*, as in the mouth, and also to insert the tubes into tumors. The metal plates upon which the radium salt is held in position by a varnish are made in various shapes, such as flat, concave, convex, cylindrical, spherical, or laminated, to fit the various portions of the surface or cavity to be treated. In those having a linen base the shape naturally is readily changed. In either case there is presented a perfectly smooth, slightly shiny surface, of brownish color, due to particles of radium which show through the transparent varnish. Their color, however, varies with age from yellowish early to brown or black later.

The reactions produced by radium are similar to those induced by *x*-rays. Varying grades of dermatitis, including superficial and deep ulceration, may follow its use. Radium is of special value in the treatment of nevi. It is also employed in several diseases where *x*-rays are valuable, such as epithelioma, keloid, and chronic inflammations.

Liquid Air and Solidified Carbon Dioxid.[1]—These methods of treatment have been developed within recent years. Their action is essentially a caustic one, induced by intense refrigeration. Liquid air is difficult to obtain and hence is not always available. It is kept in double-walled glass containers, which are not sealed, as evaporation must be allowed to occur to prevent explosion. It is applied to the skin on cotton swabs with moderate pressure. The tissue is immediately frozen white, and is very hard and depressed. Within a short period the circulation is resumed, at which time some pain is experienced. Swelling, with redness and bullous formation, soon follows. Later changes depend upon the amount of destruction induced. Carbon dioxid snow, suggested by Pusey, is more convenient, readily obtained, and fulfills much the same requirements. It is only about one-half as cold. It is obtained from the ordinary liquid carbon-dioxid containers, and when the snow is collected it may be moulded into the required shape and applied. These agents are used in the treatment of nevi and benign growths, lupus erythematosus, and small epitheliomata.

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[1] Dade, C. T., Trans. Amer. Derm. Assoc. for 1905. Whitehouse, H. H., Jour. Amer. Med. Assoc., 1907, xlix, p. 371. Trimble, W. B., Jour. Cut. Dis., 1907, xxv, p. 409; New York Med. Record, July 8, 1905. Pusey, W. A., Jour. Amer. Med. Assoc., 1907, xlix, 16, p. 1354. White, C. J., Jour. Cut. Dis., 1908, xxvi, p. 505. Heidingsfeld, M. L., Ohio Med. Jour., 1908, iv, p. 466. Zeisler, J., Zeitschrift, 1908, xv, p. 409, and Jour Cut. Dis., 1909, xxvii, p. 32.

CLASSIFICATION.[1]

THE diseases of the skin are grouped in this work in the following classes.

Class I. Hyperemias and Inflammations.
Class II. Hemorrhages.
Class III. Hypertrophies.
Class IV. Atrophies.
Class V. Pigment Anomalies.
Class VI. New Growths.
Class VII. Neuroses.
Class VIII. Parasitic Affections.
Class IX. Diseases of the Appendages.

The above arrangement conforms closely to the classification suggested by Hebra and modified by Crocker, Duhring, and others. In this edition some rearrangement has been made in some of the classes to improve the sequence and to place new diseases.

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[1] *Cf.* Auspitz System der Hautkrankheiten. Wien, 1881; Bronson, Jour. Cut. Dis., 1884, ii, pp. 161 and 202; ibid., 1887, v, pp. 371 and 427; Duhring, ibid., 1898, xvi, p. 97; Brocq, Annales, 1904, p. 193.

DISEASES OF THE SKIN.

CLASS I.

HYPEREMIAS AND INFLAMMATIONS.

HYPEREMIA.

Hyperemia denotes redness of the skin which may be of varying shades, from bright scarlet to deep bluish-red. It may be active or passive, the former being more important from the dermatological viewpoint. The dividing line between simple congestion and inflammatory reaction is very poorly defined, and it is probable that some of the disorders described under the hyperemias are really inflammations. The passive congestion indicates a stasis of the blood in which case the skin may be of a dark livid color due to local causes or to a general circulatory weakness. Livido recticularis[1] occurs in certain young persons when the skin is exposed to cold air. A marked and persistent livido occasionally occurs in syphilis, tuberculosis, hypothyroidism and other internal disorders. When a widespread and more or less passive congestion is present it is termed cyanosis. The surface temperature in these cases is either normal or subnormal, and in certain instances grave results follow simple injuries. Galloway[2] reported the case of a patient who had suffered from twelve to fifteen years with general and almost constant congestion of the whole cutaneous surface. The slightest injury produced nodules, which were followed by necrosis and ulceration. Death occurred from what appeared to be a spontaneous gangrene of the breast and trunk.

The active hyperemias are described under the general term Erythema.

ERYTHEMA.

Erythema denotes simple redness of the skin, which upon pressure temporarily disappears. There are two classes ordinarily described, the first due to hyperemia, which is short-lived and induces no changes in the skin. In the other forms an exudation occurs and is accompanied by the changes induced by inflammation. It is difficult to draw sharp distinctions between the two classes.

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[1] Adamson, Brit. Jour. Derm., 1916, xxviii, p. 281.

[2] Brit. Jour. Derm., 1903, xv, pp. 235–249. incl.

Erythema Hyperemicum (*seu* Simplex).—**Definition.**—Erythema simplex is a hyperemic disorder exhibiting various shades of redness, diffuse or circumscribed, temporarily disappearing under pressure, the lesions differing in size, hue, and shape according to the extent and the degree of the hyperemia which has been induced.

The active erythemata are divided into two general classes, idiopathic and symptomatic. In the former class are placed all the simple hyperemias having a demonstrable external cause. The latter are surface expressions of systemic disturbances, various febrile disorders, such as cholera, cerebrospinal meningitis, diphtheria, enteric and other fevers. Occasionally a lurid erythema of the axillary or inguinal region may precede by some days the eruption of confluent variola. Vaccination may also be followed in from one to eight or nine days by a macular or more diffuse erythema of the trunk and extremities, usually accompanied by some febrile reaction. These erythemas are important and have to be differentiated from the contagious diseases which they simulate.

The ingestion of certain drugs also induces in susceptible individuals an erythema of this type.

Several forms of idiopathic erythema have been described.

Erythema Traumaticum occurs as the result of friction, rubbing, pressure, scratching, or other external contacts. It is observed, for example, in the part pressed by the pad of a truss, and on the sides of the nose where pressure is exerted by eyeglasses. Long-continued or severe trauma readily converts these simple erythemas into exudative affections.

Erythema Caloricum is a transitory redness of the surface of the skin induced by heat and cold. In erythema induced by solar heat, (*erythema solare*) there is frequently a moderate increase in the pigment, producing freckles and tan. The effects of light are often commingled with those of heat in cases of insolation. The actinic rays of the sun, however, are efficient without the intervention of heat.

Erythema Venenatum denotes that form of erythema induced by a number of external and toxic substances, such as dyes, and vegetable poisons. Among these may be mentioned, cantharides, capsicum, mustard, anilin, chloroform, ether, several dyes used in commerce, and some of the essential oils. Here again the dividing line between a simple erythema and an exudative process is difficult to determine.

Erythema Paratrimma is a term employed to describe the lurid redness which precedes the formation of bed-sores.

Erythema Nuchæ[1] is employed to designate the redness occurring on the back of the neck of infants, seen shortly after birth, and which lasts for a variable length of time.

Erythema Ab Igne.—This form of erythema has been described chiefly by English and American authors. It occurs, as a rule, on the anterior surface of the legs, but may also occur on other parts of the body exposed to undue heat. The lesions occur in annular and gyrate patches. Hartzell[1] describes the condition as a reticulate erythema. The color of the patches varies from a light to a deep red, or even purplish tint, and often permanent pigmentation results as the erythema subsides. The disease occurs in cooks, stokers, and other persons exposing portions of the body to the direct action of heat, and according to Hartzell, in persons whose health is below the normal standard, either as the result of previous illness, alcoholism, or old age. Although, as a rule, this condition is produced by the direct action of heat from stoves, furnaces and the like, cases are recorded which were induced by such things as a hot-water bag. A study of the sections in two of Hartzell's cases showed that the disease is really an inflammatory one, and not a simple staining of the skin with the blood pigments. The epidermis showed a moderate degree of parakeratosis and a slight broadening of the rete layer, and there were a few scattered polymorphonuclear leukocytes lying between the cells of the granular layer. In the corium a moderate amount of cellular exudate, particularly in the vicinity of bloodvessels and about the coil glands was noted. All sections showed deposits of brown, granular pigment in the basal cells of the rete mucosum.

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[1] Stelwagon, Diseases of the Skin, Seventh Edition, p. 145.

Erythema Pernio.—(*Pernio, Chilblains;* Ger., *Frostbeule;* Fr., *Engelure*).—Erythema pernio is an erythematous disorder occurring usually on the hands and feet of persons having feeble peripheral circulation. It is characterized by various shades of red, accompanied by tenderness, itching, or burning sensations, and occurs only during cold weather.

The disorder is readily recognized by observing that the redness disappears on pressure, and also that the parts are actually cool rather than hot, the coolness being appreciable by the touch. Not rarely the involved surfaces are both cool and moistened with sweat. Pernio may closely resemble an early stage of lupus erythematosus, but the latter does not vary regularly with the seasons, as does pernio, which usually disappears in summer and reappears in winter. The two conditions are at times related, as individuals are seen with pernio of the hands and feet and lupus erythematosus of the face. Cases are recorded in which the site of a recurring pernio has become the seat of a typical lupus erythematosus.

Treatment.—The treatment of pernio should be directed to the improvement of the circulation and general health. Warmer clothing to protect the affected parts, together with active exercise, may do much to prevent the recurrence of the disease. Fowler's solution is considered a prophylactic if given in small doses at the beginning of cold weather. The local treatment is by brisk friction and stimulating lotions, such as camphorated soap-liniment; acetous, spiritous, and vinous lotions; or the use of ordinary bay rum of the shops. Afterward the part should be painted with a 50 per cent. solution of ichthyol, well dusted with talcum powder, and bandaged or wrapped in cotton. The severer forms of the disease are considered as a dermatitis calorica.

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[1] Jour. Cut. Dis., 1912, xxx, p. 461.

Erythema of Jacquet.—This is an eruption of the napkin region of infants, the importance of which has been emphasized by Adamson,[1] and the following description has been largely taken from his work. On account of a close resemblance in certain cases to congenital syphilis, the disease should be recognized. The cases are divided into four groups: (1) Simple erythemas; (2) erythemato-vesicular or erosive forms; (3) papular or post-erosive forms; (4) ulcerating forms. The lesions are situated on the convex surfaces, between the flexors, and may occupy or be situated upon the inner parts of the thighs, the perineum, and genitals, or extend over the buttocks, the posterior surfaces of the thighs, the lower part of the abdomen, and upon the calves and heels. The erythema is described as having a deep red and shining appearance, and its selection of convex areas, leaving the flexors, is striking. In the more severe cases, the three subsequent subdivisions occur, consisting of vesicles, papules or ulcerations. The distribution of the eruption upon the prominent convex surfaces suggests local irritation by wet or soiled napkins as a factor in these eruptions. Adamson suggests a disturbance in the vasomotor system as a predisposing factor. In a patient recently seen by the author, malnutrition played an important rôle in the condition. This was a typical case and strongly suggested congenital syphilis. It is said that in children's clinics these cases are far from uncommon. There is no evidence that subjective sensations are present.

Pathology.—The histological changes described by Ferrand and concurred in by Adamson consists of a spongioid transformation of the prickle-cell layer, desiccation, acanthosis, parakeratosis, cell-exudation, and dilatation of the vessels in the papillary layer of the corium.

Diagnosis.—The important question is the recognition of the fact that the disease is not a manifestation of syphilis. The absence of lesions about the mouth and lips, and other signs of congenital syphilis readily settle the question.

Symptomatic Erythema.—In addition to the various general conditions heretofore mentioned as being surface expressions of internal diseases, certain other conditions are described under this title. The simplest form of symptomatic erythema is exhibited as blushing. Ordinarily, this is purely a transitory hyperemia due to emotional causes. Cases occur in which the hyperemia thus induced persists for hours, together with palpitation and other evidences of circulatory disturbances. Here the erythema is symptomatic of either physical or mental disorder.

Erythema Læve.—This is a term now rarely employed but formerly used to designate the shining redness of the skin in edema of the lower extremities.

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[1] Brit Jour. Derm., 1909, xxi, pp. 41 47 incl.

Erythema Fugax is a term applied to transitory redness of the skin, usually occurring in small areas, evanescent in character and similar to urticaria. Its nature seems to be an urticarial erythema.

Erythema Urticans is a variety of urticaria.

Erythema Infectiosum.—The disease bearing this title appears to be an acute exanthematous disorder somewhat resembling rötheln. Apparently it was first recognized by Escherich.[1] Other reporters are Heimann[2] and Shaw.[3] The disease is epidemic and occurs in children, commonly between the ages of four and twelve years. The incubation period is from six to fourteen days. The eruption, as a rule, begins on the face, spreading like erysipelas, having sharply defined outlines. There is intense redness and turgescence of the cheeks. On the extremities, bluish-red, erythematous patches occur. In some cases, in these regions the eruption resembles *rötheln*. It lasts, as a rule, from six to eight days and then gradually fades, first on the face and later on other areas. It is not followed by desquamation or sequelæ.

Closely allied to, if not identical with, erythema infectiosum is an acute exanthematous affection described by Fönss[4] under the title Erythema Annulare in Epidemic Form. Seven cases were observed. In the latter group the incubation period is stated to be four to six days and the eruption on the extremities were distinctly annular.

Roseola Infantilis is sometimes described as a distinct affection, in which there are fever and constitutional disturbance lasting for a few hours or even a few days. The exanthem varies greatly in extent and distribution. It is usually macular or punctate, but may be finely papular. It is most common on the trunk, but may appear on other parts of the body. It may closely simulate scarlatina or measles. This eruption is generally a manifestation of some systemic disorder.

Diagnosis.—The diagnosis of simple erythema is usually not difficult, since without exudation there is absence of all other elementary or secondary lesions of the skin. The members of the idiopathic group are readily recognized on account of their external causes. Those of the symptomatic group may offer considerable difficulty, and the chief obstacles to overcome here are their differentiation from the exanthematous diseases on the one hand, and the determination of the internal cause on the other.

Treatment.—In the management of simple forms of erythema removal of the cause is the chief object. Alkaline washes, zinc oxid and aqua calcis lotion, calamine lotion, or diluted black wash may be employed, followed by the application of a dusting powder. The exclusion of irritants, such as washing the parts with soap and water is advisable. Ordinarily a simple dusting powder is all that is required.

Erythema Scarlatiniforme.—**Synonyms:** Scarlatinoid erythema, Desquamative scarlatiniform erythema, Scarlatinoïde, Erythema punctatum, Roseola scarlatiniforme, "Scarlet rash," Dermatitis scarlatiniformis recidivans. Fr. *Erythème infecteux.*

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[1] München. Med. Wchnschr., 1904, xxiv.

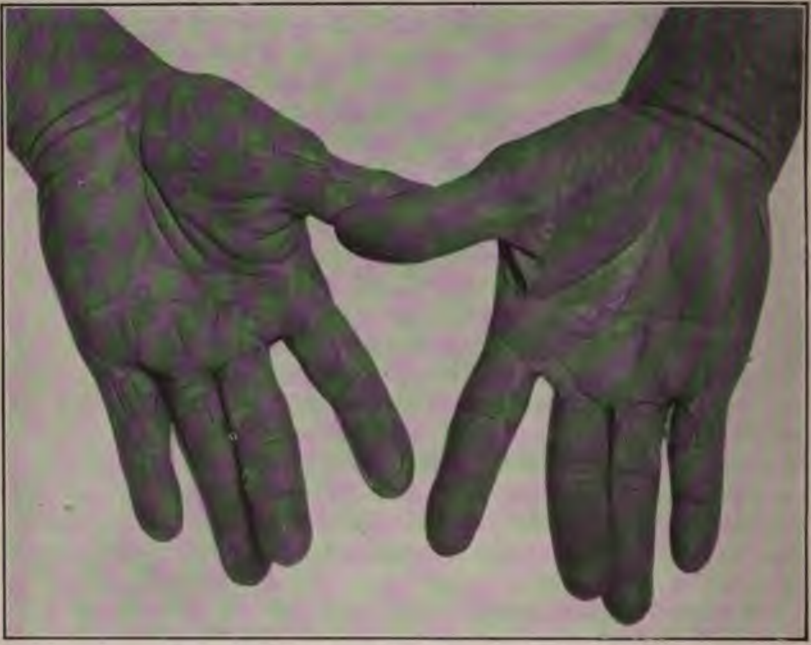
[2] Arch. f. Kinderh., Berlin, 1904, lx, p. 421.

[3] Amer. Jour. Med. Sc., January, 1905, p. 16.

[4] Ugeskrift f. Loeger, January 28, 1915; Abstr. Brit. Jour. Derm., 1915, xxvii, p. 328.

Definition.—Erythema scarlatiniforme indicates an eruption arising from several causes and varying considerably in character, but having a tendency to simulate the rash of scarlatina. This condition has been described as an idiopathic disease, but it is very often demonstrated to be a symptom only of other disorders. Besnier, Brocq, and other French authors describe an *erythème scarlatinoïde*, which is acute in type, and which is always secondary to other infectious diseases, to autotoxemia, or to medicinal or food toxemia; and an *erythème scarlatiniforme desquamatif*, which is subacute in type, and which may be idiopathic, secondary to other infectious diseases, or produced artificially by drugs.

FIG. 36



Desquamation following erythema scarlatiniforme.

Symptoms.—In the acute type, which is the more common of the two forms, the rash may be preceded by a day or two of fever and other evidences of constitutional disturbance, frequently lapsing with the occurrence of the eruption, or it may appear suddenly without premonitory symptoms. The exanthem spreads rapidly, and in a few hours, or at most in two or three days, reaches its full development. The eruption is commonly universal, or at least generalized, but may be more limited in distribution. The rash may be punctiform, macular, or diffuse, and the color may be any of the shades of red, but it is usually a bright scarlet. In some instances, the appearances are those of a typical scarlatinal rash. There are usually some fever (100° to 103° F.), malaise, and other constitutional disturbances, that may vary greatly in intensity, depending upon the cause of the disease in that particular instance. The mucous membrane of the mouth, the tongue, and the fauces may be reddened or be denuded of epithelium, but is not swollen. The nails and hair may be shed, but only in exceptional cases. Desquamation usually begins in from two to six days, sometimes before the disappearance of the rash; and it may even occur on surfaces which had not perceptibly been reddened. The scales are usually furfuraceous, but they may be large and abundant. In rare instances, the entire epidermis of the hand may be shed in glove-like form. Complete involution may require from a few days to several weeks. Rarely, the process terminates in a persistent exfoliative dermatitis. Recurrences are common, but in some instances may be prevented by the discovery of the active cause.

The subacute forms of erythema scarlatiniforme differ from those described above in that constitutional disturbances are less, the rash has a greater tendency to be universal, and, together with the desquamation, may persist for weeks or months, recurrences being common. At times they are so frequent as to make the condition practically continuous and clinically indistinguishable from the milder forms of dermatitis exfoliativa.

Etiology.—Idiosyncrasy is a most important factor in the etiology of those forms of erythema which appear in certain predisposed individuals as a result of causes totally insufficient to produce the same phenomena in most persons; as, for example, in persons exceptionally susceptible to quinin administered by the mouth. The exciting factor is usually if not always some form of toxemia. Among many causes reported are infectious diseases, septicemic conditions, toxemias of varied origin, renal disease, peritonitis, rheumatism, ague in children, gonorrhea, abscess, empyema, serums, tuberculin injections, sewer-gas poisoning (Crocker), certain articles of food, and many drugs, the most important among the latter being mercurial inunctions. The author has seen a typical case following the application of iodoform, and this case had recurrences without renewal of the original cause.

Diagnosis.—It is important to distinguish this rash from that of scarlet fever. Commonly, the diagnosis is not difficult, but occasionally the greatest difficulty is encountered. As a rule, in erythema scarlatiniforme the constitutional symptoms are slight; the rash appears rapidly, beginning irregularly on any part of the body; the lesions are exclusively cutaneous; desquamation begins early (in from two to three days), and is extensive; the fauces, though red, are not swollen; and there is absence of the "strawberry tongue," of leukocytosis, and of all history of contagion. Occasionally, the rash may resemble that of measles or rötheln; but the history of the case and the absence of other symptoms peculiar to these affections should make the diagnosis clear. As a rule, an examination of the rash alone is insufficient, and a diagnosis of erythema scarlatiniforme should not be made until other exanthemata have been considered and excluded.

Treatment depends entirely on the underlying cause or condition. Toxins present should be eliminated as rapidly as possible. The eruption itself rarely calls for treatment. If there be itching or burning sensations, a simple dusting-powder, with or without an antipruritic or a soothing lotion or ointment, may be used to make the patient more comfortable. In extensive cases, the unguentum aqua rosæ gives much relief. In the subacute recurrent cases, careful search should be made in the alimentary tract, and good results have been obtained by so regulating the diet as to promote free elimination and prevent the accumulation of waste products.

Prognosis.—As a rule, the eruption disappears promptly and the general health of the patient is unaffected. Recurrences are frequent, and in some cases terminate in a more or less persistent exfoliative dermatitis.

Shedding of the Skin (*Deciduous skin, Keratolysis*).—Cases are reported of individuals whose skin is shed periodically, like that of a serpent. Dr. Hyde had the opportunity of observing the symptoms in the case reported by Frank and Sanford[1] during several of the periods in which the patient's skin was exfoliated. The subject was thirty-three years of age, well formed and apparently in perfect health. No cause for the skin shedding could be found. He stated that ever since he could remember, and certainly since he was eight years old, he had had peculiar symptoms, which began between 3 and 9 P.M. of July 24 of each year. He would suddenly experience a feeling of lassitude or weakness, followed by muscular tremors, nausea, and vomiting, with rapid rise in temperature. Accompanying these symptoms, the mucous membranes were hyperemic, and the skin became hot, dry, and destitute of perspiration. After three or four hours, the acute symptoms began to subside, but the skin remained red for thirty-six hours longer. The shedding of the skin began usually on the second or third day, and was completed in from three to ten days. The mucous membrane of the tongue and mouth exfoliated on the third day. The epidermis was removed from the trunk and arms in large sheets on the sixth day, and from the remainder of the body, except the hands and feet, within the next three days. Complete casts of the hands and feet were shed on the seventeenth day, and the nails all came off within a month from the beginning of his illness.

This case was observed the following year and reported by Sligh.[2] His report confirms the facts above recorded. Similar cases are reported by Stelwagon,[3] Stone,[4] and others. Cases of constant exfoliation of the skin rather than periodic have occurred. These are rare, and are described under the term *keratolysis exfoliativa congenita.*

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[1] Amer. Jour. Med. Sci., August, 1891.

[2] Internat. Med. Mag., 1893, p. 463 (two plates).

[3] Diseases of the Skin, 5th Ed., p. 143.

[4] Jour. Amer. Med. Assoc., September 1, 1900, p. 557 (two cuts).

Erythema Intertrigo.—**Synonyms**: Intertrigo, Eczema Intertrigo, Chafing. Fr., *Erythème intertrigo.*

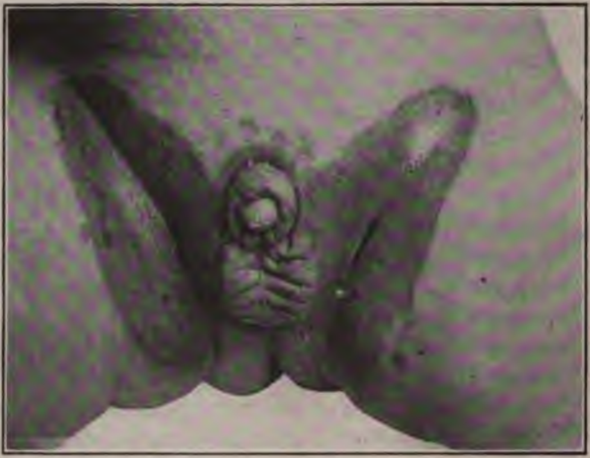
Definition.—Erythema intertrigo is a hyperemic condition of those cutaneous and muco-cutaneous surfaces which are in constant apposition, and between which there is a hypersecretion and retention of sweat.

Symptoms.—The erythema is limited to parts of the integument which lie in contact with each other. The sites of such contact in the human body are the axillæ, the groins, the cleft between the nates, the intermammary and inframammary spaces in women, the superior and inner faces of the thighs, the scroto-femoral and the labio-femoral clefts in the sexes respectively, the flexures of the joints, and in especially obese individuals all those parts where the integument is thrown into fleshy folds, as about the neck of infants, and even over the crest of the ilium in fat subjects. In these localities, the disorder, beginning as an erythema traumaticum, proceeds by its irritative effects to stimulate the secretion of sweat, which is freely poured out between the adjacent folds of the skin and may there be imprisoned temporarily. The surface, heated and reddened, is also somewhat macerated by the effused perspiration, and the latter, when chemically altered, as it is frequently under these circumstances, adds still further to the original disorder. The ground is thus well prepared for an exudative process, which not infrequently supervenes in the form of a dermatitis; but the disorder may be limited to mere hyperemia with hyperidrosis, and disappear before the supervention of actual inflammation. Superficial abrasions of the macerated epidermis occur, and one such abrasion is always especially significant. It is the linear and superficial excoriation which marks the line of deepest contact of the two apposed surfaces of the skin at the bottom of the angle formed by the two. An offensive odor usually proceeds from the part, in consequence of the chemical changes in the secreted fluid. The secretions of an intertrigo stain but do not stiffen the linen of the patient, and they thus differ from the serous fluid poured out in an exudative dermatitis. The subjective sensations are those of heat and tenderness.

Etiology.—The disease is chiefly induced by heat, friction, and moisture. These causes occasionally coöperate. The heat may be merely that of the natural temperature of the body, or it may be increased by that due to season or climate. The friction may be merely that originating between the surfaces in apposition, or may be increased by clothing or other articles worn next to the skin. The moisture which produces maceration of the epidermis is the sweat-secretion, this being stimulated by heat and friction. As aggravating causes may be named physiological excretions and secretions retained in contact with the surfaces affected with an intertrigo. Thus, the feces, the urine, the milk in nursing women, retained lochial, menstrual, and similar discharges, and glycosuria are all efficient in this regard, and are particularly likely to induce that form of dermatitis to which the intertrigo then plays a subordinate part. Fleshy and gouty persons chiefly suffer from these accidents.

Diagnosis.—The recognition of a simple erythema intertrigo is a matter of no difficulty if regard be had to the exciting and aggravating causes enumerated above and to the special localities in which such hyperemia generally originates. If an eczema or a dermatitis supervene, the fact will appear from increased subjective sensations (usually severe itching), from an infiltration of the affected integument, and from the appearance of those lesions and discharges which are significant of these forms of inflammation of the skin. It must be remembered that transition from a simple erythema to a dermatitis of these regions is of frequent occurrence. Erythema intertrigo may occur as a mild form of dermatitis seborrheica. The disease is to be differentiated, also, from tinea cruris, the latter being more inflammatory, exhibiting scaling, and under microscopic examination of the scales an epidermophyton fungus is found. In babies, congenital syphilis and the erythema of Jacquet are to be differentiated. The deeper involvement by both of the latter disorders, as a rule, and concomitant symptoms in one of them, serve to differentiate them.

Fig. 37



Erythema intertrigo in an infant.

Treatment.—Intertrigo is an exceedingly common affection of the skin, and it occasionally proves of great annoyance to those suffering from it. Gouty patients always require limitation of the diet, and often also medication with alkalies and mercurial cathartics.

The affected surfaces should be cleansed gently by ablution with soap and warm water, and the offensive odor of the secretions remedied by the addition to the water of a weak solution of formalin, of phenol, or of the dilute liquor sodæ chlorinatæ. The parts are then to be carefully dried with a freshly laundered towel or soft gauze, and afterward one of the dusting-powders very thoroughly applied. To be of service, these powders must be impalpable, and, if compounded by a druggist, be sifted through fine silk bolting-cloth. The articles chiefly used for this purpose are zinc stearate with acetanilid, bismuth, starch, zinc oxid, French chalk, lycopodium, or, when an antipruritic effect is desired, camphor. Combinations of several of these are at times effective. The formula of McCall Anderson is highly esteemed:

℞—Zinci oxid. pulv.,	℥ss;	16	
Camphoræ pulv.,	ʒjss;	6	
Amyli pulv.,	℥j;	32	M.
Sig.—Anderson's dusting-powder.			

For the purpose of absorbing excessive perspiration, magnesium carbonate is the most effective of all the powders.

The following is the formula for a dusting-powder recommended by Klammann:[1]

℞—Talc. venet. pulv.,	ʒv;	20	
Acid. salicyl.,	gr. iij;		20
Magnes. ust. subtil. pulv.,	ʒjss;	6	M.
Sig.—Dusting-powder.			

Finely bolted starch answers well alone or in combination with some of the other articles above named. Unna's salve-muslins and pastes will be found effectual and neat applications in many forms of intertrigo.

The affected surfaces of the skin must also be separated in order to prevent further friction. A thin strip of lint, gauze, antiseptic cotton, or medicated wool may be used for this purpose, and must be inserted as far as the deeper portions of the cleft in which the secretion chiefly forms. Occasionally, it will be found useful to anoint this absorbent layer with borated cold-cream salve or with vaseline. Where an astringent effect is desired, lycopodium or other dusting-powder may be compounded with tannin, alum, or similar substances. The list of lotions also at times may be consulted with advantage. Thus, cologne-water, saturated aqueous solutions of pyoktanin blue, weak spirit lotions containing tannin, aromatic wine, or zinc oxid and lime-water may each be serviceable. Lastly, equal parts of lime-water and olive-oil, spread thickly upon linen, will possibly give more relief than other articles named, the chief objection to it being the consequent soiling of the patient's linen.

Erythema Multiforme.[2]—**Synonyms.**—Erythema exudativum multiforme. Fr., *Erythème polymorphe.*

Definition.—Erythema multiforme is an acute, inflammatory disease characterized by crimson-red or purplish-red macules, pustules or nodules; occasionally by vesicles or bullæ, and rarely by pustules. The lesions are variously grouped, or isolated and prone to recurrence.

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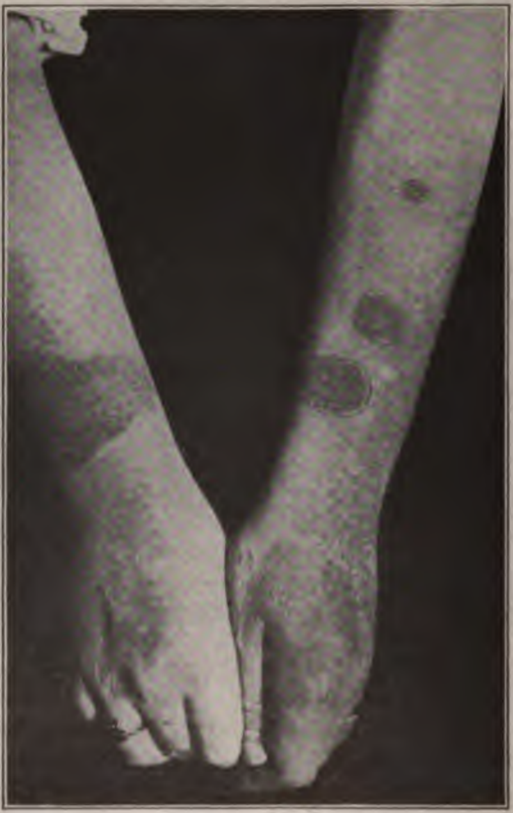
[1] Hebam. Kalend., Obstet. Gazette, March, 1882.

[2] Symposium on the Toxic Dermatoses, Jour. Cut. Dis., 1912, xxx, pp. 119–167 incl. Discussion on Erythema Multiforme by Members of the Royal Society of Medicine, London, Brit. Jour. Derm., 1912, xxiv, p. 429.

Erythema multiforme as described by Hebra is a comparatively simple disease, usually limited to the hands, forearms, face and neck, which runs its course in two to four weeks, and is commonly recurrent. It is of unknown origin. A very large number of cases with symptoms simulating those found in multiform erythema have since been recorded and a description of the disease, therefore, includes more than that outlined above. A clear conception of the disorder may be had by studying it first as it appears in mild form with superficial lesions situated on particular areas, prone to recurrence and practically limited to the skin in its manifestations, and of unknown etiology. Second, as a symptomatic erythema of several diseases of known infectious nature. Third, as a surface expression of visceral disease. Fourth, as cutaneous manifestations produced by the ingestion of many different drugs and the injection of serums and, finally, as a symptomatic

FIG. 38

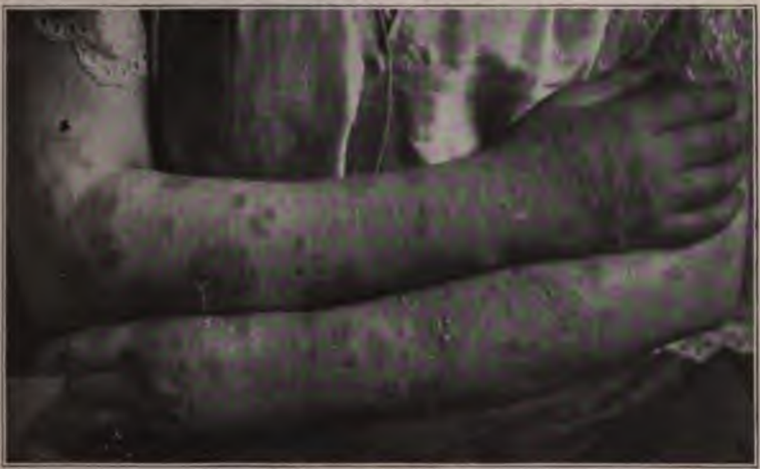


Erythema multiforme.

erythema, the lesions corresponding in type to those of multiform erythema, but of such severe grade that difficulty is experienced in distinguishing them from certain forms of pemphigus.

Symptoms.—In the idiopathic variety the most common lesions are edematous looking macules, flattened papules, and large, flat nodosities. Occasionally, vesicles and bullæ are present. While multiformity is the rule, one type of lesion usually predominates in each case. The eruption is nearly always symmetrical and is usually found on the dorsum of the hands and feet, the legs and forearms, and often on the face and neck. It occurs exceptionally on other parts of the body but rarely upon the mucous membrane of the nose, mouth and conjunctiva. It has been seen on the sclera. The eruption, which is generally recognized in well defined patches, usually begins with pinpoint to finger-nail sized macules of dark, bluish or purplish-red shade, which in the course of some hours exhibits tumefaction in various degrees, thus producing the papules and nodules already mentioned.

FIG. 39



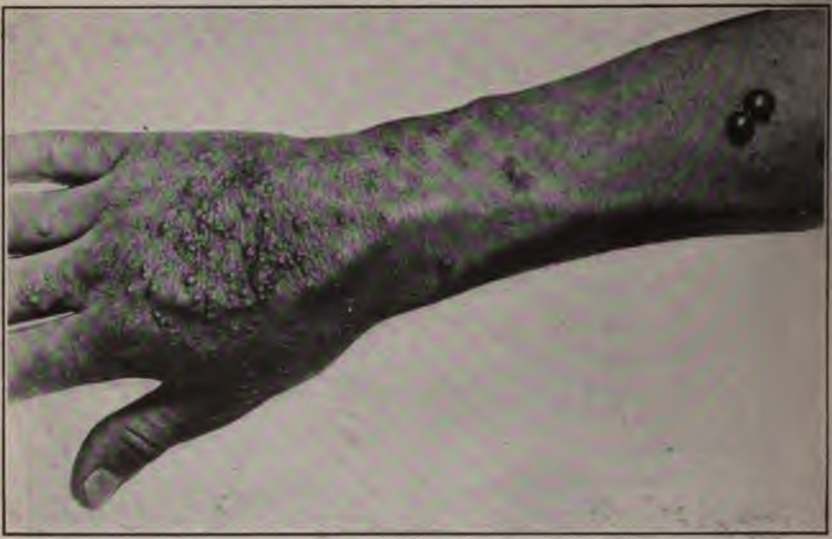
Erythema multiforme.

The disease runs its course in from a few days to several weeks, the average simple case lasting from two to four weeks. Recurrent attacks through a period of years are not uncommon and these are likely to appear in the spring and autumn. At the height of the process the coloring matter of the blood may escape from the vessels and produce in the lesions the color characteristics of simple bruising. Peculiar shades of reddish-black, purplish-blue, red, yellow, orange and black-and-blue are exhibited. The lesions occur in different shapes, sizes and shades and the number of names used to designate their several processes require explanation.

The term *erythema annulare* or *circinatum* is used to designate the lesions having a depressed center and an erythematous margin forming a ring. Occasionally these rings are arranged concentrically. When several rings coalesce by peripheral extension gyrate figures are formed and this is termed *erythema figuratum*. *Erythema marginatum* indicates a form in which a distinctly elevated and well defined marginal band is left as the sequel of the erythematous patch. *Erythema papulatum* or *papulosum* and *erythema tuberculatum* or *tuberculosum* refer to that type of the disorder in which the lesions are papular or nodular.

Occasionally severe itching is present and the lesions have an urticarial appearance. This type is designated by the term *erythema urticatum*. *Erythema vesiculosum*, and *erythema bullosum* are rare forms in which the exudation is sufficient to produce vesicles and bullæ in addition to the edema which occurs in some common types. Vesicles or bullæ may be situated at the center or periphery of an erythematous patch or occupy the center of a papule or nodule. As a rule, the fluid from these lesions is absorbed.

Fig. 40

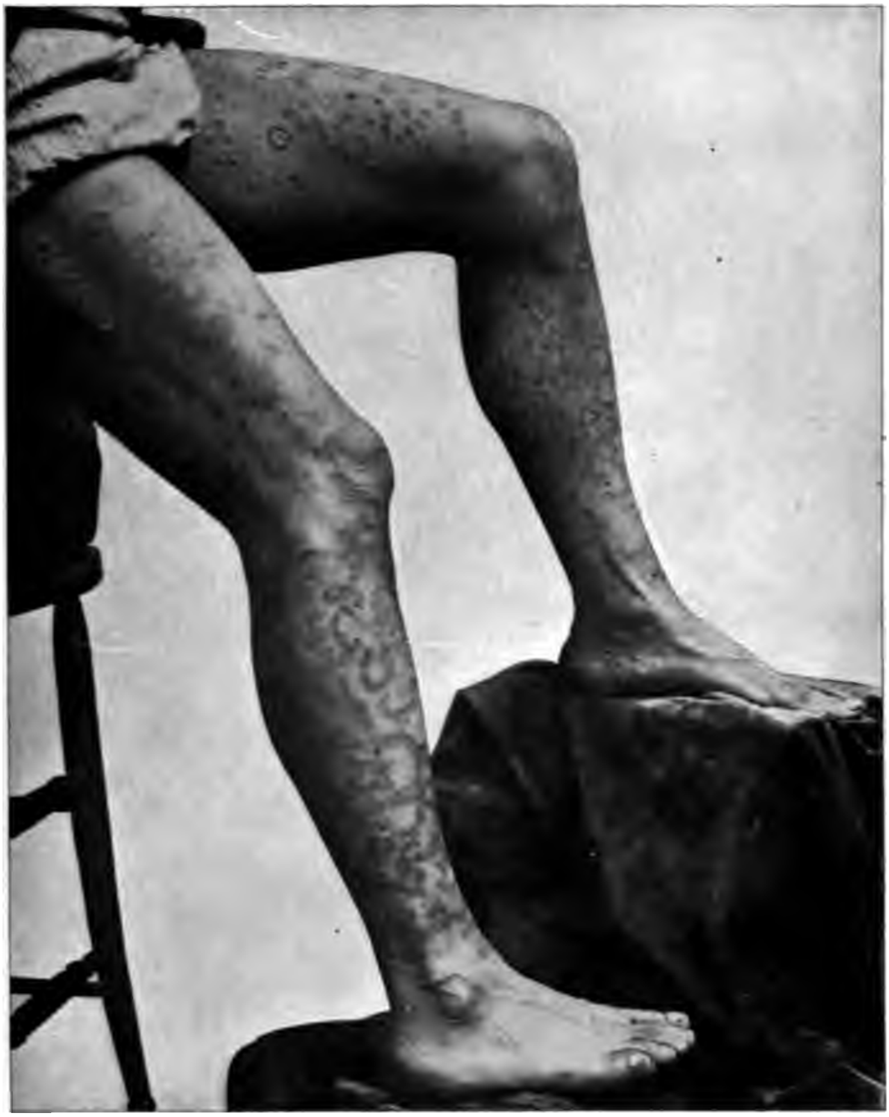


Vesico-bullous erythema multiforme.

Erythema iris (*Herpes iris*, *Hydroa vésiculeux*). In this variety the lesions occur in variously colored concentric rings usually on the dorsal surface of the hands, though the mouth, face and even the trunk are occasionally involved.

The early lesion may be a light-red macule upon which a single or several vesicles or bullæ develop or the latter may occur without the erythema. In the former case there soon appears a ring of vesicles or bullæ in the surrounding advancing erythematous border. The latter continues to advance and new rings of lesions occur until several concentric rings are formed. As the lesion enlarges peripherally the center undergoes resolution. The vesicles or bullæ dry and disappear

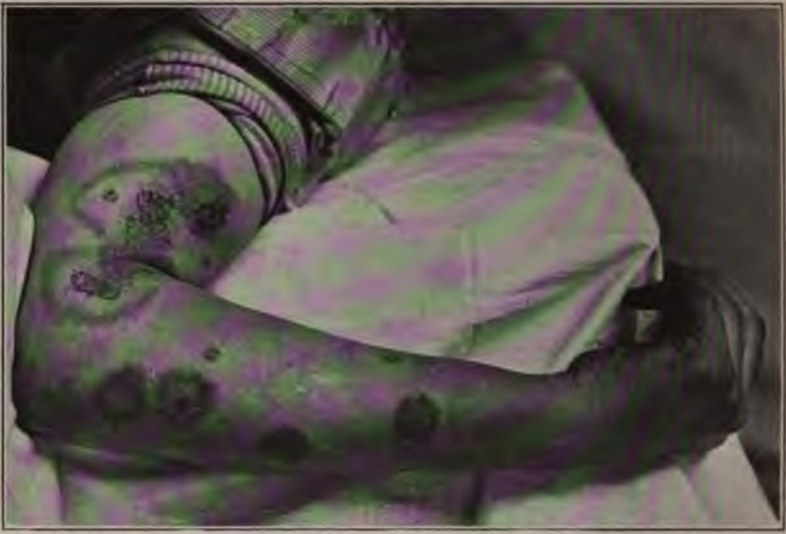
FIG. 41



Erythema multiforme, circinate type.

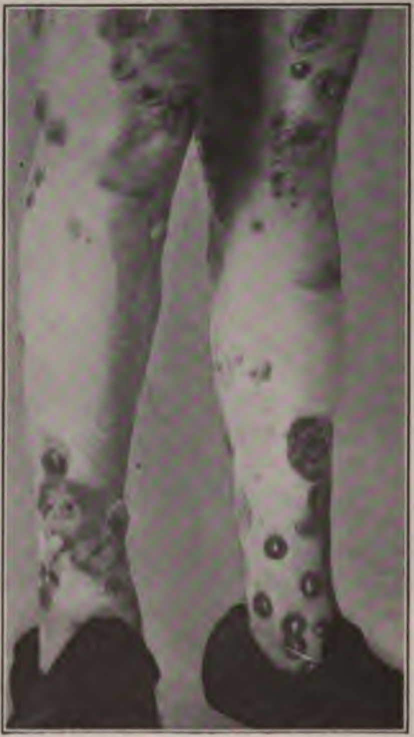
and the color changes occurring are those seen following a bruise. From periphery to center the color varies from bright red to dark or purplish-red, yellow, brown or darker hues. Duhring describes an

FIG. 42



Erythema multiforme, iris lesions. (Fordyce.)

FIG. 43

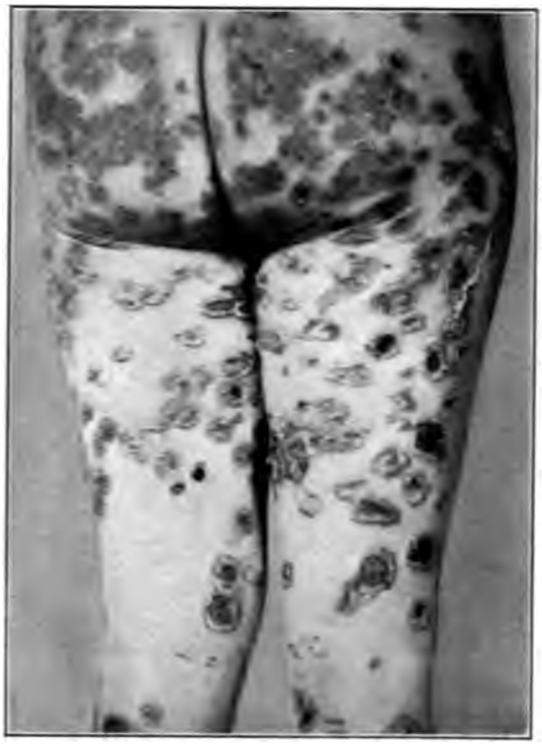


Erythema bullosum. (Howard Morrow.)

uncommon variety in which large patches are formed on the trunk chiefly comprised of concentric rings of vesicles or bullæ without accompanying erythema and the peculiar color changes above described. The patches formed as above described may be an inch or more in diameter and limited in number or many may form in different areas.

The subjective sensations are usually mild. Occasionally the mucous membranes are affected in other types. When involved the lesions occur as hyperemic macules, papules or bullæ and are seen upon the

FIG. 44



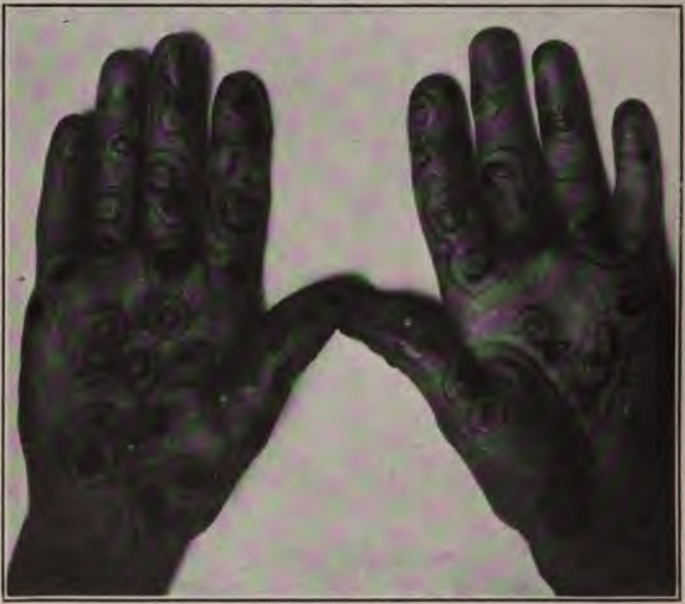
Erpthema bullosum. (Howard Morrow.)

upper and lower lip, the inner face of the cheeks, the gums and hard palate, and at times the pharynx. These lesions begin as pin-point to large pea-sized, isolated, deeply-colored elevations. In severe cases painful erosions form. The parts then become swollen and tender and when hemorrhagic erosions occur ulceration may follow, which is usually superficial in character.

The subjective symptoms are exceedingly mild and quite out of proportion to the severity of the eruptive manifestations. Slight burning and smarting are occasionally present.

The term "multiform" given the disease is well illustrated in most cases. Lesions representing different types occur simultaneously or successively and individual lesions change their type. In most instances the disorder above described is a simple one, disappearing in the course of a few weeks without sequels. The erythema multiforme symptom-complex may accompany such diseases as cholera, typhus, glanders, measles, septicemia and malaria. It is also seen comparatively often as the result of the ingestion of drugs, chief among which are quinin, arsenic, belladonna, chloral, salicylic acid and iodin and bromin compounds. It sometimes follows the injection of certain serums, particularly diphtheria antitoxin. It occasionally occurs in connection with visceral disease, in which cases it is attended by general malaise, fever, gastro-intestinal disorders, limited involvement of the articulations, organic changes in the heart, lungs and kidneys, all of which conditions have been noted as coincident or causative phenomena. The visceral involvement in connection with lesions of multiform erythema was early emphasized by Lewin[1] and more recently by Osler[2] and Galloway. Osler has shown that the cutaneous symptoms may be merely surface expressions of the visceral disorder. In 29 cases studied by him, there were three sets of symptoms: (*a*)

FIG. 45



Herpes iris.

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[1] Berl. klin. Wchnschr., 23, 1896.

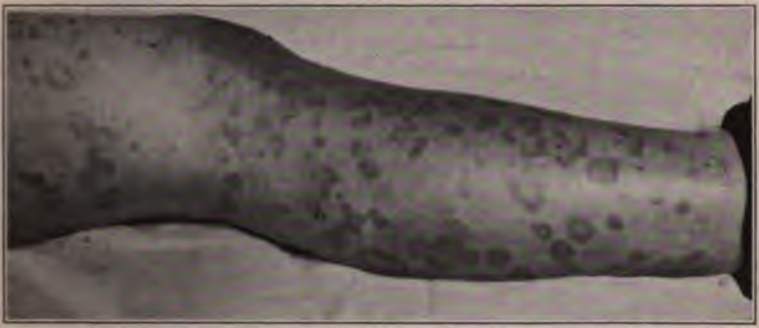
[2] Amer. Jour. Med. Sc., 1895, n.s. cx, p. 629; Brit. Jour. Derm., 1900, 12, p. 227, and Amer. Jour. Med. Sc., 1904, cxxvii, p. 1.

Polymorphous skin lesions, including acute circumscribed edema, urticaria, purpura, and ordinary forms of erythema multiforme; (*b*) visceral lesions, including (1) gastro-intestinal crises, in which severe colic, with or without vomiting, diarrhea, or bloody stools, was frequent, (2) hematuria and nephritis, (3) hemorrhages from mucous surfaces, (4) cerebral symptoms, (5) pulmonary complications; and (*c*) infiltration of synovial sheaths and periarticular tissues and arthritis. In some of these cases a given visceral lesion has been accompanied at different times in the same individual by each of the types of cutaneous lesions.

A case well illustrating the multiform erythema symptom-complex occurring as the result of infection was noted by Corlett.[1]

Etiology.—The affection occurs commonly in the spring and autumn and most often in the young, or early periods of life. Mackenzie[2] had called particular attention to the relationship of erythema multiforme to rheumatism and to purpura rheumatica. Adamson[3] believes that erythema multiforme, purpura rheumatica and Henoch's purpura are probably modifications of one and the same malady and, furthermore, believes that the question is unsettled as to whether it is a toxemia or due to microbic infection.

FIG. 46



Erythema multiforme.

Pathology.—Erythema multiforme is essentially a hyperemia of the integument, that under certain influences advances more or less rapidly to the stage of a mild grade of inflammation, with consequent exudation. Crocker, examining a patch of erythema tuberculatum, recognized merely a cell-effusion in the upper part of the corium extending sparsely below, and then chiefly along the ducts and follicles. There was slight rete-proliferation. The principal histological changes, accordant to Unna, are dilatation of the vessels, perivascular cell-infiltration, and edema of the collagen and elastin; the changes being chiefly noted in the papillary body. Secondary changes in the epidermis consist of edematous swelling of the epithelium, with moderate proliferation and dilatation of the lymph spaces. In two cases of the moist type, Pardee[1] found simply an acute exudative inflammation of the upper part of the corium. Török[2] and Kreibich[3] also found the condition to be a simple dermatitis and not an angioneurosis.

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[1] Jour. Cut. Dis., 1908, xxvi, p. 7; Erythema Exudativum Multiforme: its Present Significance, with Report of a Case of Erythema Circinatum Bullosum et Hemorrhagicum Following a Gunshot Wound, Apparently Due to Streptococcus Infection and Terminating Fatally. Bibliography.

[2] Brit. Jour. Derm., 1896, viii, p. 116.

[3] Ibid., 1912, xxiv, p. 429.

Diagnosis.—The tendency of the disease to bilateral symmetry, the recurrence of lesions evidently dating from several periods in which successive crops appear, and the absence of all history of external injury will usually suffice to establish a diagnosis. Important points to be considered are: the recognition of the vivid coloring of most of the lesions; their edematous character; their symmetry, as a rule; the pigmentation following those situated on the lower limbs; their frequent association with rheumatism or rheumatoid pains, febrile phenomena, malaise, and other constitutional disturbances. From urticaria, it is distinguished by the type of the lesions; the wheals of the latter are smaller, more whitish centrally, more closely packed together, less symmetrical, rarely grouped, and, as a rule, more acute than those of multiform erythema. Cases difficult to assign to either disease are common, and an error in either direction is not serious. Certain rare forms are difficult to distinguish from dermatitis herpetiformis. Whitfield[4] reports cases of multiform erythema of the papulo-pustular type which were mistaken for variola. The author has seen two similar cases. Finally, certain rare forms are practically indistinguishable from pemphigus.

Treatment.—As in the majority of instances the disease under consideration undergoes spontaneous resolution within a few weeks, the treatment is directed toward prevention of recurrence. For the immediate relief of the subjective symptoms a dusting powder and sedative or antipruritic lotion, such as recommended in eczema, may be employed. In most cases a mercurial followed by a saline laxative is of service. When arthritic complications are present alkalies and diuretics are recommended in addition to acetyl salicylic acid. So-called intestinal antiseptics, such as salol and kaolin have been recommended. Gilchrist reported favorable results from the use of vaccine consisting of staphylococcus albus. Vaccines made from organisms obtained in the intestinal canal have also been employed. The local lesions in the mouth require appropriate treatment. Fifteen drops of iodized phenol in one-half glass of water is of benefit when painful erosions are present. Soothing and alkaline mouth washes should also be used.

Prognosis.—It will be gathered from what has been said that the prognosis is usually favorable, but necessarily varies with the constitutional disease of which the erythema may be a mere symptom. The malady may relapse in susceptible individuals at those periods of the year when it is observed most frequently. Fatal cases, such as the ones quoted by Corlett and others, naturally make the prognosis in certain cases unfavorable.

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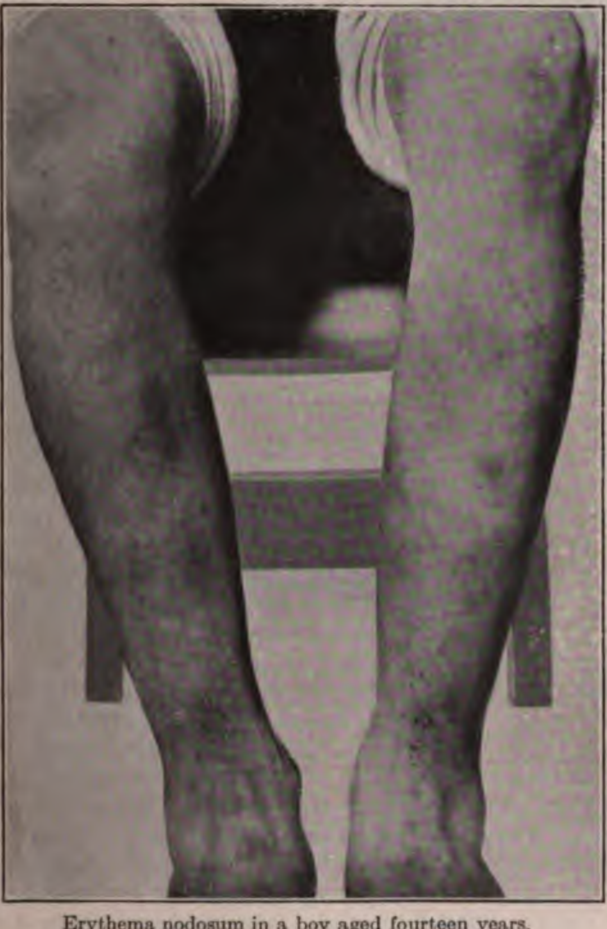
[1] Johns Hopkins Hosp. Bull., 1898, ix, p. 165.

[2] Archiv, 1900, liii, p. 243.

[3] Ibid., 1901, lviii, p. 125.

[4] Brit. Jour. Derm., 1903, xv, p. 273.

Fig. 47



Erythema nodosum in a boy aged fourteen years.

Erythema Nodosum (*Dermatitis contusiformis; Fr., Erythème noueux*) is regarded by some authors as a distinct affection, and by others is classed as a form of erythema multiforme. Unna sees a distinction between this disease and erythema multiforme in the fact that the lesions of erythema nodosum never widen concentrically, never produce bullæ, and never exhibit annular vesicles, although lesions of both types may occur in one patient. Erythema nodosum rarely if ever recurs, and, while there are many points of resemblance, it will be described here as a separate affection.

Symptoms.—The disease is usually ushered in with some constitutional disturbance, and is commonly associated with joint pains. The lesions occur chiefly on the legs below the knees, on the anterior surface. They are semiglobular, pea- to fist-sized nodules, pale-red, pinkish to livid blue in color, tender on pressure, and exhibit in their involution the variegated hue seen in a contusion. In addition, they may occur, though rarely, on the arms, trunk, and face. Though occasionally so soft to the touch that fluctuation seems to be present, they do not terminate by suppuration. At times, they appear to develop in crops. They are usually tender on pressure, and often painful. They may disappear in a fortnight, but occasionally remain for several weeks. The petechial appearance of the spots where they have existed is that of the characteristic black-and-blue mark. In rare instances the mucous membranes may be involved.

Etiology.—Like multiform erythema, the disorder occurs most often in the spring or autumn, and most frequently in women, and is very commonly associated with rheumatoid pains. The eruption may occur in tuberculous subjects,[1] and it also occurs often in the poorly nourished and ill-housed. Neara and Goodbridge[2] recorded a case of erythema nodosum in a patient the subject of acute and fatal tuberculosis. Other causes cited are malarial chills, temperature changes, endocarditis, urethral irritation (blenorrhagic, instrumental), the ingestion of drugs, alcoholic excesses, and dentition. Some stress has recently been laid on syphilis as an etiological factor. Joynt[3] reports erythema nodosum following measles in from ten to fourteen days in 9 out of 300 cases. As cases not infrequently develop following a pharyngeal or tonsillar infection, it seems probable that the causative factor responsible for the so-called rheumatic affections starting in this way is identical.

Pathology.—Duhring[4] regards the disease as in most instances infectious. Mackenzie believes true erythema nodosum to be a manifestation of rheumatism, and in view of the modern conception of the infectious nature of many cases of rheumatism some light is obtained concerning the pathogenesis of the disorder. The histological study shows the ordinary signs of inflammation, including vascular dilatation, small, round-cell infiltration in the papillary and subpapillary layers of the corium, choking of the lymphatics and bloodvessels, and edema. Various micrococci and Demme's bacillus[5] have been discovered in the blood of patients suffering with erythema nodosum.

Rosenow[6] has isolated a microörganism from the subcutaneous nodes and blood of 6 patients suffering with erythema nodosum. He describes the organism as a polymorphous, sometimes clubbed, often curved, barred diplobacillus; and by injecting this organism intravenously in animals has produced subcutaneous lesions, exhibited as hemorrhages, followed by infiltration and migration of leukocytes, with enlargement of the regional lymph-glands. From these areas the organism has been repeatedly isolated.

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[1] Morfan, La Presse médicale, June 26, 1909, p. 457: "Erythema Nodosum and Tuberculosis." Foerster, Trans. Amer. Med. Assoc., Sec. on Derm., 1914, p. 328: "The Association of Erythema Nodosum and Tuberculosis"

[2] Amer. Jour. Med. Sci., 1912, cxliii, p. 393.

[3] Brit. Med. Jour., April 15, 1911.

[4] Cutaneous Medicine, Part II, p. 276.

[5] Fortschr. der Med., 1888, vi, p. 241.

[6] "Etiology of Erythema Nodosum: Preliminary Note;" read before the 38th Annual Meeting of the Amer. Derm. Assoc., June, 1914.

Diagnosis.—The characteristic picture presented by erythema nodosum, with the bilateral localization of the lesions, usually presents no difficulty in diagnosis. Syphilitic nodes and gummata are distinguished by the absence of pain; the fewness of the lesions; their overlying integument being untinted save when actually softening and approaching disintegration; their obvious subcutaneous site; their unilateral distribution; and the concomitant symptoms of late lues. Erythema induratum of Bazin is much more chronic, unattended by subjective sensations, and has a tendency to break down and ulcerate. It must also be remembered that potassium iodid and bromid produce lesions resembling this disorder, and in any case contusions and bruises should be ruled out. This latter question, at times, has a bearing in medico-legal questions, especially in the case of young children.

Treatment.—As a rule, rest in bed is essential. This, with a light diet and free elimination, and the administration of sodium salicylate or aspirin, is all that is required. It is well to combine the sodium salicylate with bicarbonate of sodium, giving 0.33 of the former and 0.66 of the latter four times daily. A mercurial purge, followed by a saline cathartic, to begin with, is of value. Locally, the application of heat will relieve the tenderness, and bandaging promotes absorption.

Prognosis.—As a rule, in from two to six weeks the disease has run its course and clears up. Some patients have much temperature and suffer with sufficient pain to cause them great discomfort; but even in these cases the prognosis is good within the above-mentioned time limit.

Erythema Perstans.—Several disorders have been described under this title. Some of them were probably examples of multiform erythema of unusual duration. Under the title of *Erythema perstans faciei*, Kriebich[1] described a toxic erythema developing on lupus erythematosus as a sequel of influenza-pneumonia. This type is described under the title *Erysipelas perstans faciei* (*Kaposi*). Under the title *Erythema figurata perstans*, Wende[2] described a group of cases presenting symptoms which resembled multiform erythema. The same author[3] later reported two cases under the title *Erythema perstans presenting circinate lesions*, as follows: The disease begins with isolated papules which spread peripherally and fade in the center, forming circinate lesions. The outer half of the advancing margin is described as being smooth and slightly raised, while its inner edge presents scaling. Some pigmentation follows. The lesions vary in size from that of a silver twenty-five cent piece to that of the palm. The configuration also is variable, gyrate forms being common owing to confluence of circinate or annular lesions. Subjective sensations are sometimes noted. Great variation occurs in the length of time required for the development of the lesions. Exacerbations also are inconstant as to time, the rule being that some of the lesions remain all of the time. The disease may occur at any age.

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[1] Zeitschrift, 1908, Bd. 15, Heft. 8, p. 522.

[2] Trans. Sec. on Cut. Med., Amer. Med. Assoc., 1908, p. 75.

[3] Jour. Cut. Dis., 1906, xxiv, p. 241.

Mook[1] reported two cases similar in clinical manifestations and histological findings and suggests that they are chronic *erythema multiforme* of peculiar type.

Under the title *Erythema perstans* Fordyce and MacKee[2] and others have reported cases representing a type presenting erythema with marked pigmentation. The lesions or sequels were constantly present over varying periods of time. The lesions were round or oval and red, purple or violaceous during periods of recrudescence and brown in the interim. Relapses consisted in renewed activity in the pigmented areas.

Etiology and Pathology.—Although the cause is unknown, it is suggested that a toxemia originating in the intestinal tract is probably responsible for most of these cases. In one case phenolphthalein produced a relapse.

The microscopic changes described show a superficial inflammatory process with dilatation of bloodvessels, edema and perivascular cell infiltration consisting of round, plasma and connective-tissue cells. Secondary changes in the epidermis are shown to be edema and parakeratosis. In the type, with pigmentation, Abramowitz[3] found that the pigmented areas were formed by chromatophores and concluded after a histological study that these cases belonged in the multiform erythema group.

Treatment.—Treatment has been of only temporary value. Chrysarobin and radiotherapy have both been used, the former with better results. Internal medication has been valueless.

GRANULOMA ANNULARE.

Synonyms.—Ringed eruption (Colcot Fox), Eruption chronique circinée de la main (Dubreuilh), Lichen annularis (Galloway), Granuloma annulare (Crocker), Sarcoid tumors (Rasch), Neoplasie circinée et nodulaire (Brocq), Erythémato-sclérose circinée du dos des mains (Audry), Tumors benigni sarcoidei cutis (Galewski), Stéréophlogose nodulaire and circinée (Pellier).[4]

Definition.—A comparatively rare disease characterized by papules or nodules grouped in a ringed or circinate arrangement, pursuing a chronic course and devoid of subjective symptoms.

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[1] Jour. Cut. Dis., 1917, xxxv, p. 635.

[2] Ibid., 1917, xxxv, p. 546; Wise, Ibid., p. 184; H. Fox, Ibid, 1918, xxxvi, p. 252.

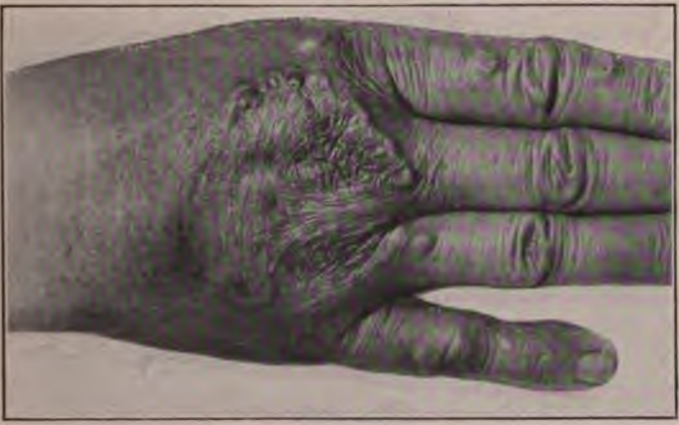
[3] Ibid., 1918, xxxvi, p. 11.

[4] Annales, January, 1910, p. 28; Abstr. Brit. Jour. Derm., 1911, xxiii, p. 32.

The disease has been studied by E. Graham Little[1] in addition to the above reporters, and much of the description found here is taken from his excellent résumé.

Symptoms.—The primary lesion is a deeply-seated nodule or a ring of closely-grouped nodules of firm consistency, elevated, sharply circumscribed, whitish, pinkish, reddish, purplish or bluish-red in color. In one instance the lesions resembled those of xanthoma tuberculosum in color.[2] The rings are oval or round and vary in size from one-half to two or more inches in diameter. The border is usually elevated about one-sixteenth of an inch but in occasional instances this elevation may be doubled. The lesions may be surrounded by a reddish areola and the center may be normal, reddened, or even atrophic or cicatricial. The ring may undergo involution irregularly and have crescentic and festooned figures. There may be but a single lesion or large numbers, forty or more having been noted. The lesions may develop suddenly or slowly and may persist for months or years. There is little tendency to spontaneous involution, although this may at times occur. The disease is seen most frequently on the dorsum of the fingers, about the wrists, elbows, neck, feet, ankles and buttocks. Gaskill[3] records lesions situated on the palm. In the five cases studied by the author (two histologically) in one the lesions occurred on the fingers, below the knee and on the ankle. In the other case in which histological study was carried out the lesions were numerous and widely separated. The age of the patients has varied from eighteen months to fifty-two years. Haldin David[4] reports the

Fig. 48



Granuloma annulare.

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[1] Brit. Jour. of Derm., 1908, xx, pp. 213–248 and 281–317. A critical study of forty-nine cases, six personal. Full bibliography to this date.

[2] Stillians, Jour. Cut. Dis., 1919, xxxvii, p. 580.

[3] Jour. Cut. Dis., 1915, xxxiii, p. 588.

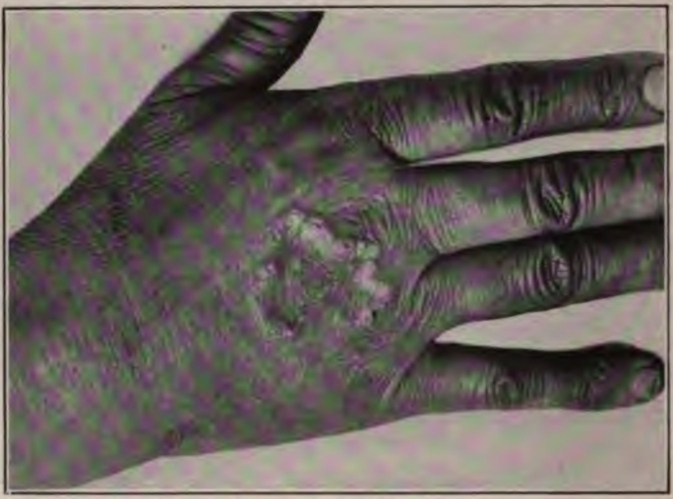
[4] Brit. Jour. of Derm., 1910, xxii, p. 90.

case of a patient sixteen months of age. The major portion of the cases occur in children and young adults.

Etiology.—The cause of the disease is obscure. Crocker found evidences of tuberculosis in some of his cases and Little found marked tuberculous antecedence in several cases and was inclined to view tuberculosis as an etiological factor.

Pathology.—In Little's collective study the histology is summarized as follows: In the deeper corium and hypoderm, groups of cells constituting a microscopic nodule were noted about coil-glands, hair-follicles, and bloodvessels. Areas of degeneration were occasionally noted in these cell-masses. In addition, there were rows of cells extending upward along the tracts of coil ducts and hair follicles, also some horizontal rows of cells, as well as a scattered cellular infiltration in the corium. The type of cell is given as large mononuclear, spindle, pear-shaped and oblong connective-tissue cells and the so-called epithelioid cell. The epidermal changes were insignificant; some thickening of the rete and stratum corneum was noted in certain cases, and in some the normal wavy line between the epidermis and corium was obliterated over a nodule. Hartzell[1] stated that a striking feature in all sections examined was a central area of necrosis in the nodules surrounded by radiating lines of oval and spindle cells. A similar histological picture was described by Stillians. In an early case studied by the author the histological changes were those usually seen in keloid; in the second case with numerous lesions the necrosis and other features described by Hartzell were present.

FIG. 49



Granuloma annulare.

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[1] Trans. Sec. on Derm., Amer. Med. Assoc., 1914, p. 27.

Treatment.—As a rule, simple local treatment is sufficient. Salicylic acid plaster, ichthyol, or resorcin have been found efficient. Jadassohn used arsenic internally. Radiotherapy and carbon dioxid snow are efficient methods of treatment.

Prognosis.—Untreated, the lesions persist and tend to spread and last indefinitely. Under appropriate treatment they readily disappear but the disease may recur.

Erythema Elevatum Diutinum.—Under this title Campbell Williams and Crocker[1] describe a rare disorder of the skin which at the present time is considered by Little, Hartzell and other observers to be a clinical variety of granuloma annulare. The disease as described by the above authors is characterized in most instances by the appearance of pea- to bean-sized, firm painless nodules, which are pink at first but gradually assume a purplish hue. In the early stages the nodules are well-defined and discrete but later tend to coalesce and form irregularly lobed infiltrations, flat raised plaques or, in exceptional cases, distinct nodular tumors. The lesions have been encountered chiefly on the extensor surfaces of the limbs and joints but also on the palms and soles, buttocks and ears. In most of the cases reported the lesions persisted for years, although some gradually underwent involution. The patients have been children or young adults, and usually females. They showed either a personal or family tendency to gout or rheumatism. Stelwagon[2] records four cases of a similar nature occurring in adults past forty years of age, the lesions being situated on the face, nose and cheeks.

Pathology.—Williams and Crocker describe a fibrocellular process of inflammatory origin situated deeply in the corium. Other observers have since described histological changes similar to those previously described in granuloma annulare.

Treatment.—In the cases described treatment proved unsatisfactory.

URTICARIA.

Synonyms.—Hives, Nettle-rash; Fr., *Urticaire;* Ger., *Nesselsucht, Nesselausschlag.*

Definition.—Urticaria is an acute or chronic disorder of the skin, characterized by the presence of wheals, which are, as a rule, hemispherical and of a white or reddened color, and which induce varying degrees of itching, tingling, and smarting sensations.

Symptoms.—The disorder may begin with constitutional symptoms, such as inappetence, malaise, headache and moderate elevation of temperature, lasting for a few hours or a day or two, or the eruption may appear suddenly without general symptoms. Two types are usually seen. The first an acute type is commonly due to some ingested material; the second type is chronic and very frequently of undiscoverable origin. The disease is characterized by lesions in the may be transitory or last for some time. Occasionally this condition exists in persons not the subject of ordinary wheals.

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[1] Brit. Jour. Derm., 1894, vi, pp. 1–9 and 33–38.

[2] Diseases of the Skin. 7th Ed., p. 169.

The subjective sensations in urticaria are usually marked but change according to the sensitiveness of the individual. Itching, burning and stinging sensations are most common, but in addition tickling, crawling, and pricking sensations are described. The lesions may appear on any part of the body as well as on the mucous membranes of the mouth, pharynx, and larynx. The rapidity of the appearance and disappearance of the lesions is a characteristic feature of the disorder. The course of the disease varies, being, as a rule, acute, the recurring lesions disappearing within a few days. In other cases the disease may be chronic, lasting for years by the constant development of new lesions. This type has been termed *urticaria recidiva*. Some peculiar types have been recorded. For example, Baker[1] reported a case of urticaria tuberosa characterized by the presence in various parts of the body of persistent, yellowish-red tubercles, that proceeded to ulceration. The parts most affected were the knuckles, the elbows, and the ears. These tubercles were said to have begun in the manner which characterizes the onset of evanescent urticarial wheals and tubercles. A somewhat similar case was observed by McCall Anderson.[2] In occasional instances the urticarial lesions may be followed by pigmentation which bears a slight resemblance to the lesions of urticaria pigmentosa. These lesions are usually chronic. Several chronic types of urticaria have been described under such titles as *urticaria perstans* (*tuberosis cutis pruriginosa*)[3] and *acne urtica*[4] varieties of urticaria in which pigmentations, nodules, and verrucous- and lichen planus-like lesions dominate the clinical picture.

Urticaria in Infants and Children.—(*Lichen urticatus*).—Under this title Colcott Fox[5] described what he considered *the* urticaria of childhood. It differed in many respects from that seen in the adult, and he and many others state that the usual urticaria wheals seen in the adult cases are not common in childhood. At a later period the same author[6] describes this disorder under the title *Lichen urticatus*, a disorder originally described by Bateman.

The distinguishing features of this variety of urticaria are the following: It is persistent, lasting for many years, having exacerbations, periods of quiescence, and relapses. It is usually worse in the summer season and is characterized chiefly by papules. These are the predominant lesions seen when the patient is examined, and are found on any part of the body, but are most numerous on the trunk. They are prurigo-like, pale or reddish in color, and irregularly disseminated. They are preceded at night according to Fox's observations, by the occurrence of a wheal or wheal-like area, which excites the greatest distress in the infant during that time. In the center of this area the papule forms. Occasionally, in places, papules, vesicles, vesicopustules or pustules occur. In other cases, on the hands, large vesicles and bullæ appear, strongly resembling scabies. In addition, at times, secondary infections are present together with all the symptoms of a marked traumatic dermatitis.

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[1] Lancet, August, 1881, i, p. 153.

[2] Brit. Med. Jour., 1883, i, p. 1103.

[3] Archiv, vol. lxxxi, p. 208.

[4] Ikonographia dermatologica, F.A.S.C., i, Tab. ii.

[5] Brit. Jour. Derm., 1890, ii, pp. 133 and 176.

[6] Allbutt and Rolleston's System of Medicine, vol. ix, p. 238.

These cases are to be differentiated chiefly from scabies and prurigo. After careful observation through a period of years, Fox concludes that lichen urticatus and prurigo are different disorders.

Etiology.—Urticaria occurs as the result of the action of various toxic substances introduced from without or produced within the body in sensitized individuals. In many instances the condition is an anaphylactic reaction induced by the introduction of a foreign protein. This occurs following the use of diphtheria antitoxin and various sera and vaccines employed in medicine. Urticaria is a common disorder, is seen at all ages, and in both sexes, children being particularly susceptible. Acute urticaria is frequently induced in predisposed individuals by the ingestion of certain foods, among which are eggs, cheese, pork, sausage, lobster, clams, several species of fish roe, including caviar, strawberries, and similar small fruits, oatmeal, pickles, nuts, canned fruits, meats, and vegetables. Any article of diet that is indigestible for a particular individual may provoke an attack. The hypersensitiveness on the part of the individual to certain foods may be inherited as well as acquired. The frequent occurrence of acute urticaria in the summer is probably due to toxic substances present in food owing to decomposition. Although most cases of urticaria produced by proteins are of animal origin, vegetable proteins have been proved to be causative by J. Chandler Walker.[1]

Many drugs act similarly, among which are quinin, the salicylates, balsam and turpentines, antipyrin, and many others. Very small quantities of the ingested material, such as one grain of quinin, a single strawberry, or a small portion of lobster, may provoke the disorder, thus illustrating the importance of sensitization or susceptibility. In addition to gastro-intestinal disturbance from food,[2] intestinal parasites[3] through their action on the food are responsible for the appearance of the disorder. Many examples of urticaria are recorded as resulting from psychic disturbances, such as fear, anger, and shame. Kreibich and Sobotha[4] describe an example. Hartzell[5] reports a case of a patient who would become covered with urticarial lesions within five minutes after an interview with a stranger. It is believed that some of these are due to sudden arrest of the functions of gastro-intestinal tract, resulting in the production of toxins. The author has seen urticaria in a patient suffering with an error of refraction. Correction of this defect relieved a chronic urticaria, but it returned when the wearing of proper glasses was discontinued. Oliver[1] recorded a similar example. Urticaria occurs with malaria, prurigo, dermatitis herpetiformis, obstructive jaundice, and other diseases of the liver, purpura, syphilis[2] and some others. Disturbances of the glands of internal secretion may be caustive.[3] The puncture or spontaneous rupture of hydatids with escape of the fluid into the abdominal cavity has been followed by urticaria. Other causes are functional or organic diseases of the generative organs in women, including the irregularities attending the menopause.

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[1] Jour. Amer. Med. Assoc., March 30, 1918; report of two cases of urticaria produced by horse dandruff protein, and three by ragweed pollen.

[2] Eustis, New Orleans Med. and Surg. Jour., 1914–15, lxvi, p. 730. Urticaria; an experimental lesion produced by the local application of Beta-imidazoylethylamin. Its relation to intestinal toxemia.

[3] *Cf.* McBride and Schorer, Jour. Cut. Dis., 1916, xxxiv, p. 70; "Erythematous and Urticarial Eruptions Resulting from Sensitization to Certain Foods." A valuable clinical and experimental study with report of results in 60 cases.

[4] Archiv, xcvii, p. 187; Discussion of the Subject of Psychic Urticaria.

[5] Diseases of the Skin, 2d Edition, p. 60.

Urticarial lesions are produced by the action of many external irritants, such as the bites or stings of mosquitoes, gnats, wasps, bees, lice, fleas, bedbugs and caterpillars. In wounds inflicted by jellyfish the lesions may develop beyond the area of injury. This occasionally happens following stings of the various insects above mentioned. Some vegetable substances also produce local lesions, chief of which is the nettle (*Urtica urens* and *U. dioica*). The lesions induced by this are so characteristic that they are responsible for one of the names attached to the disease.

Pathology.—The wheal of urticaria is a circumscribed edema of the corium, the nature of which has been exhaustively studied. Most early observers believed urticaria to be an angioneurosis, in which the toxin was supposed to act through the nervous system. The later view is that a toxin either circulating in the blood or deposited locally acts on the vessel walls.

Gilchrist[4] describes a true wheal as an acute inflammatory edematous swelling due either to local inoculation of irritating substances, as in insect bites, or to drugs, or to some toxin probably originating in the alimentary canal. In sections removed at various stages of evolution of the wheal he found fragmentation of the nuclei, which seemed to show death of cells preceding the inflammatory changes. At a later stage there was marked edema of the connective tissue and fixed cells, profuse immigration of polynuclear leukocytes, immigration of lymphocytes, pronounced fragmentation of polynuclear leukocytes and fixed cells, increase in mast cells, swelling of the cells of the sweat glands, and a deposit of fibrin, scattered throughout the corium. Török[5] also found evidence of simple inflammation. Török and Hári[6] and Philippson[7] as the result of numerous experiments concluded that urticaria is due to a direct action of an irritant upon the vessels at the point where the cutaneous lesions are produced, and that the disorder is, therefore, not an angioneurosis. In a wheal produced by the nettle, Unna (p. 25) found an acute spastic edema. The lymph channels and vessels were greatly dilated and there was a condition of stasis in the large veins which caused compression of the capillaries resulting in localized anemia of the central portion of the lesions. There was no leukocytic infiltration and no evidence of inflammation.

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[1] Philadelphia Medical Journal, January 14, 1899.

[2] Hazen, Amer. Jour. Syphilis, 1917, i, p. 750.

[3] Ravitch, Jour. Cut. Dis., 1907, xxv, p. 512; "The Thyroid as a Factor in Chronic Urticaria."

[4] Duhring's Cutaneous Medicine, i, p. 129; Transactions VI, International Dermatological Congress, New York, 1907, ii, p. 905; Jour. Cut. Dis., 1908, xxvi, p. 122.

[5] Archiv, 1900, liii, p. 243.

[6] Ibid., 1903, lxv, p. 21.

[7] Ibid., p. 387.

Diagnosis.—The diagnosis of urticaria is readily made. The chief points are the rapidity of evolution of lesions, their ephemeral character and the peculiar sensations they produce. Not infrequently the diagnosis must be made from the history and possible relics of the disorder, when at the time of examination no active lesions are present. In some cases careful examination will reveal the minute wounds inflicted by parasites which have been the cause of the disorder. From papular erythema it is differentiated readily by absence of subjective symptoms in the former and the marked differences between the large, flat papule and the urticarial wheal. The lesions of multiforme erythema are more persistent and are also devoid of subjective symptoms.

Treatment.—The first indication is the removal of the cause if it can be discovered. In chronic urticaria this includes a minute examination of the patient and his history, particularly in regard to the character of food, drink, and medicine taken, other habits of living, the occupation, clothing, and mental state. In acute urticaria a brisk emetic or a cathartic will empty the stomach and bowels of toxic material. As a rule, a light diet should be instituted for several days. In many cases the alkalies are of value and the preparations of sodium, potassium, or magnesium may be administered. Even when the presence of irritating ingesta cannot be demonstrated, laxatives such as rhubarb, magnesia, or cathartic mineral waters, and, in cases of children, castor oil, are frequently indicated. The local treatment employed in these cases may be in the form of baths, lotions, or powders. The alkaline bath, which contains sodium carbonate, sodium biborate, or potassium bicarbonate, either singly or in combination, in a strength of six ounces (180.) of the salt to 30 gallons of water is beneficial. One to two ounces (30. to 60.) of potassium sulphuret may be substituted. If it seems desirable to use an acid bath, one-half ounce (15.) of either hydrochloric or nitric acid is added to 30 gallons of water; or one dram (4.) of mercuric chlorid may be used in the same amount of water. Sometimes a demulcent bath acts well, and this is made by adding starch, gelatin or bran to the water. A starchy solution is sometimes effective and this may be prepared as follows: The starch is mixed with cold water and then boiled until the solution is of the consistency of thin mucilage. To each pint of this one dram (4.) of zinc oxid and two drams (8.) of glycerin are added before boiling is completed. When cool, and applied to the surface, this solution often gives prompt relief. Among other valuable lotions are liquor carbonis detergens, one-half ounce (15.), lotio nigra and olive oil, of each sufficient to make eight ounces (240.). The zinc oxid and aqua calcis lotion, or calamine lotion, in which ½ to 1 per cent. of phenol, or the same amount of dilute hydrocyanic acid has been added are also valuable. Of the powders, Anderson's is of much assistance:

℞—Camphoræ pulv.,	ʒiss	6
Zinci. oxidi. pulv.,	℥ss	15
Amyli pulv.,	℥j	30

In some instances a fatty substance gives more relief and this is particularly true in the chronic cases where a traumatic dermatitis is also present. In a simple ointment base salicylic acid may be used in a strength of 2 per cent., menthol, ½ to 1 per cent., or a combination of the two.

In chronic urticaria the internal management varies. In many cases an etiological factor can be determined and the treatment is regulated according to the findings. Food tests should be employed and treatment given as indicated by the findings. Very frequently nothing is learned in this way and the treatment has to be more or less empirical. The so-called intestinal antiseptics, which include salol and kaolin are of some service. Acetyl salicylic acid in a dosage of five grains four or five times daily is also helpful. Calcium lactate in a dosage of three grains after meals in connection with an acid-free diet proves of assistance in many cases. Quinin is of marked value in the malarial cases and frequently in those in which malaria cannot be demonstrated. Other preparations which are sometimes useful are sodium nitrite, pylocarpin, atropin, or the fluid extract of jaborandi. Seifert[1] found calcium chlorid in 0.1 gram dosage valuable in urticaria of obscure origin. In the chronic cases due to syphilis remarkable results are obtained by the injection of small amounts of arsphenamin, and similar results are sometimes obtained from this remedy in other chronic cases. In certain others the intravenous injection of 100 grams of Fischer's solution accomplishes the same result. Severe attacks are promptly relieved by the hypodermic administration of a solution of 1 : 1000 adrenalin chlorid. This is particularly serviceable when interference with breathing is caused by lesions in the larynx. Thyroid extract has proved of benefit in a small number of cases. When urticaria is due to some associated disorder the treatment is naturally directed toward the relief of that condition.

The local treatment of chronic urticaria is similar to that suggested above for the acute stage of the disease.

Prognosis.—The disorder is ordinarily simple and runs its course in a short time. The chronic cases not infrequently exhibit fairly marked symptoms over a period of many years, a fact to be borne in mind when outlining the future in any case. Even in these good results are frequently obtained by efficient treatment.

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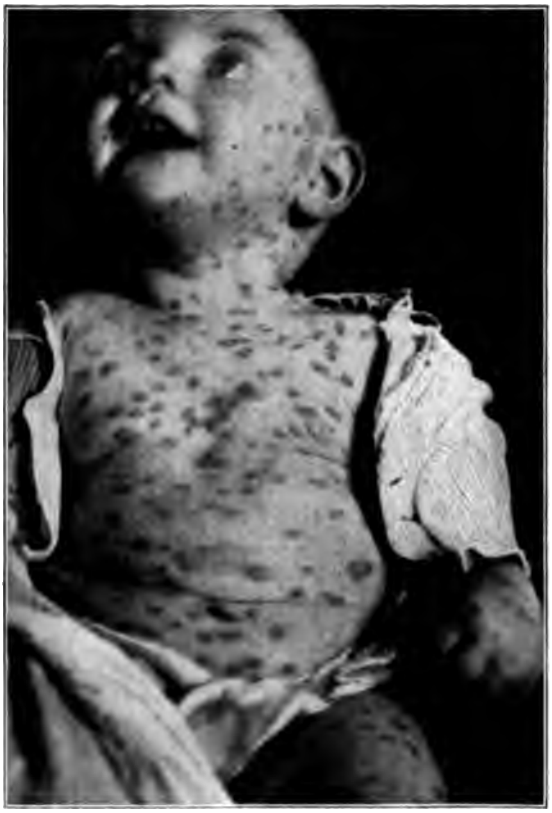
[1] Derm. Wchnschr., 1915, lxi, p. 971.

Urticaria Pigmentosa[1] (*Xanthelasmoidea*). — **Definition.** — Urticaria pigmentosa is a chronic disease of the skin usually beginning in early life characterized by pigmented macules and nodules. Urticaria pigmentosa is among the rare diseases, although a large number of cases are now recorded. It has been noted in all large centers of population, and unquestionably a great many cases have been seen and more

Fig. 52



Urticaria pigmentosa with xanthoma-like lesions.

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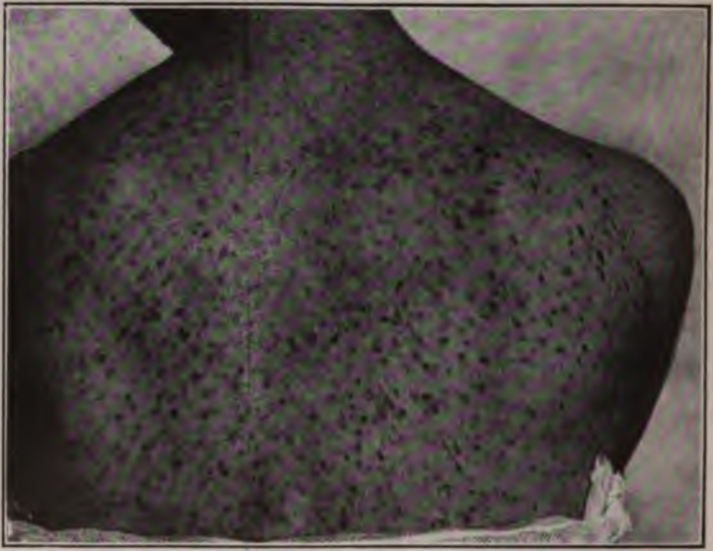
[1] For bibliography incorporating practically all the recorded cases, see Blumer (Monatshefte, 1902, xxxiv, p. 213, with a review of the clinical and pathological features of the disease); Reiss (ibid., 1903, xxxvii, p. 93); Duhring's Cutaneous Medicine, vol. ii, p. 300; Wolff (Mraček's Handbuch, i, p. 599); Perrin (La Pratique derm., iv, p. 772); E. Graham Little (Brit. Jour. Derm., 1905, xvii, pp. 355, 393 and 427; and ibid., 1906, xviii, p. 16. A thorough exposition of the subject, with a report of 14 cases, with histology and a review of the recorded English, German, Austrian and French cases, a total of 154); Knowles (Trans. of the Amer. Derm. Assoc., 1914. A report of four cases and a special study of the histology, with bibliography); Thin, Hoggan, and Fox (quoted by Unna, p. 955). Adult cases. Ware, Brit. Jour. Derm., 1915, xxvii, p. 426; Cussar, Annals, March, 1916; Little, Brit. Jour. Derm., 1916, xxviii, p. 327; Ormsby, Jour. Cut. Dis., 1917, xxxv, p. 855; Hartzell, Ibid., 1917, xxxv, p. 756. Late urticaria pigmentosa, Bibliography. Pusey, Ibid., 1917, xxxv, p. 48. Lieberthal, Ibid., 1918, xxxvi, p. 243.

or less studied without being reported. The disorder was first described by Nettleship, in 1869, and its present name was given by Sangster, in 1878. Blumer, in 1902, compiled 83 cases, and in 1905 Little collected additional ones, making a total of 154, and many more have appeared since.

Symptoms.—The disease, as a rule, begins in the first year of life, though many true examples have occurred at a later date. According to Little's statistics, 70 per cent. appeared before the end of the first year. The early lesions may be urticarial, occasionally bullous, or of the type which is characteristic of the disease, which is exhibited as a pigmented macule or nodule. The disorder is described as occurring in three types: first, those exhibiting plane or macular lesions; second, those which are distinctly nodular or tubercular; and, third, mixed varieties. Of 121 cases collected by Little in which the type was indicated in the description, 83 were of the macular, 10 of the nodular, and 28 of the mixed type. The lesions vary in color through all the shades of brown, yellowish-brown and reddish-brown. When irritated they become reddened and temporarily lose some of the brownish shade. Occasionally, in the nodular variety, a zone of redness surrounds the brownish discoloration. The lesions vary in size, and, according to Little, are from split-pea- to coin-sized and larger, and as a rule are uniform in size in a given patient, the macular variety presenting larger lesions. In shape they are oval or circular. While, as a rule, the lesions remain discrete, they may coalesce and form large

FIG. 53



Urticaria pigmentosa in an adult. The patient was a young woman, aged twenty-four years. Mast-cells were not present.

patches, more commonly in the macular variety, when by fusion irregular figures are formed, and in certain cases large areas of the cutaneous surface become involved. While there is no special distribution of the lesions, the trunk, especially over the back, may be most extensively involved. Occasionally, they spread to and involve the neck, face, scalp, palms and soles, and the buccal mucous membrane and palate. On the mucous membrane of the mouth Little describes small, brownish-yellow, slightly raised patches, which were devoid of subjective sensation. As these were not examined histologically, he was not certain that they were true lesions of the disorder. The lesions vary in number from a few to a great many. Little describes a case with but two lesions, and Colcott Fox[1] one with less than a dozen macules. Another case of widespread dissemination is described in which the greater portion of the cutaneous surface was involved. The lesions usually develop rapidly and then remain for long periods of time, the pigment in most cases being permanent. Fresh outbreaks occur at irregular intervals. One case is recorded of fifty years' duration. As a rule, no sequels other than the pigment are left, but in a moderate number of cases scars have been noted. The surface of the macules is smooth, while that of the nodules may be corrugated and roughened. An important clinical manifestation is the reddening, swelling, and enlargement of the pigmented lesion when irritated. This phenomenon does not occur in any other of the pigmented disorders. The subjective sensations may be marked, moderate, or absent. In a number of cases itching is not a prominent feature. In consequence of this, the general health is but little affected in most cases. As a rule, dermographism is present, even in cases not subject to itching. General enlargement of the glands was noted in a number of cases, even in those in which itching was absent. The glands are described as being hard and shotty, and resembling those found in syphilis. In one of the author's patients the lesions closely resembled those of xanthoma, and the diagnosis was only made after a histological examination.

Etiology.—The cause of the disorder is unknown. It occurs more frequently in fair than in dark people. The male sex shows a greater number of cases. It usually begins in the first year though many instances are recorded of its development in adult life. Pernet[2] recorded the case of an infant presenting lesions at birth. Several incidents have occurred which have been attributed as factors in the production of the disease, but these may have been coincidences. The disorder has been seen to follow vaccination, varicella, and measles. Rarely a shock to the nervous system has apparently been a factor. Hutchinson was of the opinion that insect bites were responsible. In one case, the eruption appeared after a sulphur bath. In another, the administration of morphin injections to the mother during pregnancy was believed to be the exciting cause. Neisser believed the disease to be allied to the nevi.

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[1] Allbutt and Rolleston's System of Medicine, 1911, ix, p. 236.

[2] Brit. Jour. Derm., 1916, xxviii, p. 327.

• Little states that it seems to be a justifiable inference that there is a general tendency, probably congenital, to over-production of mast-cells in the skin of patients suffering with urticaria pigmentosa. The local excessive accumulation, clinically represented by macules or nodules, may be determined, as the clinical experience would indicate, by various accidental phenomena: vaccination, varicella, urticarial lesions, and even an emotional stimulation, such as fright, acting upon a skin already fundamentally abnormal. He further states that, inasmuch as uniform blood-changes were found in his cases, the disease is probably a congenital blood-disorder of the same class as hemophilia, pernicious anemia, and lymphadenoma.

Knowles believes it is due to a toxin of unknown nature acting upon a congenitally abnormal skin, and that the disease should be dissociated from urticaria.

Pathology.—The histological changes found in this disorder are characteristic. They were described early by Hoggan, Fox, and others, but the character of the cells was not recognized until the classical description given by Unna in 1887. At that time, discovery was made that the cellular infiltration was composed of mast-cells, and it is the presence of these in large numbers that produces the characteristic histological picture. According to Unna's early description, these cells are found in the corium, distending the papillary body and flattening the epidermis above. They are closely packed, mast-cell to mast-cell, and arranged in columns by the persistence of collagenous tissues, between which, when spastic edema is added, wide lymph-spaces open. In his early work Unna believed these to be migrated wandering-cells, but later decided that they developed locally from connective-tissue cells which had taken up mast-cell granules. In the epidermis, aside from more or less stretching and flattening, no other changes were noted, except an increased amount of pigment in the basal layer of the rete. Unna's findings have since been confirmed by practically all observers, early among whom should be mentioned Baumer,[1] Gilchrist,[2] and Brongersma.[3] More recently Little,[4] Knowles, and others have made a particular study of the histopathology of the disease.

In addition to the rows and columns of mast-cells found between the collagenous bundles, cells are also found about the blood-vessels, hair-follicles, and sweat-glands, and granules from these cells may be occasionally detected lying free in the lymphatic spaces of the corium and subcutaneous tissue. The cells are cubical and fusiform in shape, but aside from their special arrangement present no peculiarities. The collagen-bundles are separated and spaced out, especially in the superficial part of the corium, and the elastin shows a similar arrangement. Little states that the pigment is found in special cells in the basal layer of the epidermis, and that these extend upward and invade several superimposed layers of the rete. The same cells may be seen free in

[1] Archiv, 1896, xxxiv, p. 323

[2] Johns Hopkins Hosp. Bull., 1896, vii, p. 140.

[3] Brit. Jour. Derm., 1899, xi, p. 179.

[4] Loc. cit.

the corium, usually near the basal layer of the epidermis. The amount of pigment is variable, and, according to Little, the color of the lesion depends upon the number of mast-cells as well as the amount of pigment. Gilchrist and Little found mast-cells in apparently normal skin in these cases. Mast cells have been absent in some of the adult cases reported. The bloodvessels of the corium appear to be increased in number and are dilated, and it is about these structures that the major portion of the cellular infiltration occurs. Edema is described by a number of observers throughout the whole cutis, with dilatation of the lymph spaces about the vessels and glandular structures. Little also found an intracellular and occasionally an intercellular edema in the epidermis. Unna believed the brown color of the lesion was due to the ordinary pigment of the epidermis, which was increased in the basal layer of the rete; but according to Little's observations the pigment occurs in special cells situated not only in the basal layer but above it, and also a few in the corium, and he repeatedly demonstrated the granules of pigment to be melanin.

Diagnosis.—Urticaria pigmentosa is to be distinguished from the slight pigmentation left after well-marked urticaria of later years by the beginning of the disease in infancy and by the persistence of the nodules. Xanthoma in all its forms is readily distinguished by its occurrence in special regions (the eyelids, for example); by its first appearance, in most patients, at a later period of life than infancy; and by its characteristic chamois-leather-yellow shade.

Treatment.—Treatment is entirely unsatisfactory. When an urticarial tendency is present, this may be treated in accordance with the rules laid down for that disorder.

Prognosis.—The active reproduction of lesions commonly subsides spontaneously after a certain number of years. The hyperpigmentation may remain indefinitely. Certain cases have been reported as having been relieved in a short time, but in the major number the disease persists after long periods of time.

Angioneurotic Edema.[1]—**Synonyms**: Giant urticaria (Milton), Urticaria edematosa (Hardy), Acute essential edema (Etienne and Galliard), Wandering edema, Quincke's edema, Acute circumscribed edema.

This unusual disorder was early described by Milton,[2] Bannister,[3] Quincke,[4] and many others. The disease is characterized by circumscribed edematous swellings of the skin and subcutaneous tissues. It appears suddenly, lasts from a few hours to a day or two, and is accompanied by sensations of tension and by varying degrees of itching. It occurs, as a rule, about the face, genitals, and extremities, and has a marked tendency to recurrence, often showing an hereditary history and continuing its activities through years to a lifetime.

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[1] For literature, *Cf.* Index Catalogue of the Surgeon-General's Library, 2d Series, vol. xii; and Cassirer's monograph: Die vasomotor-trophischen neurasem, Berlin, 1901; also Osler's Modern Medicine, 1909, vi, p. 648; Bullock, The Treasury of Human Inheritance, Part iii, 1909.

[2] Edinborough Med. Jour., 1878.

[3] Chicago Med. Review, June 20, 1880.

[4] Monatshefte, 1882, p. 129.

Symptoms.—The lesions appear suddenly, often in the early hours of the morning, and may be single or multiple, and of great variation in size, consistence, color, and shape, depending upon their situation. In size they vary from one to two inches in diameter on the hands and eyelids; while on the trunk and lower limbs large-nut- to orange-sized globular tumors are seen. Osler describes swellings on the hands as large as light-weight boxing-gloves, and saucer- to plate-sized swellings on the trunk and thighs. On the eyelid and lip the author has seen, in common with many others, swellings of such size as entirely to distort the features of the patient. In color the lesions may be of the normal hue of the surrounding skin, opaque-white, translucent, or waxy-looking, or of a slightly yellowish tinge; or they may be reddened in varying degrees. The local temperature may be normal or subnormal. In consistency the lesions are firm, elastic, and at times hard. On the eyelids the lesions are softer, and pitting may be detected; in the declining stages in other situations the lesions become softer, and after their disappearance the skin may be flabby and wrinkled. The overlying skin is smooth and shining, and in certain cases bullæ may form (Osler). As a rule, the lesions occur asymmetrically, though a symmetrical arrangement may be noted on the hands, and occasionally the whole face may be involved. The areas of predilection are the face, genital region, and extremities, though the trunk and other situations may be attacked: the mucous membranes of the mouth, pharynx, larynx, the accessory sinuses and other portions of the upper air passages, the gastro-intestinal mucosa, and the conjunctiva. Edema of the glottis produces alarming symptoms and numerous fatalities are recorded from this involvement. A remarkable example of this is portrayed by Osler (p. 655).

The progress of the disease is marked by recurrence of lesions, frequently in the same situations, through a period of years. In a patient under observation for some years the lesions constantly recurred on the tongue, upper lip, eyelid, and dorsum of the hand. The swelling remained for several hours to one day and was intense.

Subjective symptoms are usually noted only in the feeling of tension, with moderate itching or sensation of heat. As a rule, there are no constitutional symptoms, though concomitant attacks of colic have not infrequently been noted. In ten of Osler's cases colic was present, and he cites examples in which abdominal operations have been performed for erroneously suspected appendicitis, gall-stone, or renal colic.[1] In addition, there may be various grades of indisposition (headache, dizziness, and depression) as accompaniments. In other cases there have been noted hemoglobinuria, albuminuria, and various cutaneous eruptions belonging to the urticarias, erythemas, and purpuras.

Etiology.—The affection occurs most frequently in early adult life, and is rare after the sixtieth year. Fox[2] records an example occurring at the age of three months. All writers agree that the disease occurs most frequently in people of the private-patient class. Relative to sex, the disease occurred in 14 females and 4 males in Osler's list of 18 private cases; while, on the other hand, Collins (quoted by Fox) found in a collection of American cases one-third more females, and Cassirer,[1] in his review of the literature, found the condition reversed, namely, 70 males to 63 females.

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[1] Amer. Jour. of the Med. Sc., 1904, cxxvii, p. 751.

[2] Allbutt and Rolleston's System, 1911, ix, p. 228.

Among predisposing causes various factors are recorded, such as nervous influences, which Osler believes most important, and which are exhibited as migraine, neuralgia, exophthalmic goiter, and melancholic tendencies. Other factors are malaria, alcoholism, menstrual disturbances, emotional disturbances (fright, anxiety, worry), overwork, insomnia, and mental exhaustion. Morichau-Beauchant[2] divides the acute circumscribed edemas into three groups: the arthritic, the peliotic, and the true angioneurotic edema, and believes they have a common cause in gastro-intestinal toxic infection. Schlesinger[3] observed the disease associated with erythromelalgia, both disorders being induced by over-indulgence in alcohol. Heredity is an important factor. Osler[4] recorded its occurrence in 22 people in five generations, in which it caused two deaths. Ensor[5] recorded 49 cases in seven generations, 12 of whom died of edema of the glottis. Other much less remarkable herditary examples have been noted.

Pathology.—The pathogenesis of the disorder is unknown. Its close relationship to urticaria is admitted. The local lesion, according to Osler, is an extreme urticarial one, with exudation of serum, leukocytes, and occasionally red-blood corpuscles. Its relation to the purpuras and erythemas is suggestive.[6]

Diagnosis.—In well-defined cases, with recurring large, tense lesions occupying the classical regions, the diagnosis is clear. Localized edemas from well-known causes must be differentiated, such as those occurring in local thrombosis, stasis, infections about the nose inducing lymphatic obstruction, and the early edematous stage of scleroderma. The lesions of angioneurotic edema are more acute and transient, and by a consideration of the accompanying features of other disorders are differentiated. The difficulty has occurred particularly in those cases involving the gastro-intestinal mucosa, accompanied by colic. The symptoms here are erroneously attributed to other disorders.

Treatment.—The treatment, in the main, resolves itself into the general care of the patient. Careful general systematic examination must be made, and all departures from the normal corrected if possible. Winfield[7] recorded a case of angioneurotic edema entirely relieved following successful treatment of cholelithiasis; and Oberndorf[8] reported disappearance of the symptoms of angioneurotic edema following an appendectomy. In some cases changes in the dietary have been beneficial, in others useless. Stimulating, highly seasoned, and indigestible foods, and spirits are to be interdicted.

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[1] Loc. cit.

[2] Annales, 1906, vii, p. 22; abstr. Jour. Cut. Dis., 1907, xxv, p. 130.

[3] Med. klin., Berlin, 1906, ii, p. 94; abstr. Jour. Cut. Dis., 1907, xxv, p. 129.

[4] Amer. Jour. Med. Sc., 1888, xcv, p. 362.

[5] Guy's Hosp. Reports, 1904, lviii, p. 111.

[6] Osler, Brit. Jour. Derm., 1900, xii, p. 227.

[7] Jour. Cut. Dis., 1907, xxv, p. 217.

[8] Jour. Amer. Med. Assoc., 1912, lix, p. 623.

Strychnin, quinin, and arsenic, as well as the bromids, ergot, belladonna, the salicylates, and aspirin, are of value. In Osler's experience the nitrites and nitro-glycerin, given in large doses, and calcium salts were of the most value.

Prognosis.—The milder cases run a benign course and recover in a reasonable time. Others are persistent and troublesome for years; while in severe mucous-membrane involvement edema of the glottis is always a menace. In 170 cases tabulated by Bullock, death occurred from this cause in 36.

PRURIGO.[1]

Synonyms.—Prurigo of Hebra, Prurigo gravis, Prurigo ferox, Prurigo agria, Prurigo mitis.

Definition.—Prurigo is a chronic, exudative, cutaneous affection, commonly beginning in infancy or early childhood, continuing through life, and characterized, at first, by urticarial symptoms, and later by the occurrence on the extensor surfaces of the extremities, and also on the trunk, of minute, pale or reddish papules, accompanied by an intolerable itching. The disease formerly was chiefly seen in Austria, but of late years has been described in many parts of the world, including America. In this country, as a rule, only mild cases are seen among the native population.

Prurigo is one of those terms which in the past have led to considerable confusion in the nomenclature of cutaneous disease, the term having been applied to many different affections. It is here limited to the disorder originally described by Hebra under that name, the characteristics of which are outlined below.

Symptoms.—The earliest symptoms are usually displayed, in the latter portion of the first year of life, in the form of an urticarial rash, which persists, and which is finally succeeded by typical papules of the disease. Fox[2] maintains that the initial eruption may be papular and not urticarial. These papules are millet-seed- to hemp-seed-sized, in color not differing markedly from that of the normal skin. They are intensely pruritic, and rapidly become covered with blood-stained crusts in consequence of the induced scratching. As a result of this trauma, there ensues a long train of complications, including pustulation, fissures, excoriations, dense infiltrations, crust-formation from exuded serum and dried blood, edema, lichenification, and diffuse, dark-brown pigmentation of the skin-surface in large areas. The glands which receive the lymphatic flow from the excoriated areas are enlarged. This adenopathy is conspicuously shown in the inguinal and cubital glands. Fully developed, the disease presents in general the same physiognomy in patients of different ages. The extremities always exhibit the severest manifestations of the disease, and of these the leg and forearm are usually affected more severely than the thigh and arm; though the trunk, the forehead, the neck, the face, and the scalp may also be involved. Hebra found the disease exhibited with increasing severity from the scalp downward. The extensor surfaces are invariably selected by the disease, while the flexor surfaces, such as the axillæ and the groins, except as regards adenopathy, are free from change. The general health of the patient manifestly suffers from insomnia and nervous agitation induced by the state of the integument. Emaciation, malnutrition, and cachexia are common sequels. The mental and moral tone of the patient thus harassed from early childhood throughout an entire life is necessarily profoundly impaired. Insanity and suicide are reckoned among its remote consequences.

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[1] Sir Malcolm Morris: Prurigo, Pruriginous Eczema, and Lichenification. Brit. Med. Jour., June 29, 1912, p. 1469; with discussion (Brit. Jour. Derm., 1912, xxiv, p. 251) by Colcott Fox, Galloway, Leslie Roberts, Whitfield, E. Graham Little, and MacLeod.

[2] Allbutt and Rolleston's System, ix, p. 271.

Mild and severe forms of the disease are distinguished under the terms *prurigo mitis* and *prurigo ferox* or *agria;* they agree with respect to the evolution of symptoms; the only difference to be observed is in their intensity. In the former the papules are fewer, the recrudescence rarer, the itching less intense, and the amenability to treatment more pronounced. Although incessant and judicious treatment, favorable climatic influences, and comfortable conditions of life are factors which mitigate the symptoms, nevertheless the difference between the two forms is probably largely determined by the intensity of the causal elements which first establish the disease in the individual. A prurigo which begins with severe symptoms may persist in the ferox form throughout life; while a prurigo mitis is such from the first appearance of the disorder. A prurigo-like eruption not infrequently occurs in association with lymphadenoma.

Etiology.—The disease occurs chiefly in Austria, a few cases being recorded elsewhere. Wigglesworth,[1] Campbell,[2] Zeisler,[3] and others have reported cases in America. The actual cause of prurigo is not positively known. It is encountered more often in the male sex, is never contagious, and is never induced by lice; but, according to Hebra and Kaposi, it may be grafted upon an hereditary predisposition. Several cases have been known to appear in one family, suggesting strongly an hereditary element. "Scrofula," tuberculosis, malnutrition, "misery," poverty, anemia, and filth are held to be severally favorable to its development. Boas,[4] after a series of tuberculin injections in twelve patients, concludes that tuberculosis is not a factor in the prurigo of Hebra. The disease is practically limited to the poorer classes living under wretched hygienic and social conditions. Some authorities, especially among the French, hold that the disease has a neurotic base; that the itching is the essential element, the papules developing from the irritation of scratching. Others believe that a toxic cause operates, because of the urticaria at the beginning and the frequency of stomach and bowel disturbances in those who are afflicted. It is highly probable that both theories have a more or less true relation to the etiology of the disease.[1]

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[1] Amer. Jour. Syph. and Derm., 1873, iv, p. 21.

[2] Archiv. Derm., 1878, iv, p. 119.

[3] Jour. Cut. Dis., 1889, vii, p. 408.

[4] Nord. med. Archiv, Abt. ii, p. 24; abstr. Brit. Jour. Derm., 1912, xxiv, p. 374.

While typical prurigo ferox, as described by the Vienna school of authors, is rare in America, the opinion is gaining ground that the same disease with milder manifestations (prurigo mitis) is much more common here than has been believed. Imported cases from Vienna, presenting severe grades of the disorder, have been seen by Dr. Hyde in his clinic. Cases of the milder type are a matter of not uncommon record.

Pathology.—Kaposi practically admits that, striking as is the clinical portrait of this disease, its anatomical features are indistinguishable from severe forms of obstinate papular eczema, or from other forms of chronic dermatitis accompanied by hyperplasia. The microscope reveals proliferation and swelling of rete-cells, cell-infiltration, and edema of the papillæ, most marked around the vessels, and frequently dilated lymph spaces. There is a scattered deposit of pigment in the corium, and many cutaneous muscles (erectores pilorum) are thickened and shortened. Holder[2] states that these muscles are not only hypertrophied, but also are contracted, and that the papule has an urticarial basis.

Some authors contend that the papules are solely due to traumatism of the pruritic skin. Auspitz believes that the disease is in fact a sensori-motor neurosis without an essential lesion. Riehl[3] considers it as a chronic form of urticaria. Leloir and others find the prurigo-papule invariably resulting from a cystic degeneration of rete-cells, thus forming a cavity, which at first contains clear serum, with the addition later of epithelial débris. The walls of the cyst later undergo keratinization.

Bernhardt,[4] after studying a typical case in a patient with a paralyzed arm, believes the disease is a dystrophy of the corium due to chronic irritation of the trophic centers, and that the papule precedes the itching.

White,[5] in a review of the subject which sets forth the great diversity of opinion as to the nature and cause of prurigo, concludes: "I cannot go farther than accept the existence of a condition of early childhood, allied to pruritus and urticaria in its visible manifestations, and not to be distinguished positively from them in its first stages, often becoming in certain parts of the world a chronic affection due to some inexplicable national cutaneous traits, or inherent customs of living; a condition which certainly lacks many of the essential elements of individuality."

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[1] Matzenauer, Mraček's Handbuch, Bd. ii, pp. 701–714 (with bibliography).

[2] Jour. Cut. Dis., 1901, xix, p. 489.

[3] Vierteljahr, 1884, xl, p. 41.

[4] Archiv, 1901, lvii, p. 175 (bibliography).

[5] Jour. Cut. Dis., 1897, xv, p. 2.

Diagnosis.—Remembering the extreme rarity of prurigo in America, it is to be distinguished chiefly from the various forms of papular eczema by the location of its lesions, by the course of the disease, by the age of the patient when it is first developed, by the great extent of the eruption, and by the uniform type of its lesions. In prurigo, also, the fingers and the toes, the flexor aspects of the extremities, and the face are more or less spared. Under treatment eczema commonly yields, at least in some portions of the skin, while prurigo does not.

From pruritus, prurigo is readily differentiated by its general physiognomy and history, by its peculiar pigmentations and infiltrations, and by the special regions chiefly affected. Eczema may occur coincidently with prurigo, as a result of the scratching induced by the intense itching present in the latter disorder. In pediculosis corporis the parasites usually will be found upon the underclothing, and the lesions induced by the finger-nails never form closely-packed papules. There is something highly characteristic in the widely separated excoriations, the puncta from wounds inflicted by parasites, and the inflamed papules seen upon louse-bitten patients.

In scabies the characteristic burrows of the parasite will usually be recognized, as also vesicular and pustular lesions. Urticaria can be mistaken for prurigo only in the earlier stages of the last-named disease. From lichen urticatus a period of observation may be necessary to make the differentiation.

Treatment.—In Vienna, sulphur, naphtol, tar, green soap, baths, and frequent anointings with oily and fatty substances have occasionally served to ameliorate the severe symptoms of the disease. Mercury, ichthyol, salicylic acid, phenol, boric acid, and diachylon and zinc ointments may also be employed upon different portions of the skin when indicated.

The Wilkinson salve, representing a combination of tar, sulphur and green soap, has proved of special value in many cases. Vleminckx's solution (*q. v.*), followed by hot bathing, and corrosive-sublimate baths, 1 drachm (4.) of the sublimate to 30 gallons of water, has also been recommended. Fox[1] reports a case relieved with sulphur and ichthyol ointments. Internally arsenic has proved valueless, while phenol occasionally has seemed beneficial. Cod-liver oil and the ferruginous tonics with the bitters are indicated in many patients suffering from malnutrition. A generous diet and a tonic regimen are often essential to the management of the disease. It is to be noted of all cases that they are influenced happily by the warm weather of the summer season and by special attention to cleanliness and hygiene.

Prognosis.—The disease usually persists through life. The most favorable conditions are those in which the patient is young and surrounded by circumstances which permit of provision for his needs.

Prurigo Nodularis.—**Synonyms**: Multiple tumors of the skin accompanied by intense pruritus; Urticaria perstans verrucosa.

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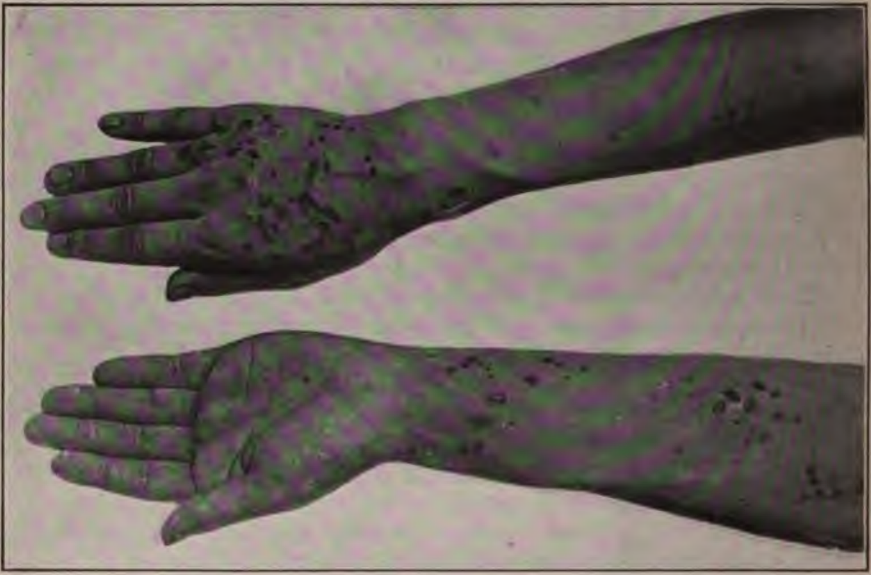
[1] Jour. Cut. Dis., 1903, xxi, pp. 148-229.

Definition.—In the year 1880, Hardaway,[1] of St. Louis, described this rare disease, which occurred in a female patient under his care, the histological study being made by Heitzmann. In 1906, Schamberg and Hirschler[2] described two cases of a similar character occurring in negroes. In 1908, a similar case was seen by Dr. Hyde, and the above title applied to this group. Zeisler,[3] in 1912, made a clinical and histological report of a similar case, and brought together several other cases described in the literature under other titles, the chief of these being *Urticaria perstans verrucosa.* He included also White's case of *Lichen obtusus corneus.*[4] In the case presented before the New York Dermatological Society in 1909 by Dr. Jackson[5] as "Multiple tumors associated with itching," Dr. Johnston stated that the general appearance, behavior, and histological character justified its assignment to the prurigo group.

FIG. 54



Prurigo nodularis.

Symptoms.—The disease appears to occur chiefly in adults, and usually in women. The lesions are nodular and verrucous in type, firm, pea- to finger-nail-sized, occurring in great numbers on the back, but chiefly over the extremities, hands, arms, feet, legs, and thighs. The smaller are at first covered with a smooth envelope, whitish, pinkish or brownish (blackish on the negro skin) in color. As they grow older they become rough, acquire a horny consistency, and often develop at the summit a verrucous condition. After scratching, which is practised in all cases, the surface of the nodule becomes furrowed, fissured, and at times hemorrhagic. In some instances the nodules become fused in a plaque of infiltration; in others they are isolated throughout. In one case they began as "blisters;" in all the others as dry papules. Itching in all cases is severe and appears to be limited to the lesions, and seems to be an essential feature of the disease.

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[1] New York Jour. Derm., April, 1880; Trans. Amer. Derm. Assoc., 1879, p. 78.

[2] Jour. Cut. Dis., 1906, xxiv, p. 151.

[3] Ibid., 1912, xxx, p. 654.

[4] Ibid., 1907, xxv, p. 385.

[5] Ibid., 1909, xxvii, p. 39.

The course of the disease is exceedingly slow, lasting from fifteen to twenty or more years. In some cases not a single lesion has disappeared after its development, and in others recurrence has happened after extirpation. That the nodules are not due to trauma from scratching was demonstrated in several cases.

Etiology and Pathology.—The nature of this rare disorder is obscure. The description of the histological changes in all the cases corresponds closely; the chief changes noted being hypertrophy of the epidermal layers, particularly the stratum corneum; vascular dilatation, perivascular cell-infiltration of the corium, with proliferation of the fixed connective-tissue elements. Mast-cells were described in excess in the case of Schamberg and Hirschler. Johnston[1] stated that in his case Dr. Welch, of Johns Hopkins, discovered that the infiltration lay particularly about the nerve-trunks, which fact would account for the intense itching accompanying the lesions. Johnston further found at times a superficial intraepidermic vesicle, similar to that found in prurigo.

Treatment.—No treatment as yet practised has been effective. In Zeisler's case temporary benefit was obtained by the combined use of *x*-rays and a 10 per cent. chrysarobin varnish.

Prognosis.—The prognosis is unfavorable as regards the comfort of the patient and the complete removal of the lesions.

DERMATITIS.

Synonyms.—Ger., *Hautentzündung;* Fr., *Dermatite, Dermite.*

A large number of cutaneous diseases are characterized by inflammation; hence, the term dermatitis alone has no special significance. The group of diseases characterized by inflammation and to which this term is attached are distinguished by a qualifying adjective. Under this title will be considered those diseases produced by thermal, chemical and mechanical agencies, which terminate upon the withdrawal of the exciting cause. In the trades and professions dermatitis occurs as the result of irritation of the skin through the above-named agencies in susceptible individuals. A separate description of these would require a volume, as about forty different occupations have been proved to cause such inflammations. In the major portion of these the dermatitis continues long after the withdrawal of the cause and recurs without the operation of the original cause. Therefore, the occupational dermatoses assume characteristics which remove them from the category of dermatitis as defined above. Clinically, histologically and historically

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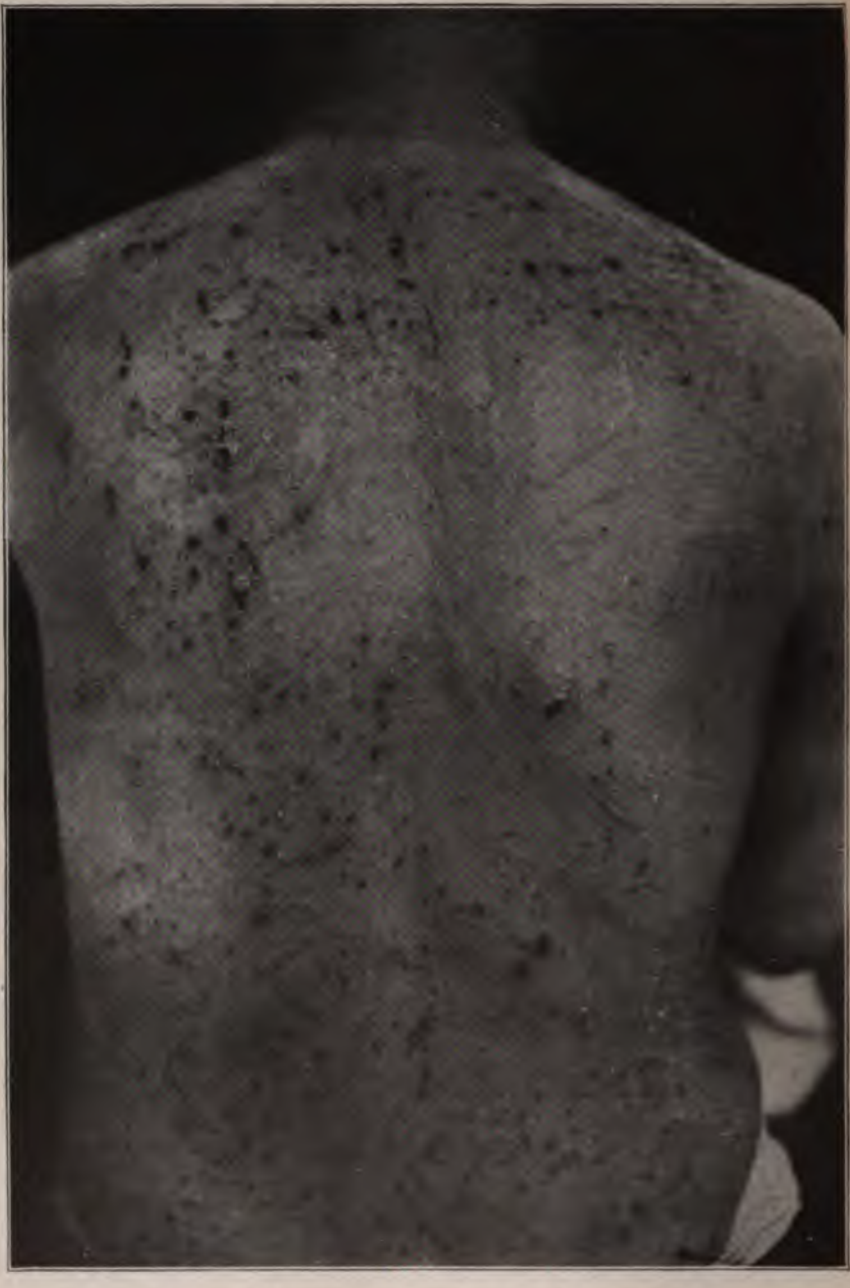
[1] Jour. Cut. Dis., 1912, xxx, p. 659.

FIG. 55

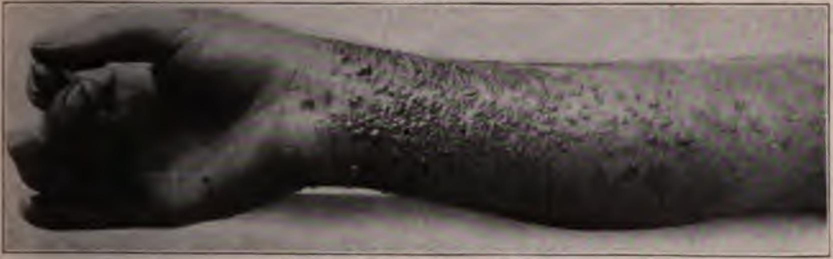


Traumatic dermatitis consecutive to pruritis cutaneous.

they are indistinguishable from the form of dermatitis known as eczema and they will be considered under that head.

Dermatitis Traumatica.—External violence, varying in character and severity, is capable of inducing dermatitis, the symptoms of which differ in degree, though their career is, in general, the same. In this list are included the inflammations produced by surgical interference with the continuity of the integument; excoriations caused by scratching, by friction with garments and other articles injuriously acting upon the skin; by the various implements handled in the trades; and by the bites or stings of beasts, insects, reptiles, and fishes, when the result is traumatic and not toxic in character. These injuries may be in the form of contusion, blow, concussion, pressure, puncture, incision, or laceration; and the consequences be declared in heat, swelling, redness and pain; in itching, burning, stinging, or pricking sensations; with subsequent inflammatory symptoms, varying in grade from mild and transitory hyperemia and exudation to the severer grades of inflammation mentioned in the preceding paragraph.

FIG. 56

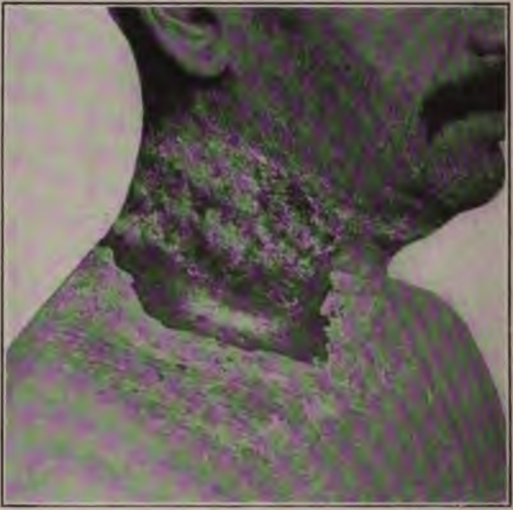


Dermatitis venenata. (Fox.)

Dermatitis Venenata.—**Definition.**—Dermatitis venenata is an acute inflammation of the skin caused by the external application of various substances of animal, vegetable or chemical nature, characterized by redness and swelling, frequently by vesicles and bullæ, and accompanied by sensations of itching and burning in varying degrees.

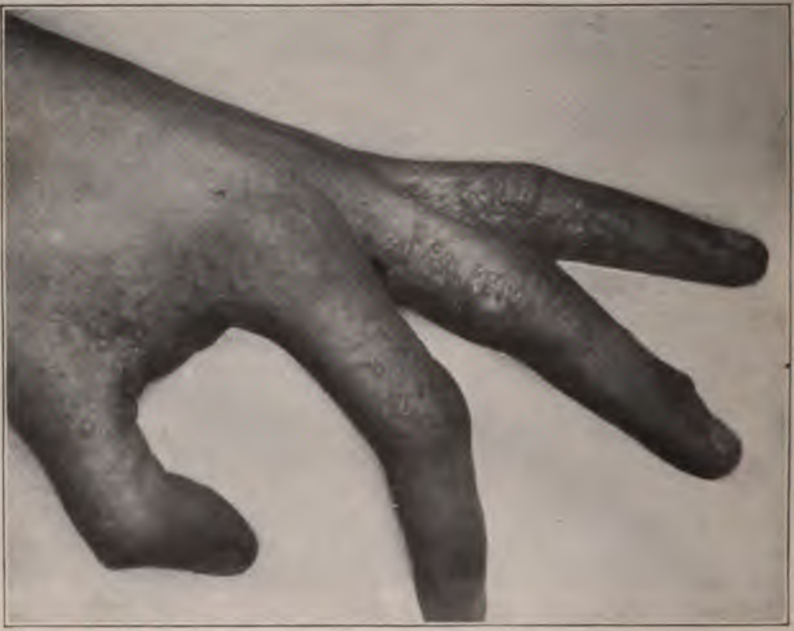
Symptoms.—The objective symptoms of dermatitis venenata range from erythema to gangrene, depending upon the exciting cause. The earliest change noted is erythema, usually limited in outline to the area of contact with the irritant. There is edema with marked swelling and upon this reddened area variously sized papules and vesicles develop. In some instances bullæ or pustules are present. After several days involution occurs with the formation of crusts or scales and the process is over in one to three weeks. At times the dermatitis spreads far beyond the demonstrable area of contact of the irritant and new areas continue to develop over a period of several weeks, involving a large part of the cutaneous surface. When the process is of severe grade subcutaneous abscesses and gangrene may occur as complications. In the variety due to ivy, which is common in America, the inflammation begins, as a rule, within a few hours after contact with the plant and is accompanied by erythema, swelling, vesicles, bullæ and a serous discharge when the lesions rupture. Much swelling occurs

FIG. 57



Severe bullous dermatitis due to caustics.

FIG. 58

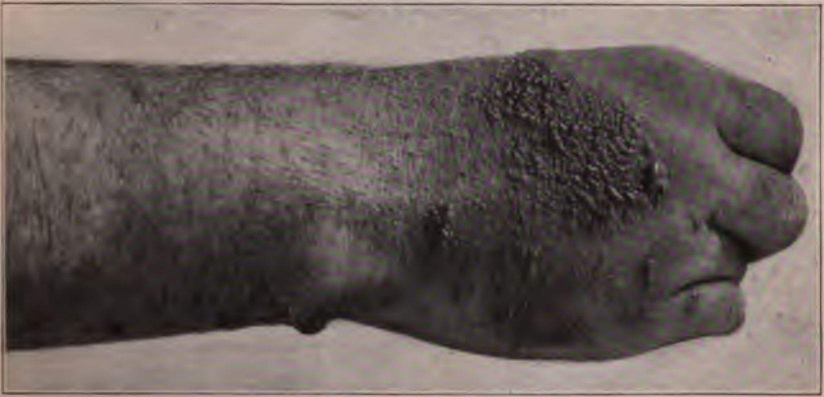


Dermatitis venenata produced by chemicals.

in areas where the tissue is lax, such as about the eyes and in the genital region. The vesicles may be small and closely set, or larger and more discrete. In recurrent cases the vesicles may occur in well-defined areas on the arms, for example, without much erythema and swelling. Abscesses may complicate the process, but scar formation rarely results. The areas commonly involved are the arms, forearms, face, neck and genital region, though at times the feet and legs or any portion of the cutaneous surface may be affected.

The dermatitis due to primrose[1] may in its acute forms resemble that described above. It may also be less acute and is then characterized, particularly in recurrent attacks, by lesions of erythemato-vesicular or erythemato-squamous type, accompanied by infiltration and scaling. This type closely resembles recurrent eczema. Foerster[2] regards primula dermatitis as not uncommon, having seen over forty cases in seven years. Sharpe[3] recorded examples produced by the *Primula farinosa*, a variety of the plant which grows wild.

FIG. 59



Dermatitis venenata. (Fox.)

Course.—Most cases develop rapidly and run an acute course, subsiding without sequels in one to three weeks. Others, by the development of new areas which run a similar course, prolong the attack for several weeks. A few cases develop into chronic eczema either through prolonged application of irritant or by the development of sensitization to other forms of irritation. Occasionally dermatitis medicamentosa is added through the absorption of the drug and its internal toxic action. Constitutional symptoms are usually absent, though in some instances where a severe dermatitis, due usually to the application of a drug or chemical substance, is present moderate or severe constitutional involvement occurs.

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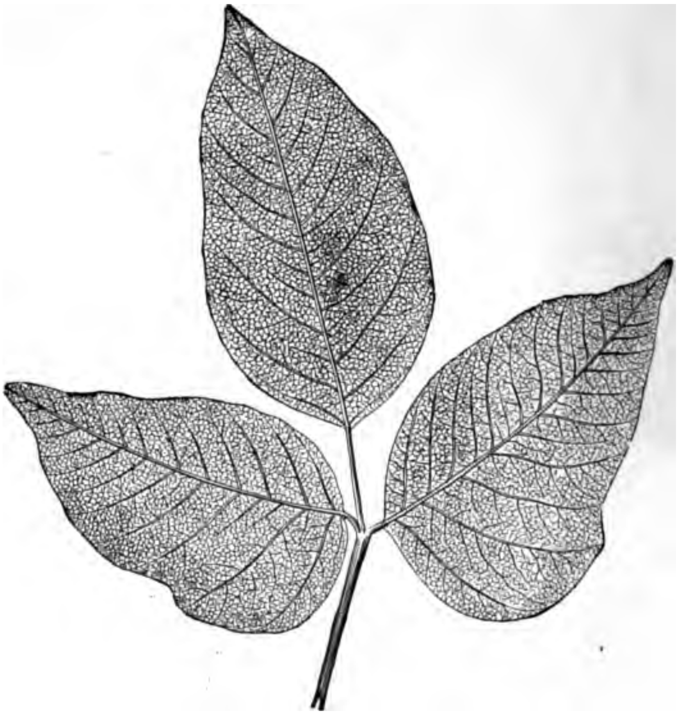
[1] Foerster, Jour. Amer. Med. Assoc., 1910, lv, p. 642; a thorough discussion of the clinical features of the disease with a description of the botanical and chemical characteristics of the plants.

[2] Loc. cit.

[3] Ibid., December 14, 1912, p. 2148.

Etiology and Special Symptoms.—The several members of the Rhus family are capable of inducing dermatitis venenata, *Rhus toxicodendron* (poison ivy) and *Rhus venenata* (poison sumach). In the primrose family are *Primula obconica, Primula sinesis, Primula cortusoides* and *Primula sieboldii,* all of which are domestic varieties. *Primula farinosa,* a variety of the plant which grows wild is responsible for cases, examples of which were recorded by Sharpe.[1] Other plants are the nettle and smartweed (*Polygonum punctatum*), cowhage (*Mucuna pruriens*) and a large number of others which are enumerated in a monograph by J. C. White.[2] Recently a group of cases due to handling tomato plants (*Lycopersicum esculentum*) was reported by Lain.[3]

FIG. 60



Rhus radicans: leaf one-half natural size. (Culbreth.)

A number of cases of dermatitis have originated in some parts of the Orient from contact with the varnish employed in the finishing of lacquered ware.[4] This lacquer is manufactured from a Rhus varnish.

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[1] Jour. Amer. Med. Assoc., December 14, 1912, p. 2148.

[2] Dermatitis Venenata, Boston, 1887, and Jour. Cut. Dis., 1903, xxi, p. 441.

[3] Trans. of Section on Drem., Amer. Med. Assoc., 1918, p. 145. Dermatitis lycopersicum esculentum (tomato plant.)

[4] Toyama, Jour. Cut. Dis., 1918, xxxvi, p. 157.

A few instances of such dermatitis have occurred in America from handling newly imported articles of this class.

A common cause of acute dermatitis about the forehead, eyes, face and neck is found in proprietary hair dyes and in furs. The chemical irritant is paraphenylendiamin, a component part of the dye.[1] Other chemicals used in the manufacture of dyes, dyestuffs or allied substances not infrequently produce dermatitis. Dinitrochlorbenzol produced a group of cases reported by Bernstein.[2] Hexanitrodiphenylamin, a component of the explosive material found in bombs, caused many cases of dermatitis during the war, as reported by Sequeira,[3] MacLeod,[4] and others.[5] These cases presented vesicles and bullæ and were accompanied by yellow staining of the skin. The vesicles were dysidrotic in type and the dermatitis usually appeared about nine days after exposure to the irritant.

An artificial dermatitis induced by Swedish matches was described in 1918 by Rasch[6] and others in Denmark. Similar cases were later reported in this country by G. H. Fox[7] and C. J. White.[8] In the foreign cases the dermatitis began on the thigh under the pocket where a box of matches was carried. In this area a well defined patch of red scaling dermatitis occurred which was followed by inflammation of varying grades upon the face, eyelids, hands and other regions of the cutaneous surface. In some instances the disorder became so serious that hospital care was necessary. These cases are characterized by recurrence and resemble eczema to a marked degree. Three well-marked cases were under the observation of the author for some time before the etiological factor was discovered. In the groups of recorded cases in Denmark recurrence ceased when the particular matches found to be the cause were discarded.

A number of common drugs produce dermatitis in susceptible individuals; among others arnica, an article much used as a domestic application for numerous simple wounds, is capable of producing severe dermatitis accompanied by constitutional symptoms. Cartier[9] reported excessive erysipelatous swelling and phlyctenular eruption and submaxillary adenopathy resulting from the use of arnica. Gangrene is also reported to have followed its application. Iodin, a very commonly used local application, induces severe dermatitis in certain instances. At times an acute vesicular dermatitis, through involvement of new areas, may become extensive. Phenol produces a vesicu-

[1] Bunch, Brit. Jour. Derm., 1915, xxvii, p. 348: "Paraphenylene diamene dermatitis." Olson, Jour. Amer. Med. Assoc., March 18, 1916, lxvi, p. 864; "Dyed fur, paraphenylendiamin dermatitis; report of 5 cases."

[2] Lancet, April 27, 1912, clxxxii, p. 4626; abstr., Jour. Cut. Dis., 1912, xxx, p. 571.

[3] Brit. Med. Jour., 1917, i, p. 894.

[4] Ibid., ii, p. 80.

[5] Robson, Ibid., ii, p. 215; Smalley, Ibid., ii, p. 152; Sequeria, G. W., Ibid., ii, p. 80; Tyson, Ibid., ii, p. 45; Rendu, Lyon Med., 1916, cxxv, p. 215.

[6] Ugeskr. f. Laeger. Kbenh., 1918, lxxx, p. 258; Olivarius, Ibid., p. 350; Tornholt, Ibid., p. 351; Jacobson, Ibid., p. 352.

[7] Jour. Cut. Dis., 1918, xxxvi, p. 530; match box dermatitis.

[8] Ibid., 1919, xxxvii, p. 125.

[9] Lyon Méd., April, 1913, p. 1884.

lar and bullous dermatitis and at times, even in mild solution, induces gangrene.[1] Iodoform occasionally is followed by a scarlatiniform eruption which spreads far beyond the point of application. It sometimes produces erythema, vesicles and wheals.[2] Mercury and its salts produce dermatitis of varying grade. In addition to these, chrysarobin, sulphur, pyrogallic acid and tar frequently produce dermatitis, the degree of which depends upon the strength of the application and its duration. Metol, a substance largely used by amateur photographers, produces in susceptible skins a marked dermatitis, similar to that caused by ivy. Several examples have been seen by the author. Beevo[3] and others report similar observations. Orthoform has been the causative factor in a number of cases of acute vesicular and bullous dermatitis coming under our observation. Dubreuilh[4] reports cases of gangrene following the local application of this drug. Formalin causes vesicular and pustular lesions of the fingers in predisposed individuals. In many instances prolonged contact is required to induce the reaction.

Other drugs which occasionally cause dermatitis are most of the strong acids and alkalies, croton oil, cantharides, tartar emetic, mezeron, turpentine, ether, chloroform, resorcin and many others. Irvine[5] reported cases of dermatitis produced by Bénétol. Cases of dermatitis occurring in workers in coco-bolo wood have been recorded by MacKee;[6] due to teak by Wilmot Evans;[7] to copra dust by MacLeod.[8]

Diagnosis.—An acute dermatitis appearing suddenly on regions of the body readily exposed to outside irritants should always arouse suspicion of dermatitis venenata. A history of contact with some irritating substance can usually be obtained. The inflammation in the beginning is limited to the areas with which the toxic agent comes in contact, the inflammatory process often reaching its point of greatest intensity and subsiding soon after the removal of the cause. In the summer months an acute dermatitis involving the hands and face should always suggest inquiry concerning ivy, primrose or other plants. In the colder seasons a dermatitis about the neck suggests investigation of the collar. At any season a dermatitis in the scalp, spreading over the ears and neck, is always suggestive of hair dye. The differentiation of acute dermatitis of this type from acute exacerbations of eczema is difficult. The history of direct contact with an irritant and the complete subsidence of the disorder upon removal of the cause are points of importance. The acute and subacute recurring forms of primrose dermatitis resemble closely similar forms of eczema, a fact to be remembered in seeking the cause of the latter disorder.

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[1] Harrington, Amer. Jour. Med. Sc., 1900, cxix, p. 1, "Report of 18 cases and review of 118 cases from the literature."

[2] *Cf.* Paper of R. W. Taylor, read before New York Academy of Medicine, 1887.

[3] New York Med. Jour., September 12, 1908, p. 506.

[4] La Presse Méd., 1901, liii, p. 233.

[5] St. Paul Med. Jour., 1912, xxix, p. 624.

[6] Jour. Cut. Dis., 1913, xxxi, p. 582.

[7] Brit. Jour. Derm., 1905, xvii, p. 447.

[8] Ibid., 1915, xxvii, p. 118.

Treatment.—Internal treatment is, as a rule, not required. Local treatment is similar to that of acute eczema. Zinc oxid and aqua calcis lotion, calamine lotion or black wash—preferably diluted, solutions of sugar of lead, or oleated lime water are all of service. In dermatitis due to ivy the application of an alkalie has been recommended for the purpose of neutralizing the poisonous volatile alkaloid in the leaves of the plant. As a rule, when the patient comes under observation the resulting inflammatory process is all that needs treatment.

A large number of remedial agents have been suggested for this condition. A calamine lotion, such as the following, has been found of value in most cases due to Rhus poisoning. One-fourth of 1 per cent. of phenol may be added, if necessary, to control the itching:

℞—Calaminæ,			
Zinci oxidi,	āā	℥ss;	15
Glycerini,			
Sodii biboratis,	āā	ʒij;	8
Aq. calcis,	q. s. ad.	℥viij;	240

In a dermatitis due to formalin, radiotherapy is of much value. In primrose dermatitis Foerster recommends the early application of alcohol. As the dermatitis subsides soothing ointments or pastes may be used to complete resolution.

Schamberg[1] recommends the following as a preventive and curative measure in ivy dermatitis:

℞—Tinct. rhus toxicodendris,		grs. xv;	1
Alcoholis,		ʒi–grs. xv;	5
Syp. aurantii,	q. s. ad.	℥iij–ʒj;	100

For immunizing purposes the preparation is administered in one-half glass of water after each meal as follows: Beginning with one drop the dosage is increased one drop each dose until the dosage reaches twenty-one drops. Thereafter one teaspoonful is administered daily throughout the ivy season. During an attack the same method is employed, beginning with one drop, increasing to eighteen after which teaspoonful doses are continued once daily. Strickler[2] suggests the employment of the toxin of various plants by intramuscular injection both as a desensitizing and curative measure.

Prognosis.—Whereas, as a rule, cases of dermatitis venenata recover in a reasonable time, there may be a recurrence when the cause again becomes operative; most important is the fact that occasionally a dermatitis of this kind so sensitizes the skin that other irritants of a dissimilar nature may cause a recurrence, until finally a persistent eczema results.

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[1] Jour. Amer. Med. Assoc., 1919, lxxiii, p. 1213. Desensitization of persons against ivy poison.

[2] Jour. Cut. Dis., 1918, xxxvi, p. 327. The treatment of dermatitis venenata by vegetable toxins.

Dermatitis Calorica.—Definition.—*Burns.*[1]—Burns follow excessive heat applied to the skin and are characterized by erythema, edema, vesicles, bullæ, gangrene, and in severe cases are accompanied by varying constitutional disturbances.

Symptoms.—Burns are produced by dry or moist heat. Fire, boiling liquids, hot steam, superheated air, inflammable liquids, such as gasoline, benzine, turpentine, molten metal and gas explosions are the most common causes. It is usual to divide the symptoms according to the intensity of the process into three degrees. The degree depends upon the intensity of the heat and the duration of contact.

Burns of the first degree are produced by a temperature of about 140° F., and are characterized by erythema, moderate edema and smarting sensations, the entire process lasting from one to three days.

With higher temperatures, 160° to 180° F. more active inflammatory symptoms intervene. The area becomes red, markedly swollen and covered with vesicles and bullæ. The latter may be isolated or confluent, and may cover large surfaces. The contents of the vesicles and bullæ are clear at first, and later become turbid. The vesicles rupture easily and thereafter become covered with crusts, or exfoliate leaving raw, tender and painful areas, or they may occasionally dry and undergo resolution without rupture. Severe pain and tenderness accompany this group. Secondary infection may supervene and constitutional symptoms of varying grade may be present. Burns of this degree commonly clear up within one to several weeks.

In burns of the third degree the temperature ranges from 210° to 440° F. and are caused by boiling liquids, gas explosions and molten metals. This grade is characterized by the formation of an eschar. The eschar is gray or brownish-black, insensitive to the touch, may be covered by abortive vesicles, and is leathery in consistency. It is usually surrounded by an infection of milder degree. The eschar is at first firmly attached to the underlying tissues, but in the course of eight to ten days becomes separated by secondary pyogenic infection which begins at the edges and spreads beneath its entire circumference. After its removal the surface is raw, granulated, and presents a seropurulent discharge and bleeds easily when touched. In a short time it becomes covered with small bluish-white islands of epithelium springing from partly destroyed glands and remnants of the rete. These islands enlarge, coalesce, and by joining the new epithelium advancing from the margins, finally cover the entire area. The resulting scar is of a pinkish hue when first formed and later becomes white. The scar may be smooth and shiny in areas, owing to the complete loss of cutaneous glands, or it may be irregular and corded, and by contracture may produce deformity. When the eschar is deeper healing occurs entirely from the edges, and the resulting scar is characterized by great irregularities, ridges, and bands across the surface, forming pockets and folds, and in many instances colloidal and hypertrophic changes result which, by contraction, induce much deformity. Ectropion and loss of the use of joints may result from these contracting scars.

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[1] McLeod, Burns and Their Treatment, Oxford War Primers, London, 1918.

The early constitutional disturbances are prostration and shock. In severe cases death occurs usually within forty-eight hours. Pain in these cases may be moderate. In milder cases great nervousness, severe pain and varying degrees of prostration occur. The shock lasts from twenty-four to forty-eight hours and usually comes on immediately, although it may be delayed for twenty-four hours. It varies in intensity and is characterized by a rapid, feeble pulse, low blood-pressure, subnormal temperature, a moist, clammy skin, nausea and thirst. The urine is diminished in amount and may contain albumin. If the patient survives the shock secondary effects occur which include septicemia, erysipelas, and tetanus. Meningitis, pleuritis, bronchopneumonia, peritonitis, duodenal ulcer and nephritis may occur as later complications.

Treatment.[1]—Owing to the large number and the severe character of burns that occurred during the late war their treatment was given intensive study which resulted in improvement over older methods. Burns of the first degree require but little treatment. A wet dressing of lead water, aluminum acetate, or sodium bicarbonate for a day or two, followed by a simple cream, such as bismuth subnitrate, 4 grams, zinc oxid, 8 grams oleum olivæ and aqua calcis āā 120 grams, or calamine and zinc oxid āā 15 grams, oleum amygd. exp. and aqua calcis āā 120 grams (MacLeod) will be sufficient.

For burns of more severe grade the surface should be carefully cleansed with a mild antiseptic solution, such as boric acid or aluminum acetate, the bullæ opened and drained under strict asepsis, then a paraffin dressing applied. Or, if the dry method be selected, the burned area is left exposed to the air and kept dry by the constant application of a powder. A tent may be necessary to hold the clothing away from the part. MacLeod recommends the following powder applied with an insufflator. Stearate of zinc 20 per cent., magnesium carbonate, 40 per cent., lycopodium, 5 per cent., diamatos, 25 per cent., paraffin, 10 per cent., oil of citronella, q. s. Stearate of zinc, or borated talcum may be employed. A natural crust forms by this method and the end-result is good. When paraffin is used an artificial crust is produced under which healing occurs. This method has been employed by the French for some time and during the war was emphasized by Barthe de Sandfort, who brought forth a proprietary preparation under the name of Ambrine. Many modifications of this were suggested.

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[1] Hull, Lancet, London, 1917, i, p. 271, Dressing (The) of Burns with Paraffin Wax; Hurd, Lancet, London, 1917, i, 855, The Paraffin Treatment of Burns; Editorial, Lancet, London, 1917, ii, p. 578, Paraffin and Ambrine in the Treatment of Wounds; Thorn, Lancet, London, 1917, i, p. 246, The Conservative Treatment of Scalds and Burns; Sollman, Jour. Amer. Med. Assn., 1917, lxviii, p. 1799, Developments in the Paraffin Treatment of Burns and Other Open Wounds; Leech, Jour. Amer. Med. Assn., 1917, lxviii, p. 1497, Ambrine and Paraffin Films.

Hull (loc. cit.) recommended a paraffin dressing composed of the following:

Resorcinolis,	grs. xv;	1	
or			
Betanaphtholis,	grs. iv;		25
Eucalyptolis,	ʒss;	2	
Olei olivæ,	ʒi–grs. xv;	5	
Paraffini molle,	ʒvi–grs. xv;	25	
Paraffini durum,	℥ii–ʒij:	67	75

The above or any similar paraffin preparation is applied by first melting it over a water bath and then spraying on or painting over the surface. A special device is required for spraying; the painting may be done with a broad camel's-hair brush. A layer of cotton wool is laid over the melted paraffin and this is in turn covered with more of the paraffin preparation, and the entire dressing held in place with a loose bandage. The dressings are renewed at twenty-four-hour intervals, the surface being gently cleansed at each dressing as at first. This method relieves pain and gives thorough protection. The dressings are easily removed without injury to the skin and the growth of secondary invading microörganisms is inhibited.

Should either of the two methods above suggested not be practicable, moist dressings may be applied, using boric acid‸ aluminum acetate; or Dakin's hypochlorite solution. The disadvantages with these preparations here lie in the injury resulting from the removal of the dressing when dry. This may be partially overcome by employing moist compresses over a lattice of gutta percha tissue strips as follows: Strips one-fourth inch wide of moist, sterile gutta-percha tissue are laid smoothly and easily over the surface with narrow spaces between them. The first layer is crossed by a second at right angles. The surface is then practically covered with the rubber tissue leaving, however, at each crossing of the strips small openings for the escape of secretion. The moist dressings may be changed frequently over this without damage to the tissues beneath.

Picric acid solution of a strength of 1 per cent. is highly recommended by many. Sims[1] recommends boric acid as a wet dressing (6.4 grams to 480 grams water) as an early or emergency measure, to be followed later by paraffin film treatment. In the deep and destructive cases it is often necessary to resort to skin grafting.

Dermatitis Congelationis.--Exposure to extreme cold produces varying grades of inflammation of the parts which is very similar to that induced by intense heat. The regions attacked are usually the ears, nose, cheeks, fingers and toes, and the process fluctuates from transitory erythema to deep, destructive inflammation, including gangrene.

In the first degree, which usually follows short exposure to extreme cold, erythema and moderate swelling occur after the parts are warmed and circulation restored. During the freezing there is slight pain, followed by loss of sensation and the area presents a pale appearance from constriction of the bloodvessels. As the circulation is restored, hyperemia and edema follow.

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[1] U. S. N. Med. Bull. Suppl., Washington, 1918, v, p. 1.

In the second degree, the edema and erythema are increased with the production of vesicles and bullæ. These undergo resolution after several days, without sequels.

In the third degree, gangrene may occur with or without the formation of bullæ. The frozen part becomes insensitive, white and cold, without the circulation of blood and lymph currents. After reaction occurs an eschar is formed which differs according to the length of the exposure to cold. If, in addition to the interference with circulation, the tissue has been destroyed, gangrene supervenes immediately upon reaction. Upon the eschar bullæ occasionally form, and not infrequently contain blood, or the skin over the area may be smooth, whitish or marbled with bluish lines, cold and insensitive. Gangrene follows and within a short time the well-known line of demarcation appears. The gangrenous portion is separated by suppurative action and after being cast off the area heals by granulation and cicatrization. Occasionally the bones of the extremities participate in the process, while in certain cases septicemia and a fatal termination result.

While freezing temperatures usually are necessary to produce the above-described symptoms, water above the freezing point in certain conditions causes similar phenomena. Early in the world war numerous examples were reported by English and other observers under the title of "Frost Bite."[1] These cases occurred among soldiers in the trenches, who frequently remained standing in water for several days without removing their boots. Pressure of the wet boots on the dependent parts, together with the cold water, was efficient in producing a dermatitis varying in degree from erythema to gangrene. Edema, bullæ (serous and hemorrhagic), and dry gangrene frequently were observed. Sensory disturbances differed and were both local and general.

Treatment.—In cases of freezing the temperature should be gradually restored, as rapid warming results in painful and dangerous changes. In cases of severe congelations the circulation should be slowly restored by friction performed in an apartment where the air is cool, to prevent too energetic reaction. Friction with snow is used with safety and success when this is obtainable. Perseverance for some time with this method is not infrequently rewarded with good results in cases that were apparently beyond restoration. The subsequent treatment consists in antiseptic dressings, usually employed for sloughing and ulcerating areas. In severe cases surgical interference is frequently necessary.

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[1] Fearnsides, Brit. Jour. Derm., 1915, xxvii, p. 33, "Frost Bite," A full description of the subject; Fearnsides and Culpin, Brit. Med. Jour., 1915, i, p. 84; Smith, Ritchie and Dawson, Ibid., 1915, ii, p. 933; Monroe and McGill, Ibid., 1915, ii, p. 926.

Dermatitis Medicamentosa.[1]—**Synonyms.**—Drug eruptions; Fr., *Eruptions médicamenteuses;* Ger., *Arzneiexantheme.*

Under this title the eruptions induced by the internal action of drugs will be considered. This includes those ingested, absorbed through the skin or mucosa, and those given by hypodermic injection. The local action induced by drugs applied to the skin is considered in the section devoted to dermatitis venenata.

Symptoms.—The eruptions caused by the internal use of drugs are numerous and varied. They may be generalized or limited to certain regions, but are usually symmetrical in distribution. Special areas, such as the face, neck, forearms—especially about the wrist, the lower limbs—particularly below the knees are more commonly selected, though a more general distribution may occur. In form they represent practically all the cutaneous lesions that are described, from superficial transient erythema to gangrene. The time at which an eruption develops after ingestion also has a wide range of variation. The erythematous eruptions usually appear soon, from a few hours to a day or two, whereas those induced by the bromids, for example, may not develop until the drug has been taken for a long period of time. In general, the eruptive manifestations are not characteristic of a particular drug, and a single drug may produce many types of eruption, and on the other hand, a single eruption is induced by many different drugs. Arsenic may produce nearly every type of eruption, yet some of these are characteristic of the drug, so also, some of the eruptions of the bromids and iodids are characteristic. Aside from these the particular drug can hardly be suspected from the cutaneous manifestations. The major portion of eruptions resemble dermatoses commonly described as being due to various toxemias, and are exhibited as simple and polymorphous erythemas and urticarias and less often the more serious ones are exhibited as vesicles, bullæ, pustules, ulcerations and gangrene. As a rule, drug eruptions appear suddenly and disappear promptly upon the withdrawal of the active cause. The iodin and bromin group are exceptions. The latter usually appear after prolonged use of the drug and are slow in undergoing involution. Commonly, the drug exanthem is brighter colored than the disorder it simulates and is usually accompanied by sensations of itching and burning. The iodin and bromin group are again exceptions. The exanthems of moderate degree are accompanied by mild, if any, constitutional symptoms, but severe symptoms may accompany generalized and intense eruptions. These are especially likely to occur in the scarlatiniform or morbilliform variety.

Etiology.—Drug eruptions occur more commonly in women and children. Defective elimination is an important factor in some instances. A neurotic temperament is said to influence the occurrence of these eruptions. From the earliest observations until the present the most important factor has been a peculiar hypersensitiveness, termed idiosyncrasy. This condition may be inherited or acquired. All attempts to discover why a hypersensitiveness exists or has developed in certain individuals, have met with failure. More recently, the action of certain drugs has been compared to that induced in some instances by the introduction of a foreign protein inducing anaphylactic reactions, and the similarity is obvious. A remarkable example of idiosyncrasy was reported by Stelwagon.[1] The theories regarding the production of drug eruptions have all met with opposition. They have been ascribed to the local toxic effect of the drug in the skin; to the action of a toxic substance induced by the drug through its absorption and excretion, and to the toxic action of the drug on the vasomotor and other nervous centers. The supposed local irritative action of the iodids and the bromids by elimination through the sebaceous glands was supported by the discovery of the substance in the local lesions. Engman and Mook[2] in an extended study found the sebaceous glands not implicated in the process, and discarded the theory that elimination through this avenue induced the eruption. They believe the eruption is due to the toxic action of the drug in the circulation, acting on tissue previously congested by various factors, such as comedones, acne, seborrheic lesions, traumatism, and scars, the resulting inflammatory condition being similar to inflammation induced by other toxic substances.

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[1] Monograph on drug eruptions by Prince A. Morrow, edited by T. Colcott Fox; The New Sydenham Society. *Cf.* for complete description with bibliography to that date. Van Harlingen, Trans. Amer. Derm. Assoc., 1880.

The histology which has been carefully studied by many observers, merely shows the ordinary signs of inflammation in varying grades.

Diagnosis.—The important points in the recognition of drug eruptions are the sudden appearance of the eruption, its symmetrical arrangement, its more or less generalized distribution, the brighter color of the lesions than those of the disorder they resemble, the presence of itching and absence, as a rule, of general constitutional disturbances (exceptions being noted by such drugs as veronal). Excluding syphilis and the exanthematous disorders, a generalized eruption of sudden onset suggests dermatitis medicamentosa. The important factor in all cases is to recognize the eruption as being produced by some drug, the specific drug not being important.

Treatment.—In the major portion of cases it is only necessary to terminate the use of the drug, when recovery soon follows. In the more serious iodin and bromin eruptions eliminative treatment is important and a local dressing of salicylic acid or ammoniated mercury hastens recovery. Crocker recommends small doses of arsenic to hasten the slow involution of the large plaques occurring with these drugs. Arsenic is also of value in preventing the acneform lesions occurring in patients to whom the iodids must be given in certain stages of syphilis.

Below will be found, arranged in alphabetical order, a list of some of the drugs which produce eruptions, and a short description of the eruption.

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[1] Jour. Cut. Dis., 1902, xx, p. 13.

[2] Ibid., 1906, xxiv, p. 502.

Acetanilid occasionally produces an erythematous or maculo-papular eruption or when long continued may cause partial cyanosis.

Acids.—Benzoic acid and sodium benzoate may produce a generalized maculo-papular eruption or well-defined patches of erythema, accompanied by itching and followed by scaling, or a scarlatiniform erythema or urticarial lesions.

Boric Acid and *Sodium Biborate.*[1]—Erythema, papules, vesicles, bullæ and lesions resembling erythema multiforme[2] may be induced by this agent. Scaling patches resembling psoriasis have been noted.[3] An erythematous eruption occurring on face, trunk and extremities is recorded by Modadewkow[4] following irrigation of the pleural cavity with a 5 per cent. solution of boric acid.

Salicylic Acid and the Salicylates.—Erythematous, scarlatiniform, urticarial, vesicular, bullous or petechial lesions sometimes accompanied by itching and followed by scaling have been noted by many observers. Engman[5] reported the case of a patient presenting an eruption of raised, shiny, smooth, erysipelatously reddened plaques undergoing resolution in the center by slight desquamation and sinking, forming more or less distinct rings.

Acetyl Salicylic Acid (*Aspirin*) induced an angioneurotic eruption in two cases recorded by Anderson.[6]

Aconite.—A vesicular eruption accompanied by itching is recorded by several observers. Bullæ and pustules rarely occur.

Antipyrin.—Ernst[7] reported five cases in which a morbilliform eruption followed the ingestion of antipyrin. Fox (T. C.) states that the eruption of antipyrin is brighter red and less concentric than that of measles. Urticarial and purpuric lesions are recorded. Veiel[8] reports edema with bullæ upon the lips, over the palate and on the toes, and urticarial lesions on the palms and soles after ingestion of antipyrin. Brocq, Darier and others have reported cases in which antipyrin has produced a more or less peristent erythema in the form of isolated, scattered, sharply defined plaques. These are usually few in number and tend to return in the same sites whenever the susceptible individual ingests the drug. Redness and pigmentation may continue for several weeks. An eruption simulating the maculo-syphiloderm was recorded by Wickham.[9]

Antitoxin.—(See Serum Eruptions.)

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[1] Wild, Lancet, 1899, i, p. 23, with bibliography.

[2] Fordyce, Jour. Cut. Dis., 1895, xiii, p. 499.

[3] Gowers, Lancet, 1881, ii, p. 546.

[4] Lancet, May 6, 1882.

[5] Jour. Cut. Dis., 1899, xvii, p. 555, with histological study.

[6] Canadian Practitioner and Review, September, 1912, xxxvii, No. 9, abstr., Jour. Cut. Dis., 1912, xxx, p. 754.

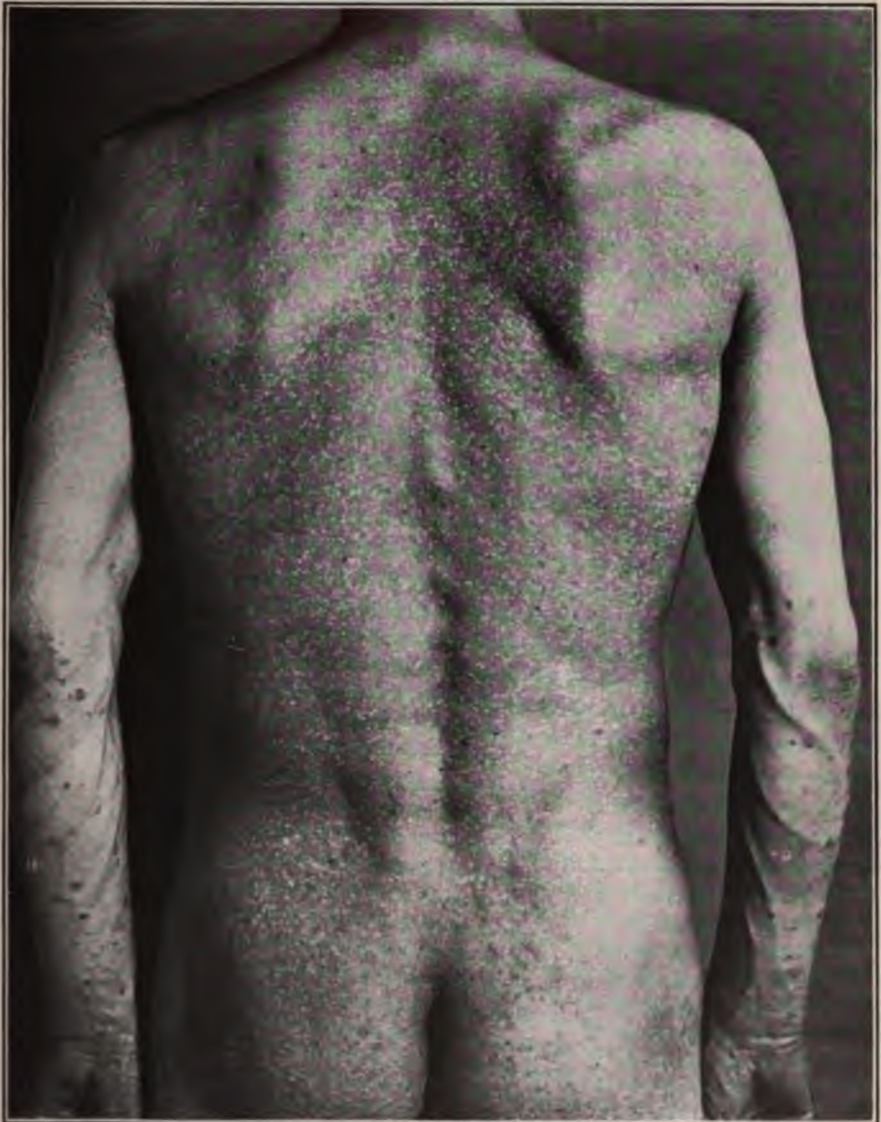
[7] Centralbl. f. klin. med., 1895.

[8] Archiv., 1891, xxiii, p. 33.

[9] Berliner Monatshefte, 1902, xxxv, p. 137; with review of literature.

Arsenic.[1]—The commonest eruptions seen after ingestion of arsenic are erythematous and pigmentary in type. Less often vesicular, bullous, pustular and ulcerative lesions occur. Herpes zoster[2] is

Fig. 61



Arsenical pigmentation. (Fordyce and MacKee.)

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[1] Brook-Roberts: The action of arsenic on the skin as observed in the recent epidemic of arsenical beer poisoning, Brit. Jour. Derm., 1901, ziii, p. 121.

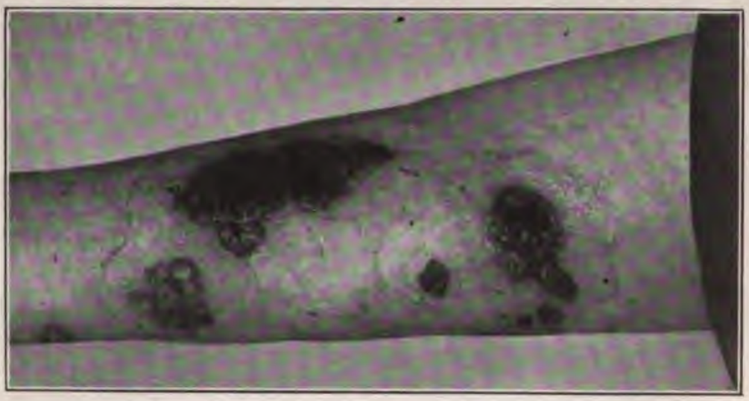
[2] See section on herpes zoster for references. Also Finlay, Jour. Cut. Dis., 1914, xxxii, p. 794; Herpes Zoster following arsenic; Hooker, Med. and Surg., Reporter Episc. Hosp., 1916, p. 101; Zoster arsenicalis.

occasionally produced by arsenic. In some cases after long-continued use hyperkeratoses are seen, upon which in rare instances epitheliomatous growths develop.[1] These untoward effects, particularly the production of hyperpigmentation and keratoses, have to be remembered when this drug is employed in the treatment of chronic diseases, such as dermatitis herpetiformis and psoriasis.

Arsphenamin (Salvarsan).—Arsphenamin and neoarsphenamin may produce a variety of eruptions, the most common being urticarial erythema and urticaria. Occasionally a scarlatiniform or morbilliform erythema, vesicular and bullous lesions, and rarely gangrene, may follow their use. A generalized dermatitis exfoliativa is an important eruption and sometimes persists over a period of several weeks or months, and in rare instances has terminated fatally.

Atrophan.[2]—Scarlatiniform erythema and other erythematous eruptions have been described as induced by this drug.

FIG. 62



Dermatitis medicamentosa due to bromids.

Belladonna.—The characteristic exanthem produced by this is a scarlatiniform erythema. This may occur in the same situations that scarlatina does and has been, as Morrow suggested, mistaken for the latter disease. The eruption has followed the instillation into the eye of atropin as a mydriatic. Constitutional symptoms sometimes occur.

Bromin and its Compounds.—The common type of eruption induced by the bromids is acneiform. By grouping and extension of these pustular lesions various peculiar types are induced. The common pustular type occurs chiefly on the face and upper portions of the trunk, and occasionally in the genital region. The distribution is that ordinarily found in acne vulgaris. The papilliform and fungating types occur more often on the extremities, though in infants these forms are seen on the face as well as other areas. In addition to the ordinary type macules, maculo-papules, moist or scaling areas of dermatitis, urticarial wheals and a variety of pustules and furuncle-like lesions, and less often vesicles, bullæ, and ulcerations occur. The characteristic eruption occurring, particularly in infants and children but occasionally in adults, especially women, consists of small-coin to nut-size, elevated nodules or flattened plaques. They resemble a flat carbuncle and have a cribriform surface, on which multiple small abscesses or pustules are visible. In infants they may assume a condylomiform appearance. These lesions may be few or numerous. They were extensively distributed over the face and limbs, with some lesions on the trunk, in two adult patients seen by the author. T. C. Fox and Gibbs[1] reported an instance of these condylomiform nodules in a case of an infant in which a careful histological study was made. Recent literature is replete with reports of examples of nodular, fungoid, papilliform and fungating lesions produced by the ingestion of the bromids. Kudisch,[2] Versilovoi,[3] Pospielov,[4] Whitehouse,[5] Oulmann,[6] Burns[7] and many others have described cases representing these varieties. A group of cases in which the papillomatous lesions resembled blastomycosis was reported by the author.[8] Similar cases

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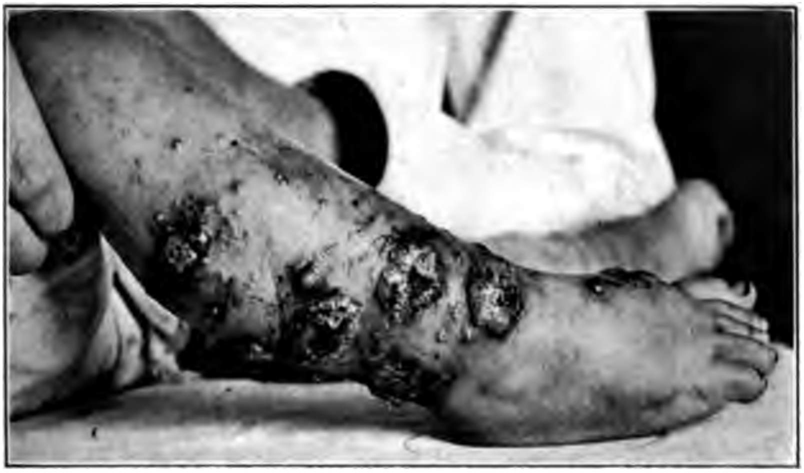
[1] Hartzell, Amer. Jour. Med. Sc., 1899, cxviii, p. 265; and Darier, Annales, 1902, iii, p. 1126.

[2] Huber-Pestalozzi, Cor.-bl. f. Schweiz. Aerzte, May, 1915; Thomsen, Ugeskr. f. Laeger Koben, 1915, lxxvii, p. 894; A case of atophan exanthem.

Fig. 63



Dermatitis medicamentosa due to bromids. (Fordyce and MacKee.)

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[1] Med. Soc. Proc., London, ix, 1886.

[2] Zeitschrift, 1912, xix, p. 713; abstr., Jour. Cut. Dis., 1913, xxxi, p. 281.

[3] Russki Journal Kojnikh E Venericheskikh Boleznei, April, 1913, No. 4, p. 311; abstr., Jour. Cut. Dis., 1913, xxxi, p. 1061.

[4] Ibid., March, 1913, No. 3, p. 200; abstr., ibid., p. 1060.

[5] Jour. Cut. Dis., 1913, xxxi, p. 497.

[6] Ibid., 1911, xxix, p. 239.

[7] Ibid., p. 35.

[8] Ibid., 1909, xxvii, p. 445.

have since been recorded by Stelwagon and Gaskill,[1] Gaskill,[2] Weiss,[3] Irvine[4] and King-Smith.[5] The eruptions produced by bromids commonly appear after the long continued use of the drug. Kaposi observed a case of bromid eruption in a nine-months-old infant whose mother had been taking potassium bromid for two months without exhibiting any eruptive manifestations. Hartzell[6] records the occurrence of a marked eruption in an infant whose mother had taken a bromid mixture during the last two weeks of pregnancy. A similar example was seen by the author in an infant at the Cook County Hospital.

Cannabis Indica.—An eruption produced by this drug is rarely seen. Such an example was recorded by Hyde,[7] in which an extensive papulo-vesicular exanthem occurred after the administration of 1 grain (0.066) of the extract.

Cantharides.—Erythematous and papular eruptions are reported in a few instances.

Capsicum.—Erythema results occasionally.

Chloral.—An erythematous eruption is the most common type produced by chloral, though urticarial wheals, red and yellowish papules, vesicles, pustules and purpuric lesions have been observed. The eruption is seen chiefly upon the face and neck, trunk and limbs, particularly on the external surfaces of the latter. Large, well defined, scaling patches of dermatitis present over the knees and lower extremities were noted by Hyde[8] and a polymorphous eruption consisting of an erythematous and scarlatiniform type commingled with urticarial and purpuric lesions occurring upon the face and neck, the chest, the extensor surfaces of the larger joints, and the dorsi of the hands and feet, was recorded by Martinet.[9]

Chloralamid.—Pye-Smith[10] reported the case of a patient in whom this drug produced a generalized scarlatiniform eruption followed by free desquamation. The mucous membrane of the mouth was hyperemic and there was a catarrhal discharge from the nose. Moderate elevation of temperature was also noted.

Chloroform.—A temporary erythema and, more rarely, purpuric spots have been noted during the inhalation of chloroform and ether.

Condurango.—Guntz[11] reports the occurrence of furuncular and acneiform lesions in 20 patients out of 1000 who were taking condurango for the relief of syphilis.

Copaiba and Cubebs.—Occasionally, the ingestion of copaiba is followed by a vividly red rash, in the form of discrete macules, more

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[1] Jour. Cut. Dis., 1913, xli, p. 429.

[2] Jour. Amer. Med. Assoc., 1914, lxii, p. 912.

[3] Trans. Sec. on Derm., Amer. Med. Assoc., 1914, p. 248.

[4] Jour. Cut. Dis., 1914, xxxii, p. 568.

[5] Canadian Practitioner and Review, April, 1915.

[6] Diseases of the Skin, 1919, p. 218.

[7] New York Med. Record, May 11, 1878.

[8] Diseases of the Skin, 1909, p. 263.

[9] Thèse de Paris, 1879.

[10] Clin. Soc. Trans., 1890, xxii, p. 137.

[11] Vierteljahr, 1882, ix.

rarely maculo-papules, invading chiefly the lower segments of the extremities and the skin of the abdomen, but often completely covering the body surface. The rash may occur in dark, mulberry-red petechiæ, and always is accompanied by itching. Inasmuch as the drug often is administered for the relief of a venereal disorder not syphilitic, care should be taken not to confound the eruption it may excite with the early macular syphiloderm. Cubebs is followed much more rarely by a similar result.

Digitalis.—Scarlatiniform erythema papules and elevated papular patches are recorded as induced by this drug.

Ergot.—Eruptions following this drug when given in medicinal doses are rare. When taken in food, however, they are common. Vesicles, pustules, small furuncle-like lesions and occasionally petechiæ are noted. Circumscribed areas of gangrene on the extremities are of more common occurrence.

Eucalyptus produces an eruption of bright-red, cherry-red, and brownish-red papules and nodules, mostly circumscribed, but confluent on the toes and fingers, in a case reported by Oppenheim.[1] The lesions were situated on the hands and feet chiefly, and were accompanied by moderate itching and preceded by mild general symptoms.

Fibrolysin.—Injections producing a general erythema are recorded by Tausard and Raillet.[2]

Guaiacum.—This drug produced a general eruption of large, elevated, erythematous plaques in a case recorded by Kingsbury.[3]

Iodin and its Compounds.[4]—The eruptive manifestations of the iodids may be erythematous, urticarial, vesicular, bullous, papulo-pustular, anthracoid or carbuncular, petechial or nodular. The common type is pustular and occurs to a mild degree in a large proportion of all those who take the drug. The pustules are usually situated upon a firm base, are more or less inflammatory, sometimes accompanied by subjective sensations, and not infrequently followed by cicatrices. By grouping and coalescence of these lesions unusual types are produced. Large cherry-sized, nodular or papillomatous flat elevations occur which present a cribriform structure showing the open ducts of several suppurating follicles. These lesions rise abruptly from the surface. A few cases are reported in which fungating tumors were found, producing an appearance almost identical with that of mycosis fungoides. Duhring described an annular patch upon the forehead made up of minute vesico-pustules which eventually developed into a globular, violaceous mass nearly two inches in diameter. Fordyce[5] recorded an unusual eruption produced by potassium iodid

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[1] Derm. Wchnschr., 1912, liv, p. 224; abstr., Jour. Cut. Dis., 1912, xxx, p. 304.

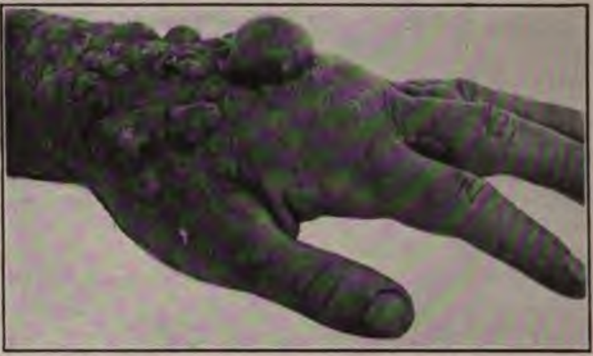
[2] Bull. Soc., Fr. de Derm. et de Syph., 1908, p. 83; abstr., Jour. Cut. Dis., 1909, xxvii, p. 44.

[3] Jour. Cut. Dis., 1912, xxx, p. 214.

[4] D. W. Montgomery, Trans. Med. Soc., State of Cal., 1900; review of subject with bibliography; and Rosenthal, Archiv, 1901, lvii, p. 3; review of subject with account of histological changes in one case.

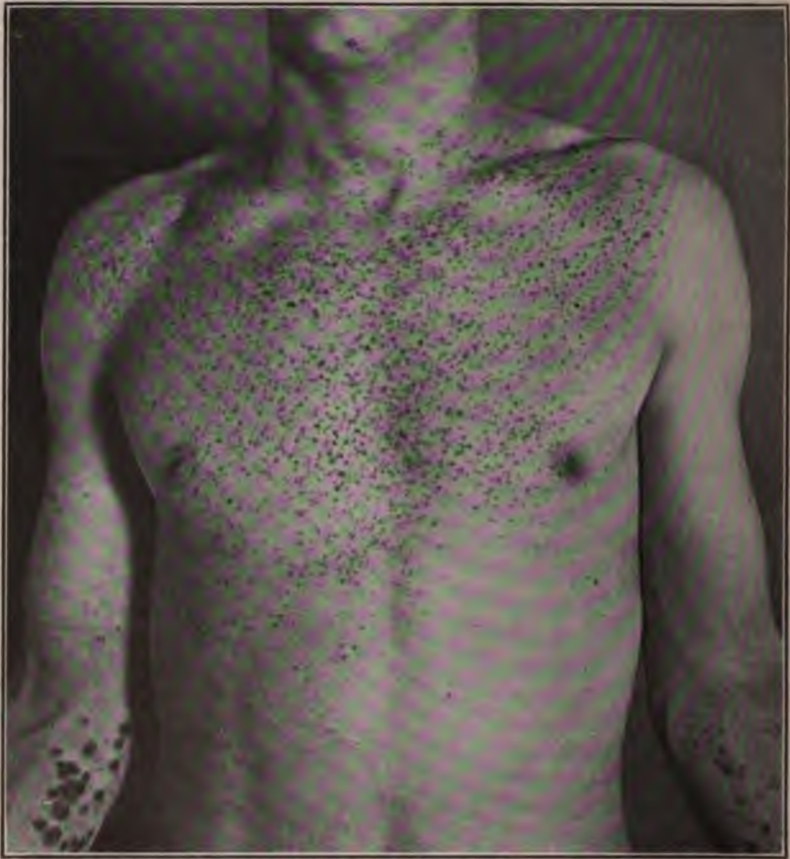
[5] Jour. Cut. Dis., 1914, xxxii, p. 805; "Nodular iododerma."

FIG. 64



Bullous eruption from potassium iodid. (Morrow.)

FIG. 65



Hemorrhagic vesicles and bullæ resulting from ingestion of iodids.
(Fordyce and MacKee.)

consisting of nodules and tumors, walnut- to orange-size, which developed suddenly and disappeared in a few days. They developed only when the drug was being taken.

The macular eruption is best seen over the upper extremities, where it is exhibited as discrete, erythematous patches, or as a diffuse blush. On the hands a cyanotic appearance may be present. Occasionally it assumes a papular type, in which case the papules are situated largely on the face. A scarlatiniform eruption associated with numerous, discrete vesicles, was recorded by Berenguier. A number of cases are on record in which the administration of the drug was followed by the production of bullæ. Bumstead, Taylor, Tilbury Fox and Hyde[1] have described bullous lesions in adults as well as in children. Hallopeau[2] reported a fatal case in which a bullous eruption followed the ingestion of potassium iodid. A purpuric eruption occasionally occurs in which petechiæ are seen chiefly over the lower extremities. In a fatal case reported by Mackenzie petechiæ developed after an infant had been given 2½ grains (0.166) grains of sodium iodid. Nodules and infiltrated plaques and tumors are recorded, occurring chiefly on the buttocks, thighs and legs. Erythema nodosum is imitated in some instances.

Iodoform.—The internal administration, or the absorption through wounds, of this drug has been followed by macular, papular, vesicular, bullous, petechial, and mixed eruptions. Grave, and even fatal systemic results are noted, including fever, delirium, emaciation, and nephritis. (For the local effects of the drug, see Dermatitis venenata.)

Jaborandi and Pilocarpin.—These drugs induce erythema, wheals and small papules accompanied by itching.

Mercury.[3]—Erythematous lesions are the most common following the use of this drug. Scarlatiniform, papular, vesicular, urticarial, pustular, purpuric and ulcerative lesions are recorded following either its ingestion or hypodermic injection.

Midal is a substance containing pyramidon, which induced erythema, wheals and purpuric lesions on the legs, accompanied by itching, in a case recorded by Bechet.[4]

Opium and its Alkaloids.—An exudative erythema is the most common type of eruption. The scarlatiniform and morbilliform erythemas with itching, followed by desquamation, together with urticarial wheals, are seen less often. More rarely, vesicles and bullæ are recorded.

Phenacetin in rare instances has induced an erythematous, urticarial eruption. A generalized erythematous eruption is recorded by Valentine.[5]

Phenolphthalein occasionally produces erythematous plaques followed by pigmentation.

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[1] Jour. Cut. Dis., 1886, iv, p. 383.

[2] Union méd, 1882, xxx, p. 481.

[3] Gottheil, Jour. Cut. Dis., 1911, xxix, p. 114; Goldenberg, ibid., 1919, xxxvii, p. 144.

[4] Jour. Amer. Med. Assoc., 1912, lix, p. 1289.

[5] Therap. Monats., 1888, p. 330.

Phosphorus.—This drug has been noted to induce bullous and purpuric lesions, associated with constitutional symptoms.

Potassium Chlorate.—An erythematous eruption induced by this drug is recorded by Stelwagon.[1]

Quinin and Cinchona Alkaloids.—The commonest and most prevalent type of eruption produced by quinin is a scarlatiniform erythema. Morrow[2] analyzed the records of over sixty cases presenting quinin eruptions and found that the prevailing type was erythematous, the eruptions being of a vivid hue, disappearing on pressure and resembling scarlatina. Other lesions produced are wheals, papules, vesicles, bullæ, petechiæ, and other purpuric lesions. Bullous formation is rare following the ingestion of quinin. Trimble[3] reported an example of this type. Quinin eruptions often follow minute dosage. Such an instance is recorded by Stelwagon.[4] The eruption usually recurs when the drug is taken and the change of form of the latter does not alter the result. An unusual eruption induced by quinin was recorded by Hyde.[5] Finger nail-sized, discrete, salmon-pinkish tinted, slightly elevated patches, accompanied by moderate itching occurred over the entire surface of the body. A single dose of 2 grains (0.133) of quinin sulphate induced the eruption and reproduced it at a later date on the repetition of a similar quantity.

The desquamation following the quinin exanthem may be moderate or marked. In some cases it closely resembles that seen following scarlet fever. Morrow suggests that many cases recorded as recurrent attacks of scarlet fever or measles were, in all probability, induced by quinin.

Salipyrin.—Edema and ulceration with loss of tissue has been reported following the administration of this drug.

Santonin.—A generalized eruption of urticarial lesions seated upon a reddened surface and accompanied by edema is reported by Sieveking as occurring in a child to whom santonin had been administered as a vermifuge.[6]

Serum Eruptions.[7]—Tuberculin, diphtheria antitoxin, and the various vaccines used as therapeutic measures frequently produce in susceptible individuals cutaneous exanthems. As the antitoxin of diphtheria is used so commonly today, the exanthems produced by its employment should be recognized.

Frequency.—Owing to the fact that different serums produce eruptions in varying proportions, and also to the fact that accurate records are kept chiefly in the hospitals, where the injections are used as a routine measure, and also by a few men especially interested in the matter, the exact proportion of persons displaying eruptions in relation to the whole number treated is difficult to determine. Hartung collected data from the literature on the subject, and from the reports of twelve observers found 294 eruptions resulting from 2661 injections, an average of 11.4 per cent.

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[1] New York Med. Record, July 21, 1883.
[2] New York Med. Jour., 1880, xxxi, p. 244.
[3] Jour. Cut. Dis., 1910, xxviii, p. 194.
[4] Loc. cit.
[5] Diseases of the Skin, 1909, p. 268.
[6] Brit. Med. Jour., February 18, 1871.
[7] Welch and Schamberg, Treatise: Acute Contagious Diseases, pp. 754-760.

Date.—The appearance of these eruptions may occur from one to thirty days after the injection. The majority appear from the sixth to the tenth day.

Character.—The important exanthems in the order of frequency of occurrence are the following: Urticarial, polymorphous, erythematous, scarlatiniform, morbilliform, vesicular and bullous, and purpuric. The last three are rare. The majority are urticarial, and may be ordinary urticarial wheals or urticarial erythema. The scarlatiniform and morbilliform varieties closely resemble the disease after which they are named. Mixed types are common and aid in diagnosis. Edema, especially of the face, about the eyelids, also of the penis, scrotum, and feet, is not infrequently noted in association with these eruptions. The distribution of the lesions is irregular. While they may occur on any part of the cutaneous surface, the sites of predilection are about the arms, legs, buttocks and trunk. The face occasionally may be attacked. The first appearance of the eruption is commonly about the site of injection. It is frequently noted that the eruption appears within twenty-four hours at the site of the injection and soon clears but reappears later generalized. The extent of the eruption varies from a few isolated, scattered patches to a profuse exanthem, involving almost the entire cutaneous surface. Its duration is usually about two days, but it may persist for three, four, or five. Purpuric lesions naturally persist for a longer period. The eruption may recur within a few days after disappearance, or after some weeks. The dates of recurrence vary from three to seventeen days. More than one recurrence may happen.

These rashes are commonly accompanied by constitutional disturbances of varying degree. There is usually a rise in temperature, with its accompanying symptoms. While this rise usually does not exceed 101° to 102°, it may be as high as 105° F. The fever lasts from one to three days, subsiding with the disappearance of the eruption. Headache, a certain amount of prostration and arthralgia are common accompaniments. The joint-pains are valuable aids in diagnosis.

It is believed that these cutaneous manifestations are induced by the serum *per se*, and that the antitoxic material has little to do with their production. Similar eruptions have been produced repeatedly by non-immunized serum.

Silver Nitrate is, in exceptional instances, followed by a bluish-gray or slate-colored change in the skin after prolonged administration.

Stramonium.—Eruptions from this drug are rare and are of the erythematous and scarlatiniform type; occasionally erysipelatous and purpuric lesions occur.

Strychnin.—Scarlatiniform eruption is said to occur from this drug.

Sulphonal.—Diffuse macular and scarlatiniform eruptions are seen occasionally. Vesicular and purpuric lesions have also been reported.

Tenacetum.—A varioliform eruption is recorded by Potter[1] as being produced by the ingestion of this drug.

Tar and Turpentine.—Erythematous, vesicular, and papular rashes are reported as resulting from the ingestion of these subjstances.

Thallium acetate induces alopecia.

Veronal.—Wills,[2] House,[3] Bulkley,[4] Wooley[5] and others have reported instances of eruptions produced by this drug. Their occurrence is due to idiosyncrasy and the lesions belong to the group of angioneurotic dermatoses. They may be exhibited as local or general exanthems. Erythema, large maculo-papules, vesicles, oval and circinate patches with dark centers resembling insect bites, scarlatiniform erythema, and edema, especially of the face, have been described. On clearing, brownish stains and petechial spots remained for a time. Constitutional symptoms of moderate grade accompanied the general eruption. Pollitzer[6] recorded a case with erosive lesions in the mouth and about the anal region, accompanied by systemic symptoms, including urinary changes. The author has seen a similar example. The patient had been in the hospital for some weeks suffering with psychosis and had been given 5 grains of veronal each evening. The eruption had a generalized distribution and appeared first on the extremities. The face was red, swollen and edematous. The eruption on the arms and trunk was morbilliform in character, in certain areas large plaques being present in addition. Intense itching accompanied the eruption. The temperature ranged from 100° to 103° F. for several days. The process subsided in about eight days.

The following drugs have induced eruptions in rare instances: Alcohol, anacardium, antimony, benzol, calcium sulphid, castor oil, creosote, chinolin, codliver oil, cocaine, conium, duboism, guarana, ipecacuanha, kava-kava, lead and its compounds, mesotan, pimpinella, rhubarb, salol, sulphonal, tanium, valerian, veratrum viride and valerian.

DERMATITIS EXFOLIATIVA.

Synonyms.—General exfoliative dermatitis, Pityriasis rubra (Crocker), Erythrodermie exfoliante (Besnier), Dermatite exfoliatrice.

Definition.—Dermatitis exfoliativa, as the term indicates, denotes a general redness of the skin accompanied by scaling. Much confusion has existed concerning this disease, and many terms have been used by various writers to designate particular groups studied. In this work the following types will be considered:

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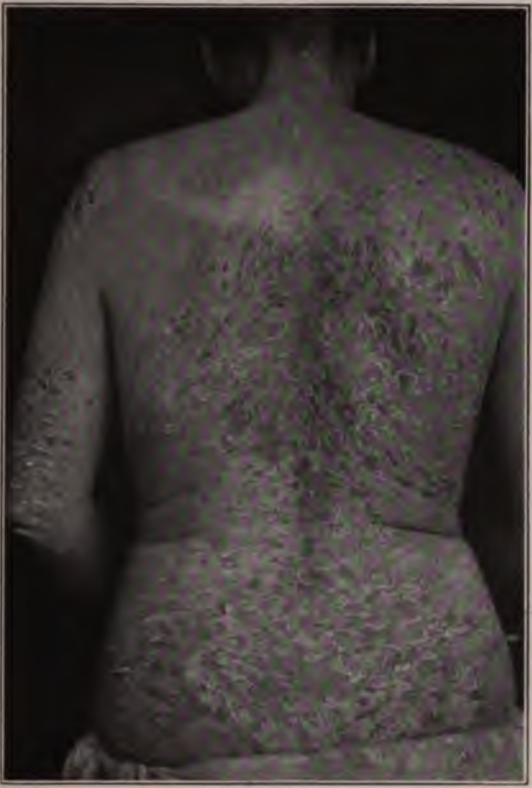
[1] New England Med. Jour., October 15, 1881.
[2] Brit. Med. Jour., March 3, 1906.
[3] Jour. Amer. Med. Assoc., 1907, xlviii, p. 1349.
[4] Ibid., 1907, xlviii, p. 1865.
[5] Ibid., 1907, xlix, p. 2153.
[6] Jour. Cut. Dis., 1912, xxx, p. 185; with additional literature.

1. *Dermatitis exfoliativa* (*Wilson*).[1] This group includes the so-called primary type. With these will be included the so-called secondary cases, originally described by Buchanan Baxter.[2] When fully developed, these secondary cases are indistinguishable from those of the primary type. This type follows such diseases as psoriasis, eczema, dermatitis seborrhoica, lichen planus, pityriasis rubra pilaris (Devergie), and dermatitis venenata, and develops either spontaneously or as the result of irritant applications to the primary disease. In this group of secondary cases, also, should be placed those produced by the local application of hydrargyrum, chrysarobin, and arnica, and possibly also those induced by the internal administration of quinin, arsenic, antipyrin, and antitoxin.

FIG. 66



Dermatitis exfoliativa.

2. *Pityriasis rubra* (*Hebra*),[3] a special type characterized by small scales, absence of infiltration, final atrophy, and commonly fatal termination.

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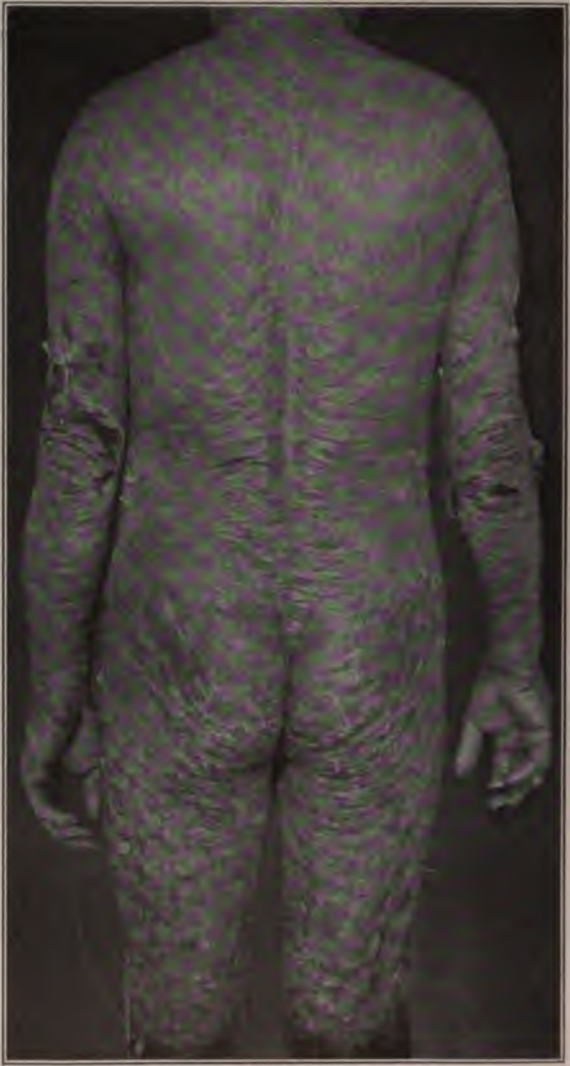
[1] Med. Times and Gazette, London, 1870, i, p. 118.

[2] Brit. Med. Jour., 1879, ii, p. 79.

[3] Diseases of the Skin. The New Sydenham Society Translation, vol. ii, p. 69.

3. *Dermatitis exfoliativa neonatorum* (*Ritter*),[1] a special type of the disease occurring in infants.

FIG. 67



Dermatitis exfoliativa. (Fordyce.)

4. *Dermatitis exfoliativa epidemica* (*Savill*),[2] a type described a occurring in epidemics, largely among the poor in homes and asylums. The recurrent scarlatiniform erythema[3] is not included in this group,

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[1] Central Zeits. f. Kinderheilk., 1878, Ed. 2.

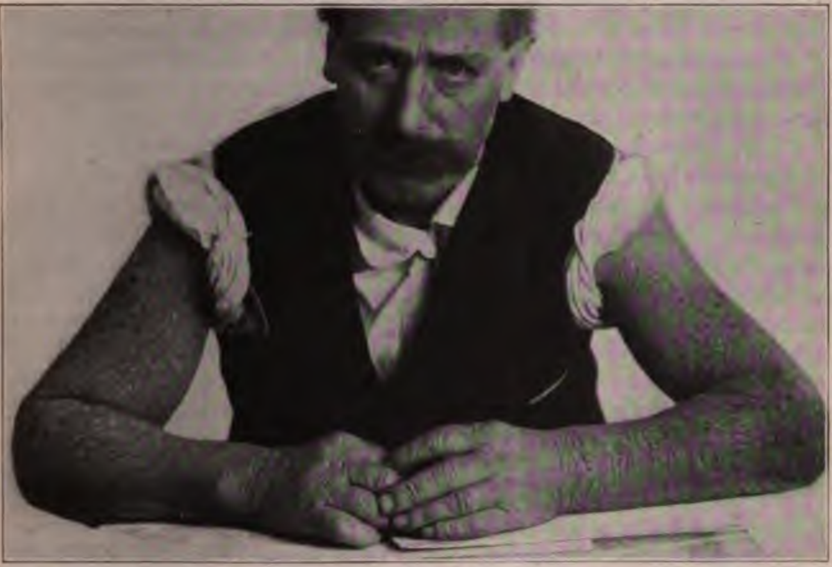
[2] On an Epidemic Skin Disease. Brit. Jour. Derm., 1892, iv, pp. 35-62 and 69–100.

[3] Féréol: Pseudo-exanthème scarlatiniforme récidivant. Bull. et Mem., Soc. méd. de Hôp. de Paris, 1876, ii, 30; and Besnier.

although it presents many features in common. In certain cases, instead of having recurrences, this disease becomes persistent, when it might be considered a member of this group (*Cf.* section on Scarlatiniform Erythema).

Dermatitis Exfoliativa (*Wilson, Brocq*).—Exfoliative dermatitis is a disorder in which, over considerable portions or the entire surface of the body, the skin is reddened and covered with lamellated scales, which are exfoliated freely from the surface. The degree of hyperemia varies in different cases. The redness displayed in the regions affected may be a bright crimson, an erysipelatous, or a purplish shade, with sometimes a faint tinge of yellow. The scales vary in size and in the rate of formation in different cases, and are usually thin and papery. They are commonly attached at one border and overlap, but in certain cases the attachment occurs in the centre. They are usually larger and more numerous over the extremities and back than over the neck, face, and chest. In the scalp they are usually matted together with sebum and form a crust. The palms of the hands and soles of the feet are usually affected late in the disease, and sometimes escape altogether. Here the epidermis peels off in large plates. In the course of the disorder the hairs may fall, and in some cases the alopecia is general. The nails become opaque and dystrophic, and there is usually thickening of the nail-bed, which pushes the nail upward, with resulting deformity. In certain cases the nails are shed.

FIG. 68



Primary exfoliative dermatitis.

The disease commonly begins with redness, upon which desquamation supervenes. The articular folds of the skin in the genital region and the head and trunk are most often the early seat of the disease, which may involve consecutively one part after another. The affection may be limited to one region, or several distinct regions may be involved simultaneously. As a rule, the disorder, beginning in patches, spreads rapidly, and in the course of from one to two weeks covers the entire surface. At times, constitutional symptoms accompany the beginning of the disorder, exhibited as malaise, chills, fever, and inappetence, and later recurring elevation of temperature is associated. In severe cases the features of the patient may be disfigured slightly by tumefaction of the lips, swelling of the ears, and puffiness of the eyelids. In most cases the skin is dry, but rarely is moistened with pathological discharge. Often there is coincident adenopathy. The mucous surfaces of the eyes, nose, mouth, and throat may participate in the general disorder and become the seat of inflammatory, and in rare cases even of pseudo-membranous and exfoliative, processes. The subjective sensations are variable. Itching and burning may be present in all degrees or absent entirely. Chilliness is always a prominent symptom.

In the secondary cases, the disease when fully developed is indistinguishable from that described above.

The course of the disease is variable; recurrences are frequent, and cases are recorded which have lasted for a half-century.[1] In certain cases there is rapid loss of strength and weight, although in many the general health is unaffected. The urine is commonly normal, although in certain cases albumin may occur. Its presence is considered of grave portent by Morris, Pringle,[2] and others. Tidy,[3] in a study of the metabolism in exfoliative dermatitis, concludes that the excretion of nitrogen and fluid in the urine is deficient, the excess being excreted by the skin, and that the excretion of uric acid is excessive; but that these changes in the urine are secondary to the changes in the skin.

Pringle[4] records cases in which the patients became insane, and gives insomnia as an important symptom.

Etiology.—Males somewhat outnumber females as subjects of the disease. The majority are between twenty and forty years of age.

The several toxemias, gout, rheumatism, tuberculosis, chronic alcoholism, the general causes of anemia, asthenia, and cachexia, have all been cited as etiological factors in the several forms of exfoliative dermatitis, and in cases each of the causes named has been effective. The disease is in many instances profoundly affected by climatic influences, often first appearing in the autumn of the year. Local applications of hydrargyrum,[5] chrysarobin, and arnica[6] have been followed by dermatitis exfoliativa, and it is possible that certain cases following the administration of quinin, arsenic, antipyrin, and antitoxin serum may be of the same type.

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[1] Mackenzie, S: On Dermatitis Exfoliativa Universalis. Brit. Jour. Derm., 1889, i, pp. 285–303, inc. A series of 21 cases.

[2] General Exfoliative Dermatitis (Pityriasis Rubra). Debate, London Dermatological Society, Brit. Jour. Derm., 1898, x, pp. 437–464, inc.

[3] Brit. Jour. Derm., 1911, xxiii, pp. 133–149, inc.

[4] Loc. cit.

[5] White: Jour. Cut. Dis., 1912, xxx, pp. 707 and 708. Case I.

[6] Crocker: Diseases of the Skin, p. 400. Bowen: Jour. Cut. Dis., 1910, xxviii, p. 1 (a fatal case).

Pathology.—The histopathology of these affections has been studied by Brocq,[1] Vidal,[2] Girode,[3] Unna,[4] Bowen,[5] and others. The findings have been exceedingly variable, due probably to the fact that the examinations have been made at different stages of the disorder, and possibly also of different types. As a rule, the following findings have been noted: a cellular infiltration of the corium, particularly the upper part, in certain cases, this being found also more deeply around the hair-follicles and sweat-glands; and other evidences of cutaneous involvement, such as dilatation of the blood-vessels, and edema, with its consequent changes. In the epidermis the changes usually noted have been parakeratosis and acanthosis. In cases of long standing pigment deposits are found in the corium.

Bowen[6] describes the histology in a typical case of the Wilson type as follows: desquamation of the horny layer in strips, with nuclei in the cells (parakeratosis); collections of leukocytes lying below the horny layer; stratum granulosum absent; rete thickened, especially the interpapillary portions (acanthosis); leukocytes between the epithelial cells; a sharply differentiated cell, stained with eosin, found here and there among the epithelial cells; in the corium vascular dilatation, with perivascular cell-infiltration in the upper portion of the papillary layer (oval and spindle connective-tissue cells, small round cells with large nuclei, and a few eosinophiles), and elongation of the papillæ. The sebaceous and coil-glands, collagen, and elastin were normal.

The pathogenesis of the disorder is not indicated by the histological findings.

Diagnosis.—The disease is to be differentiated from other scaly dermatoses. Psoriasis, lichen planus, and dermatitis seborrhoica, which sometimes develop into dermatitis exfoliativa, can be differentiated, as a rule, by recalling the distinctive features in each of these disorders. It is only in rare instances that they become generalized. The selection of particular regions of the body by these disorders, the character of the scaling, and their accompanying subjective symptoms, are all of diagnostic value. The possibility of a dermatitis exfoliativa being the prefungoid stage of granuloma fungoides should be remembered.

In more advanced stages of the disorder, the history of recurrences, and its special features would be of value in diagnosis.

From pemphigus foliaceus, dermatitis exfoliativa is distinguished by the absence of bullæ, and by the absence in most cases of grave systemic disturbance, although there may be stages of pemphigus foliaceus which present all the characteristics of a dermatitis exfoliativa.

From pityriasis rubra (Hebra) it is distinguished by the history in the latter of steady progression without remissions; the universally reddened, scaling epidermis, without infiltration; the ultimate atrophy of the skin; the not infrequent ulceration and gangrene; and, finally, the serious systemic conditions—all classic features in pityriasis rubra, not commonly found in such combination in dermatitis exfoliativa.

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[1] Annales, 1882, p. 534.

[2] Bull. Soc. méd. des Hôp., March, 1882.

[3] Annales, 1888, p. 519.

[4] Histopathology, p. 274.

[5] Loc. cit.

[6] Loc. cit.

Treatment.—As at least some cases are due to a toxemia, the general condition of the patient should be investigated thoroughly and treatment instituted to meet indications. Crocker,[1] Mook,[2] Engman, and others report favorable results after the treatment of dermatitis exfoliativa with quinin in large doses. The tolerance in the cases reported by Mook was remarkable. As high as 85 grains per day were given without producing cinchonism. Arsenic is not recommended, except in rare instances. The internal treatment should be directed toward meeting any indication suggested by the general condition. Jaborandi, pilocarpin, and aspirin may give relief, and occasionally are of value. The patient should be given complete rest, preferably in bed. Formerly applications were used to relieve itching and to keep the skin softened. The dry treatment, as suggested by Engman,[3] appears to give good results. With this treatment the patient is simply kept covered day and night with a dusting-powder. For the first day or so, this is uncomfortable, but later becomes perfectly comfortable and involution of the lesions occurs more rapidly. In case oily or greasy preparations are found necessary, the simple unguentum aquæ rosæ, U. S. P., will be found useful; or Hebra's ointment, 1 part to 4 of vaseline, with from 5 to 10 grains (0.33–0.66) of salicylic acid to the ounce (30.) of the whole, is usually grateful to the skin. An ointment often employed with great advantage in these cases over the entire cutaneous surface is:

℞—Sulphur. præcipit.,				
Acid. salicylic.,	āā	gr. ijss;		166
Bals. Peru.,		♏. x;		66
Ungt. petrolat.,				
Ungt. aq. ros.,	āā	℥ss;	15	M.

Other simple ointments and oils, with or without the addition of small amounts of salicylic acid, phenol, ichthyol, tar, or other remedies, may be of value. As a rule, mild preparations are more serviceable than the stronger remedies. One of the combinations of lime-water, olive oil, and zinc-oxid described in the treatment of eczema is occasionally of service. Emollient, starch, and hot baths are generally comfortable to the skin.

Prognosis..—In the majority of instances, the patient eventually recovers, though convalescence is protracted and delayed by frequent recurrences. A small proportion of cases progress to the formation of a universal exfoliative dermatitis, from which the patient rarely recovers. In grave and protracted cases, the general health of the patient suffers, and a fatal result may be expected. Bowen[4] reports seven cases, with a fatal issue in five.

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[1] Loc. cit.

[2] Jour. Cut. Dis., 1908, xxvi, p. 408.

[3] Quoted by C. J. White, Jour. Cut. Dis., 1912, xxx, pp. 705–715.

[4] Loc. cit.

Pityriasis Rubra (Hebra type).—**Synonyms**: Dermatitis Exfoliativa. Fr., *Pityriasis rubra aigu.*—Pityriasis rubra is a rare, chronic, and usually grave inflammatory cutaneous disease, involving, as a rule, the entire surface of the body, in which the skin, usually without infiltration, becomes deeply reddened and covered with fine scales. There is commonly no subjective sensation save that of chilliness, and the later symptoms, the sequelæ of the affection, are: shedding of the hairs, adenopathy, pigmentation, atrophy, and, as a consequence of pressure and friction effects, ulceration. The cutaneous manifestations are probably but symptoms of a systemic disease, which in the majority of cases terminates fatally.

Symptoms.—The disease is characterized by a superficial hyperemia and inflammation of the skin, declared in patches or by a diffuse redness of a vivid or lurid tint, and by an abundance of small, lamellated, bran-like scales, which are continuously exfoliated from the epidermis throughout the course of the malady. Patients rarely present themselves for observation until a considerable portion of the body-surface is involved; but Kaposi states that in two patients observed by him the disease was first noticed in the neighborhood of the articulations. There is no vesiculation, pustulation, moisture, or crusts. The palmar and plantar surfaces are usually less distinctly reddened than the face and the extremities, having at times even a pallid hue, but they always present scaling.

Under pressure with the diascope, the redness subsides or assumes a yellowish shade; while, as a rule, when the integument is gathered up between the fingers and thumb, no infiltration can be recognized. Exceptions, however, have been noticed by several observers.[1] The temperature of the skin is slightly increased. The exfoliation, as the disease progresses, is one of its most striking characteristics, the scales accumulating in large quantities in the clothing of the patient, who is engaged, as a French writer says, in the labor of stripping himself involuntarily of his epidermis.

The disease persists for months or for years, being always more severe in expression as it advances, the scales being shed more abundantly, leaving a smooth, shining, occasionally purplish, or even cyanotic, skin. In the patients observed by Jamieson[2] the skin was so dark-hued as to suggest the color of a mulatto. Gradually the patient becomes conscious of an increasing sense of chilliness, as though deprived of sufficient body-covering. Usually this is the only subjective sensation. Various grades of itching occur in certain cases, or there may be instead sensations of stiffness, burning, and tingling. Later, the integument seems to retract, as though it were insufficient to encompass the body, and becomes subject to fissure from extension and contact, while the lower extremities may be edematous. This retraction may be so marked that ectropion of the eyelids may ensue, the fingers may remain semiflexed, and wide opening of the mouth may become difficult. The skin over bony prominences becomes thin, stretched, and often fissured, or becomes the seat of superficial ulcers or gangrene. Thinning of the skin of the soles of the feet may render walking painful or impossible. The hairs and the nails lose their luster and become friable, and the hairs often fall, though the nails may escape.

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[1] Pityriasis rubra: Chicago Med. Jour. and Examiner, February, 1881.

[2] Edinburgh Med. Jour., 1880, xxv, p. 879.

The influence of this epidermal exfoliation, involving, as it does finally, every portion of the body-surface, does not fail toward the end to be felt by the vital forces. Alternating chills and febrile processes, pneumonia of a low grade, colliquative diarrhea, tuberculosis, subcutaneous abscesses, bedsores, and even gangrene of the skin, may close the scene.

Hebra and Kaposi altogether had under observation twenty-one patients affected with pityriasis rubra, who, with a single exception, died from its effects. Cases have been reported in America by G. H. Fox, Duhring, Elliot,[1] Bowen,[2] Montgomery and Bassoe,[3] Mook,[4] Wallhauser,[5] and others. We have had under observation several typical instances of the affection.

The disease is one of early or middle life, and affects preëminently the male sex. The progress of the disease is slow, lasting for years, though in a few instances it has proved rapidly fatal. The time required for extension to the entire surface of the body varies from a few days to two years or more, but averages from three to eight months. From the first, the tendency of the disease is to progress slowly to a universal atrophy of the skin. Involution of areas, or improvement of the cutaneous symptoms, is very unusual. The sweat may or may not be secreted in the course of the disease. The tongue is bright-red in the early stages; later it is covered with a brownish coat. It occasionally undergoes exfoliation. Rhagades may form, especially in the palmar and plantar regions. The chief systemic symptoms recorded are: languor, chilliness, and even severe rigors, alternating with febrile temperatures of recurrent type; albuminuria, diarrhea, pulmonary edema, icterus, interstitial pneumonia, bronchitis, rheumatism, and tuberculosis.

Etiology.—The causes of the disease are unknown. Petrini and Jadassohn[6] showed that many cases were tuberculous.

A case of pityriasis rubra of the Hebra type reported by Mueller,[7] accompanied by tuberculosis of the lymphatic glands, was more fully described, after the death of the subject, by Fabry,[8] who concludes, after recognition of the existing tuberculosis, that the contention of Jadassohn and Doutrelepont respecting the tuberculous character of a large proportion of similar cases requires further confirmation. Halle[9] is doubtful as to the existence of tuberculosis in a similar case reported by him. It must be conceded, however, that tuberculosis occurs frequently in cases of pityriasis rubra of this type. In Elliot's case[1] general tuberculosis was demonstrated post-mortem, and he believes it to be secondary to the cutaneous disease. This view, with regard to many of the cases, was held by Montgomery.[2]

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[1] Jour. Cut. Dis., 1897, xv, p. 35.

[2] Ibid., 1902, xx, p. 548.

[3] Ibid., 1906, xxiv, p. 298.

[4] Ibid., 1908, xxvi, p. 408.

[5] Jour. Amer. Med. Assoc., July 6, 1912, p. 10; abstr. Jour. Cut. Dis., 1912, xxx. p. 645.

[6] Pityriasis rubra of Hebra and its relation to tuberculosis (an exhaustive study, with bibliography and histology): Archiv, 1891, xxiii, p. 941, and ibid., 1892, xxiv, pp. 85, 273, 463.

[7] Archiv, 1907, lxxxvii, p. 255.

[8] Ibid., 1908, xci, p. 85.

[9] Ibid., 1907, lxxxviii, p. 247.

Pathology.—Tschlenow[3] states that the primary changes occur in the epidermis, producing secondary inflammation in the cutis, which ultimately leads to complete atrophy of the skin. Hebra and Fleischmann discovered coincident pulmonary, intestinal, or cerebral tuberculosis. Kaposi described an atheromatous condition of the arteries. Myelitis was discovered post-mortem in one case by Jamieson, who has been followed by others in the recognition of central and peripheral neurotic alterations. Kopytowski and Wielowicyski[4] described cocci which they think are factors in producing the disease. The histology, as revealed in the researches of Hans Hebra,[5] demonstrates that in the earlier period of the disease there is an infiltration of the integument, moderate in degree, succeeded at a later period by cutaneous atrophy, in which the rete and papillæ of the corium disappear. The connective-tissue elements undergo sclerosis; and the glands and follicles of the skin are destroyed. Pigmentation is abundant. Petrini and Jadassohn[6] reported inflammatory infiltration of the papillary and subpapillary layers of the corium, a proliferation of the connective-tissue cells, and secondary changes in the epidermis.

Diagnosis.—Many cases reported as instances of pityriasis rubra really belong to some other division of the dermatitis exfoliativa group. By recalling the characteristic symptoms, namely: the long duration, absence of infiltration, fineness of desquamation, the later atrophy of the skin, and ultimate fatal termination, a picture is presented that is characteristic and very different from psoriasis, eczema, and other disorders of this type.

Psoriasis rarely extends over the entire surface of the body, but at times it is generalized. In these exceptional forms a long history of the occurrence of typical psoriatic patches may be usually obtained; while the bleeding surface beneath the scales and the character of the latter will point to the true nature of the disease. Psoriasis occurs frequently in healthy, pityriasis rubra in cachectic, constitutions.

Extensive erythematous or squamous eczema, apart from other symptoms, can be recognized at once by the excessive distress occasioned by the eruption. In every case of generalized eczema, at one point or another, there always will be a surface which weeps. In the early stages of pityriasis rubra, the patient is not distressed with his disorder, but may have a listless expression. The scales are not scanty and adherent; they are abundant and exfoliate freely, and there is no history of moisture.

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[1] Loc. cit.

[2] Loc. cit.

[3] Archiv, 1903, lxiv, p. 21.

[4] Beitrag. zur. Klinik und Pathologischen Anatomie der Pityriasis Rubræ: Archiv, 1901, lvii, p. 33 (bibliography to date).

[5] Vierteljahr, 1876, Heft. 4, S. 508.

[6] Loc. cit.

In its early periods, pityriasis rubra can be distinguished from pemphigus foliaceus by the absence of bullæ and of the intolerable stench which is often emitted by the sufferer from the latter disease. When, however, there is present merely a generalized exfoliative dermatitis, the two disorders may well nigh be indistinguishable.

Treatment.—Arsenic administered internally seems powerless in pityriasis rubra. Cases are recorded of fatal results after the administration of this drug in large quantities for long periods of time. Kaposi records a single patient relieved by the use of phenol internally. Thyroid extract has been suggested.

A roborant treatment, including the employment of cod-liver oil, iron or quinin, is generally indicated. Quinin, as recommended by Engman and Mook, has been given a trial by us in some cases. The dose was progressively increased from the medicinal quantities usually given to 50, 60, or even 90 grains a day, care being taken that no ill effects occurred relative to the heart or ears. The tolerance of the drug was in all cases distinct and improvement marked.

Locally, the simplest are the best measures. Unguentum aquæ rosæ, U. S. P., petrolatum, lanolin, diachylon ointment, and various oily creams may be employed. In certain cases the continuous bath gives temporary relief; in others the dry treatment with dusting-powder suggested for the ordinary type of dermatitis exfoliativa is advised. The clothing should be ample and non-irritating, and the diet selected with a view to supporting the strength.

Prognosis.—The majority of all cases of pityriasis rubra of the Hebra type have terminated fatally.

Dermatitis Exfoliativa Neonatorum (*Keratolysis neonatorum. Ritter's Disease*).—Under this title Ritter von Rittershain[1] describes a rare exfoliating disease of the skin in nursing infants from six days to five weeks old, occurring most commonly in foundling asylums. The disorder begins usually as a reddened, exfoliating patch, most frequently on the lower part of the face, though it may appear first on any part of the body, and rapidly spreads until the entire surface is reddened and exfoliating. In some instances vesicles and bullæ appear early, a fact which led Richter and others to class the disease with pemphigus neonatorum. The angles of the mouth and the mucous outlets of the body frequently show fissures and are covered with crusts. Often the mucous membranes of the mouth, nose, and conjunctiva are involved. The surface of the skin beneath the scales is red, usually dry, and often excoriated. Occasionally, the surface is moist and crusted.

The duration of the disease varies. In most cases there is complete involution in from seven to ten days, with few or no constitutional symptoms. Severe cases may last a month or longer, with disturbance of the digestion and assimilation, and production often of marasmus. Pneumonia is of frequent occurrence. As the result of secondary infection, furuncles and abscesses are common; gangrene and sepsis may follow. When healing occurs, it is accomplished as a simple and gradual diminution of the erythema and cessation of the scaling. Recurrences are not uncommon.

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[1] Central-Zeitung f. Kinderheilk., 1878, Bd. ii, and Vierteljahr, 1879, vi, p. 129.

Etiology and Pathology.—Ritter[1] believed in its pyogenic origin. In two cases studied by Hedinger[2] *Staphylococcus pyogenes aureus* was recognized on bacteriological examination, and the author concludes that dermatitis exfoliativa of the newborn is merely a malignant variety of the pemphigus of infants. Kaposi considered it an exaggeration of the normal exfoliation of the newborn. Brocq[3] suggests that certain cases described as Ritter's disease may have been examples of pemphigus, and states that Behrend regards it as analogous to pemphigus foliaceous of Cazenave. Caspary[4] considered it a form of epidermolysis. Histological examinations[5] show merely a superficial inflammation, often with free exudation and excessive exfoliation of the epidermis. Skinner[6] sums up his histological findings as follows: Dilatation of the blood-vessels in the corium and hypoderm, edema of the prickle-cell layer of the epidermis, and the lifting up *en masse* of the horny layers of the epithelium from a rapid exudation of serum, tending to collect in lakes. Hazen[7] describes a high leukocyte count (45,000) and the finding of *Staphylococcus albus* in fresh vesicles.

Diagnosis.—Dermatitis exfoliativa neonatorum is likely to be confused with pemphigus neonatorum, and rarely with general exfoliative dermatitis due to syphilis. The differentiation between the first two is difficult, and, in fact, the former appears to be a variety of pemphigus neonatorum so called, the latter having recently been proven to be an impetigo of streptococcic origin. From a general exfoliative dermatitis associated with syphilis it may be differentiated by other signs of syphilis which are usually present. A close resemblance between the two has been noted by the author.

Treatment.—The nutrition of the child should be sustained with proper feeding, and the warmth of the body maintained. Locally, the surface should be kept covered with a soothing oil or soft ointment, and great care should be taken in changing the dressings not to damage the sensitive skin.

Prognosis.—The prognosis is unfavorable, as about 50 per cent. of the infants affected with the disease die, the outcome depending largely upon the strength and vitality of the child.

Erythroderma Desquamativa (*Universal dermatitis of children at the breast*).—Under this title Leiner[8] described a general exfoliative dermatitis which he considered different from all other types. His observations included 43 cases at a children's hospital in a period of five years. Of these 28 recovered and 15 died. The disorder is characterized by a general redness of the skin, with scaling and seborrheic dermatitis of the scalp and eyebrows. In the early stages the skin becomes intensely red and covered with grayish-white scales which later become lustrous and easily removable from the slightly inflamed skin. This condition is diffuse on the trunk and limbs, but occurs in areas on the hands and feet. The scalp is described as being covered with yellow crusts and scales, beneath which there is slight inflammation. The eyebrows presented similar symptoms. The skin on the flexor surfaces was dark red and edematous but presented no scales. The nails were dystrophic, and the nail bed was frequently hyperkeratotic. The glands were swollen but never broke down. In most instances there was marked intestinal disturbance. The disorder was chronic, lasting several months. In fatal cases the intestinal disorder increased and the cutaneous manifestations became exaggerated. The surface became dry, parchment-like, particularly about the mouth.

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[1] Loc. cit.

[2] Archiv., 1906, lxxx, p. 349.

[3] Le Traité elementaire de Dermatologie pratique, ii, p. 254.

[4] Vierteljahr, 1884, p. 122.

[5] Winternitz, Archiv, 1898, xliv, p. 397; Luitheln, ibid., 1899, xlvii, p. 323; and Mraček's Handbuch, Bd. i, p. 757 (full bibliography).

[6] Brit. Jour. Derm., 1910, xxii, p. 75.

[7] Jour. Cut. Dis., 1912, xxx, p. 325.

[8] Brit. Jour. Children's Diseases, 1908, v, p. 244.

The disorder was thought to be autotoxic, from intestinal involvement. In the skin *Staphylococcus albus* and pseudodiphtheritic bacilli were commonly found and, rarely, *Staphylococcus aureus* and *pyocyaneus*. No microörganisms were found in histological sections. Leiner differentiates this affection from Ritter's disease, which it closely simulates, by its chronicity and the presence of the seborrheic condition in the scalp. Lowe[1] reported 6 cases of the above disorder, four of which were in breast-fed infants. No abnormal findings were noted in the parents or elsewhere in the patients. She considered the disease as mild. The treatment suggested was abstinence from bathing and the use of dusting powder or a simple ointment, with proper regulation in the diet.

Epidemic Exfoliative Dermatitis (*Epidemic skin disease* (*Savill*), *Savill's disease*).—During the summer and autumn of 1891 an epidemic disorder with cutaneous symptoms developed in several London asylums, infirmaries and hospitals, affecting about 500 patients. The disease was studied with special care by dermatologists and other medical men. The brief sketch given below is based upon an excellent monograph, with colored and photographic illustrations, by Savill,[2] on various communications made on the subject in the columns of the *British Medical Journal* and the *London Lancet* for 1892, and on the description given by Crocker in his treatise. American cases have been recorded by Fordyce,[3] and Winfield.[4]

The disease occurred in two distinct clinical types: one with catarrhal exudation from the skin, resembling the moist forms of eczema; the other dry and non-discharging, resembling pityriasis rubra, and, according to Crocker, indistinguishable from that disease.

The eruptive features were apparently not preceded by prodromata, but gastro-intestinal disturbance (vomiting, diarrhea), and in some cases sore throat, either preceded or accompanied the appearance of the dermatosis. Except in patients of advanced years, there was usually post-occipital and cervical adenopathy, not to be explained as sympathetic with a cephalic eruption. The regions most frequently involved were the upper limbs, the scalp, and the face; the lower limbs less frequently.

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[1] Archiv. de Méd. des. enf., 1915, xviii, p. 629.

[2] Loc. cit.

[3] Jour. Cut. Dis., 1897, p. 141.

[4] Ibid., 1898, p. 73.

The skin lesions were pruritic, and were irregularly grouped, acuminate papules, with a follicular site. The face and upper extremities were more extensively invaded than the lower extremities.

The stages of the exanthem, as given by Savill, were:

(*a*) A papulo-erythematous stage, lasting from three to eight days, in which shot-like papules could be felt beneath the skin. These were discrete, and seated on a reddened, thickened, even an indurated or edematous, integument. In some cases the onset was in the form of marginate and circular nodose patches, resembling those seen in erythema nodosum. A few cases resembled ringworm, the flattened papules enlarging to a circinate annular group, with minute central vesicles, which were readily ruptured.

(*b*) An exudative stage, lasting from three to eight weeks, in which macules, vesicles, or papules soon formed a confluent eruption, the skin being of crimson hue, thickened, and scaling in flakes or in lamellated crusts in consequence of the exudation. In the moist type the papules developed to vesicles with exudation; in the dry type the exfoliation occurred in pure scales, pints of which, in some cases, could be collected from a patient's skin in a day. In other cases this exfoliation was in the form of an impalpable powder. This was characteristic of all well-marked cases.

(*c*) A stage of subsidence, in which the disease proceeded to involution, leaving the skin at first indurated, polished, and brownish in color. In many cases the new skin was raw and parchment-like, smooth, shining, and readily fissured, resembling in this respect ichthyosis. In a few instances, ectropion resulted, as a sequel of conjunctivitis. In severe cases the hair and all the nails were shed. Complications occurred with pneumonia, gangrene, and albuminuria. A few of the attendants upon the sick (children and patients of somewhat older years) were attacked; but for the most part the patients, and especially those succumbing to the disease, were individuals of advanced years of both sexes, inmates admitted for the management of other disorders to the institutions in which the disease prevailed.

Etiology and Pathology.—The cause of the disease was not satisfactorily determined. Savill and Russell[1] isolated a diplococcus from vesicles and scales which resembled *Staphylococcus pyogenes albus*, but differed culturally, in that it did not liquefy gelatin, and experimentally in its effect on inoculated animals. Its etiological importance is not settled.

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[1] Brit. Jour. Derm., 1892, iv, p. 105.

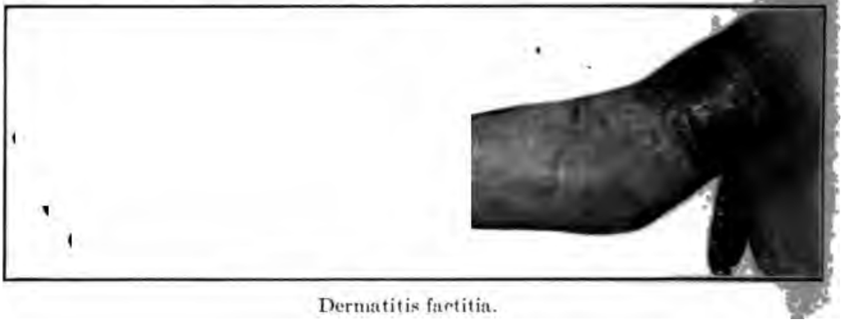
Echeverria[1] described the histology, laying stress on the presence of a peculiar change in the nuclei of the prickle-cells (peridiaphania), which he considered pathognomonic. The remainder of the changes described were in the main those found in a superficial dermatitis, including cellular infiltration in the cutis, hypertrophy of the rete, and parakeratosis with scaling.

Treatment.—Treatment, on the whole, was unsatisfactory. Local parasiticides were beneficial to a degree. Crocker recommended the treatment employed in other cases of dermatitis exfoliativa.

Prognosis.—There was a mortality of from 5 to 13 per cent., death resulting from exhaustion, with the usual signs of subsultus, shallow respiration, and coma.

Feigned Eruptions.[2]—**Synonyms.**—Dermatitis factitia; Hysterical dermatoneuroses; Hysterical gangrene; Neurotic gangrene; Spontaneous gangrene; Erythema gangrenosum.

Fig. 69



Dermatitis factitia.

Definition.—Feigned eruptions are those induced by the patient and are characterized by variable degrees of dermatitis, from simple erythema to gangrene, and are usually produced by some caustic or other irritant in neurotic or hysterical subjects.

Symptoms.—The lesions of this disorder are commonly observed on portions of the body accessible to the right hand. They are, therefore, most numerous on the anterior surface of the body, on the left arm, forearm and hand, the lower extremities and the right side of the face and neck. They are rarely seen on the right hand or arm, unless the individual be left-handed. The palms, soles, eyelids, mouth, nose, ears, scalp and genitals are usually spared. The mild and superficial forms are more common, but gangrene and ulcers are not infrequently seen. The degree and severity of the process depend not only upon the agent employed, but also upon the strength of the solution, the duration of application and the susceptibility of the tissue to which the agent is applied. A moderately weak solution of phenol

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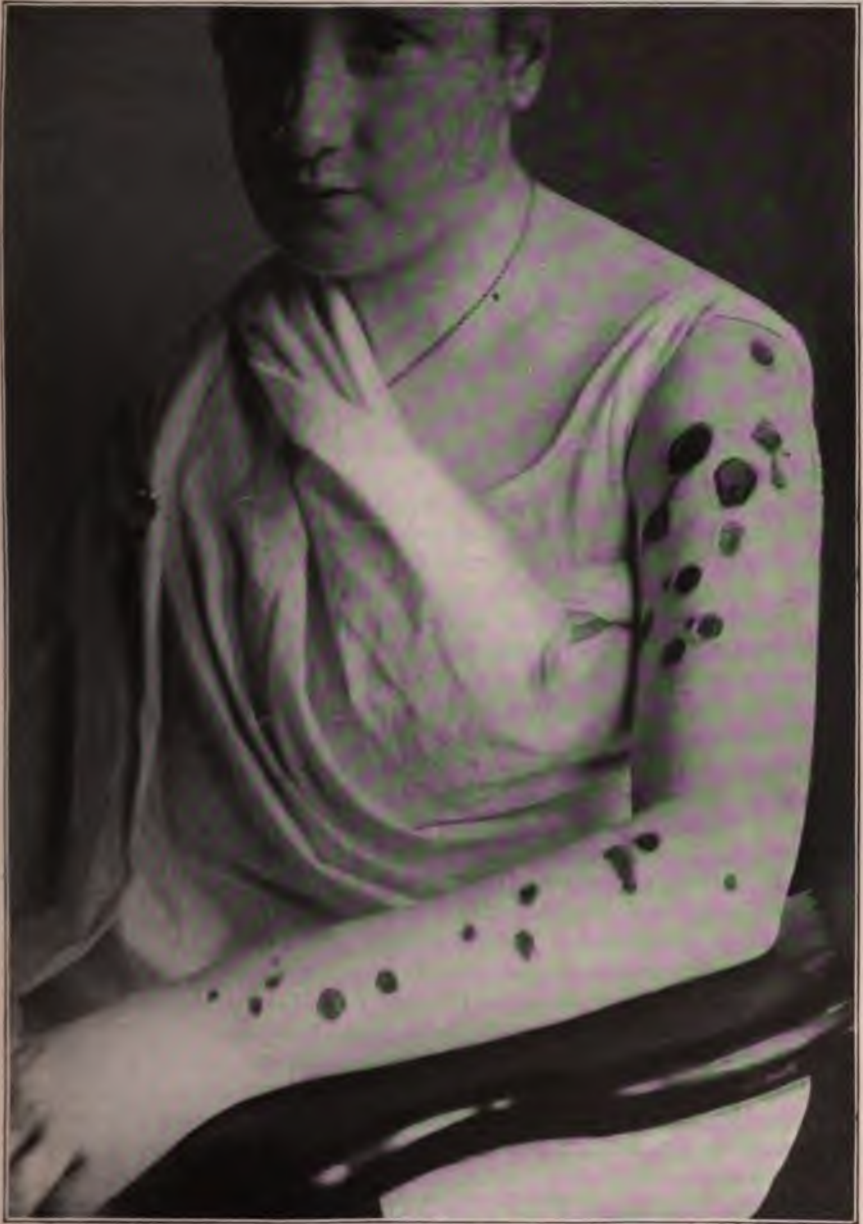
[1] Brit. Jour. Derm., 1895, vii, pp. 9-16.

[2] Ormsby, Jour. Amer. Med. Assoc., 1915, lxv, p. 1622; "Factitious Dermatoses;" a report of 34 cases, discussion of the subject and bibliography; Gaskill, New York Med. Jour., January 22, 1916, Dermatitis Factitia, 5 cases; Simpson, Jour. Cut. Dis., 1917, xxxv, p. 493; "Dermatitis Factitia and Neurotic Gangrene."

FIG. 70



Dermatitis factitia.

if applied for a few moments only will produce in most individuals an erythema or superficial dermatitis of a few days' duration. If the solution be stronger, or if a weaker solution be allowed to remain in contact with the skin for a longer time, more severe forms of inflammation and even gangrene may result. The lesions are always sharply outlined, and of unusual, often fantastic, shapes. They appear suddenly, at irregular intervals, usually one or two at a time, and run a fairly rapid course. When fluid caustic is used it frequently happens that one or more drops run down the skin from the point of contact, leaving a characteristic streak which is usually lighter in color and shows less inflammation than the patch from which it depends. When the caustic is applied with a needle or pin, as is frequently the case in gangrenous lesions, the border shows a finely jagged or serrated edge made by the numerous punctures. When gangrene is present it is usually superficial and separated from the normal skin by a narrow vivid red line. In certain cases under our observation the gangrenous process has been deep and has presented the usual appearance of other gangrenous processes of this grade. Occasionally the finger nails or some article of clothing will be stained by the agent employed.

Subjective sensations, usually pain and burning, may be greatly exaggerated by the patient, who complains bitterly of the distress caused by the simplest and lightest dressings. On the other hand, in severe cases the change of dressings is borne with no complaint whatever, the patient apparently being pleased to demonstrate his ability to endure pain. Patients sometimes predict the appearance of new lesions, claiming that in the region where they are to appear varying sensations are experienced.

Etiology.—The cause of these peculiar actions on the part of an individual must be situated in derangement of the nervous system. All the intricate processes concerned in hysteria could be brought forth to explain the condition here. In some cases the patient exhibits a desire to escape from disagreeable duties or surroundings. In others there is the wish to gain attention, sympathy, interest or pity, or to achieve notoriety. In certain instances no explanation can be given, and it may be, as suggested by Pernet and Towle, that in a certain proportion of cases there is a dual personality, or subconscious state, in which the lesions are produced, while in the normal condition the patient has no recollection of the acts then performed.

The disorder occurs, as a rule, in women and girls of a highly neurotic temperament, showing distinct stigmata of hysteria. In many cases a trifling injury occurred, the dressing of which suggested to the patient the production of the lesions and the necessary means of carrying out the deception. A long list of animal, vegetable and mineral substances are used in producing the lesions. Those most frequently used are phenol, croton oil, cantharides, mustard, various acids and alkalies, lye, cresol and compound solution of cresol. Other methods used have consisted in burning with matches, pieces of hot metal or bottles containing hot water. Friction with the fingers, sandpaper, and other rough substances is of common occurrence. An exceptional case is reported where lesions were produced by briskly rubbing tobacco into the skin of the genital region.

Diagnosis.—The diagnosis is usually not difficult to one familiar with cutaneous disorders since the lesions, though of a simple inflammatory nature, are different from those of any recognized disorder. The important points are the occurrence of the lesions within reach of the patient's hand in the regions above suggested; their sudden appearance, usually during the night when the patient is alone; the irregular occurrence and rapid course; the peculiar linear arrangement of the lesions and, when fluid is used, the characteristic streak extending from the main patch. Further aids in the diagnosis may be found in the general characteristics of these patients, the unusual history of the disorder, the discovery of anesthetic areas—especially in the fauces and conjunctiva, and other evidences of hysteria. Fixed dressings which cannot be removed without detection are frequently important. In serious cases, continual observation, day and night, in a hospital will clear up the diagnosis. The cunning of these individuals is frequently surprising.

Treatment.—Upon recognition of the disorder and the admission of the patient as to its cause, with cessation of the practice, immediate recovery takes place. The specific treatment depends upon the degree of dermatitis present and the remedial agents recommended for such should be used.

Prognosis.—The major portion of these patients recover without difficulty when convinced that their deception has been detected. In a few instances the practice is continued in spite of the facts being known, but in these it appears that a serious derangement of the nervous system is present.

Neurotic Excoriations.[1]—**Synonyms.**—Acne urticata (Kaposi); Dug-out excoriations (Colcott Fox).

Definition.—A cutaneous disease in which lesions, often leading to marked scar formation, are produced by the patient.

Colcott Fox, Adamson and others have reported examples of the disorder in England, and recently Pusey and MacKee have recorded a series of cases in America.

Symptoms.—Lesions are seen chiefly on the face and neck, limbs and chest, though they may occur on any portion of the cutaneous surface readily accessible to the hands. The lesions are oval or irregular excoriations and ulcers, sometimes crust-covered, pigmentations and scars. The most characteristic symptom is the uncontrollable desire of the individual to remove some supposed foreign substance in the skin, or to obtain relief from irritation, itching,burning or other anomalous sensations, by rubbing, pricking, or digging with the finger nail, forceps, point of a knife, or scissors, or other instrument. The injured area may have been the site of a lesion of some sort, or may have merely presented itching or other sensation. Adamson places the sequence of events in some cases as follows: Itching in a localized spot. This is rubbed or otherwise irritated, producing a wheal which in turn itches intensely, and is then excoriated by the finger nail.

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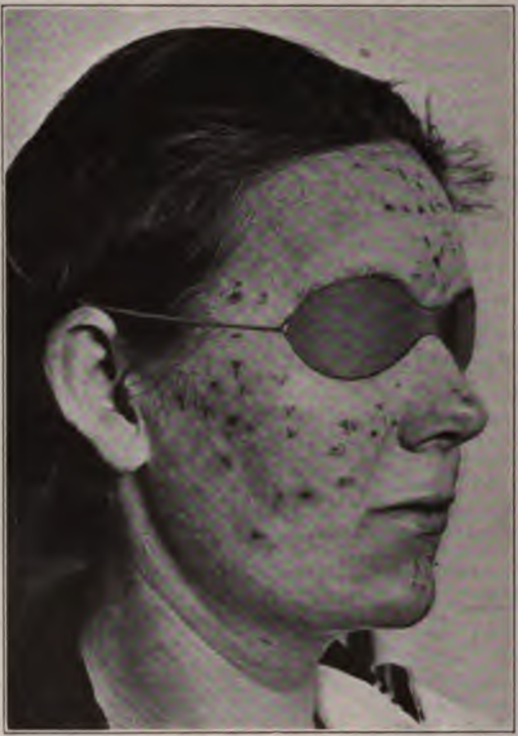
[1] Adamson, Brit. Jour. Derm., 1915, xxvi, p. 1, "Acne Urticata and Other Forms of Neurotic Excoriations;" Cf. this article for description of various types, report of cases and classification of those previously recorded, with bibliography; Pusey and Senear, Archives of Dermatology and Syphilology, March, 1920, n. s. l, p. 270; MacKee, Ibid., p. 256. These papers were read before the American Dermatological Association and the Section on Dermatology of the Amer. Med. Assoc., in 1919. They include a report of cases and complete discussion of classification, symptoms and diagnosis.

Fig. 71



Neurotic excoriations.

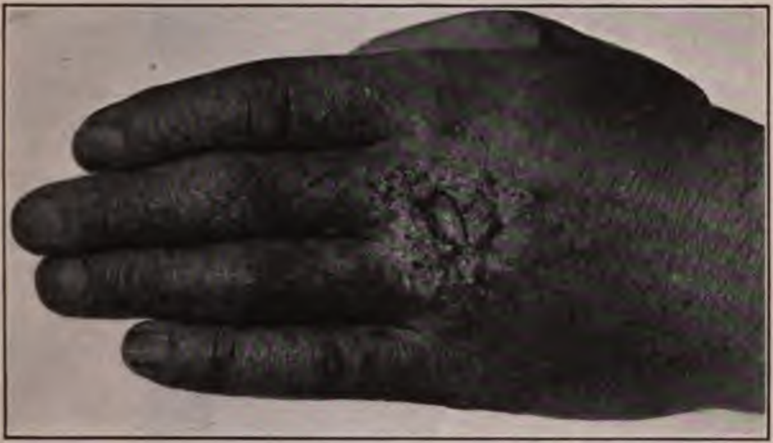
Etiology.—Women are more frequently the subjects of the disorder than men, the major portion being thirty years of age or older. Many were younger and one patient was only thirteen years old. The cause appears to be a disturbance of the nervous system. In some cases the resemblance to acarophobia is marked. This is illustrated in those who believe they can dig out parasites and thus relieve the disorder. In others there simply exists an irresistible desire to remove some foreign substance other than parasites, while still others rub, pinch, squeeze, and dig the lesions in the hope of obtaining relief from the subjective sensations present.

Diagnosis.—The ulcers and inflammatory crusted lesions and scars produced may simulate syphilis, epithelioma, tuberculosis and other scar-leaving dermatoses. The artificial appearance of the lesions, which differ from the true lesions which they simulate, is usually at once detected. Feigned eruptions are easily differentiated as the patients suffering with the disorder under consideration readily admit the fact that they have injured themselves as described above, whereas in feigned eruptions deception is always practised. The resemblance of this disorder to Crocker's acne keratosa is close (Cf. this section).

Treatment.—The coöperation of the patient is necessary if treatment is to be successful. If this can be secured and the patient has the power to cease his manipulations, recovery ensues. Local soothing and antipruritic lotions and ointments are indicated, and internal administration of tonics is of some service, particularly the use of arsenic.

Prognosis.—The outcome is problematical. Some individuals continue the practice of self-injury in spite of the facts being understood. The outcome depends upon the possibility of restoration of the nervous system to its normal condition.

FIG. 72



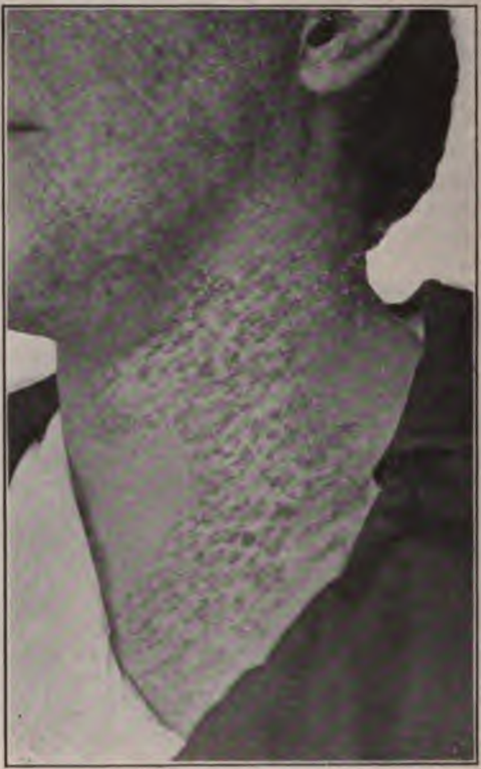
Roentgen-ray dermatitis with ulceration.

Radiodermatitis.—**Synonyms.**—*X-ray Dermatitis.*

Definition.—An acute or chronic inflammatory condition of the skin induced by the application of x-rays.

Symptoms.—A period of time varying from a few days to three months or more elapses between exposure to x-rays and the appearance of symptoms. The earliest evidence manifested is either pigmentation or erythema. The former may be lentiginous or exhibited as a diffuse, brownish discoloration of different shades, the amount of pigment varying as a rule with the complexion of the patient. Usually this disappears within a few days or weeks, though it may persist for several months. Erythema appears early and soon subsides, with superficial desquamation and pigmentation. The process usually lasts from a few days to two weeks, and is accompanied by mild itching or pricking sensations. Should the inflammatory process progress to a further stage, vesicles appear on the erythematous area. These may be either superficial and short-lived, soon drying and disappearing, or more deeply situated, and associated with greater swelling and increased redness, the whole area becoming denuded of its superficial epithelium,

FIG. 73



Roentgen-ray telangiectasia.

and showing an excoriated and weeping surface. This surface usually becomes covered with a yellowish or grayish, adherent pellicle, composed of necrotic epithelium, which gradually retracts, its place being taken by normal cornified cells. In case the pellicle does not form, bluish islands of epithelium appear over the weeping surface, which by enlargement and coalescence cover the area. The new epithelium is smooth, delicate, bluish-white in color, devoid of pigment and hair, and may remain sensitive to external influences for some time. The duration of this degree of dermatitis is from a few weeks to several months, and the subjective sensations vary; usually a burning, tingling, or itching sensation is experienced, with occasionally marked tenderness and some pain. In a dermatitis of serious portent, the subcutaneous and deeper tissue is involved. The inflammation begins with erythema, vesiculation, and marked swelling; the skin becomes cyanotic and brawny, and necrosis follows. The affected area is covered with a dry, dark-colored, leathery, adherent mass of tissue, which may persist for months, is surrounded by a reddish, inflammatory border, and is accompanied by severe pain. These lesions are chronic, lasting for months or years, and the cicatrix which eventually forms may be covered with telangiectases.

A chronic form of dermatitis occurs in the hands and sometimes on the face of x-ray operators, which is attended by scaling, atrophy, obliteration of the normal lines of the skin, telangiectases, alopecia, and at times loss of the nails. Ulcers and hyperkeratoses, some of which develop later into epithelioma, have occurred, and occasionally a condition simulating scleroderma has been noted.[1]

Pathology.—The pathological action of x-rays has been studied both in man and in animals by several observers. Schlotz[2] concludes that: First, the rays cause a slow degeneration of the elements of the skin, in which the cells, not only of the epidermis and its appendages, but also those of the corium, may participate. This degeneration affects the nucleus as well as the protoplasm of the cell. The rays also induce, but to a much less extent, a degeneration of the fibrous elements (collagen, elastin) and of the muscles. Second, when the cellular degeneration reaches a certain point an inflammatory reaction occurs in which the bloodvessels become dilated and an extravastion of serum and leukocytes results. The latter then seem to act as phagocytes and to destroy completely the degenerated cells. MacLeod[3] adds that "the inflammatory reaction induced by x-rays is peculiar in that it occurs in a tissue, the vitality of whose various elements has already been impaired by the action of the rays, and in that it is associated with greater destructive changes than those produced by actinic rays, and is apt to lead to ulceration and necrosis, and is liable to be followed by an imperfect process of repair."

Treatment.—The simpler forms of dermatitis due to x-rays may often be treated successfully with the measures recommended for corresponding phases of eczema and dermatitis due to other external causes. Frequently, however, even a mild dermatitis due to x-rays is persistent and exceedingly painful, and not infrequently is aggravated rather than relieved by measures applicable to corresponding grades of dermatitis from other causes. In such cases various applications, with or without some local anodyne, may be tried. Among those we have found the most useful are the following: The lead and opium wash, with or without the addition of a powder, glycerin, or boric acid, as recommended for the treatment of acute eczema; a mixture of equal parts of this lotion and carron oil (made with olive oil); compound stearate of zinc powder; a simple ointment containing one or two drams of orthoform to the ounce. We have found the following paste, recommended by Engman[1] very satisfactory:

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[1] Jour. Cut. Dis., 1903, xxi, p. 52.

[2] Archiv, 1902, lix, pp. 87 and 241; abstr. Brit. Jour. Derm., 1902, xiv, p. 397.

[3] Brit. Jour. Derm., 1903, xv, p. 365, with review of literature on pathological action of x-rays.

Boric acid 12 drams (48.); zinc oxid, starch, bismuth subnitrate, and olive oil, of each 1 ounce (30.); lime water and lanolin, of each 3 ounces (90.); rose water 12 drams (48.). The powder should be well rubbed up in a mortar and the lanolin added; the olive oil and liquor calcis then are mixed and slowly added. When this is mixed thoroughly the rose water is added, and the whole beaten up in the mortar into a light, creamy paste.

The surface should be kept covered with this paste, spread on old linen or on several thicknesses of gauze. A sheet of gutta-percha tissue may be placed over the dressing to prevent evaporation, unless this is uncomfortable, as it sometimes is, to the patient.

In deep-seated ulcers, which fortunately are seen but rarely, the treatment is usually surgical. The necrosed tissue should be removed and the surface covered with skin-grafts.

PURPURA ANNULARIS TELANGIECTODES.[2]

Synonym.—Telangiectasia follicularis annulata (Majocchi).

This rare disease was first described by Majocchi in 1896. About thirty cases have been recorded in Italy, France, and Germany. The first case studied in America was recorded by MacKee.[3]

Definition.—The disease is characterized by telangiectatic, purpuric, and atrophic lesions, usually occurring on the lower extremities. The symptom complex appears sufficiently characteristic to warrant its recognition as an entity.

Symptoms.—According to MacKee's résumé of the recorded cases, the symptoms of the disease may be divided into three fairly well-defined stages, as follows: telangiectatic, purpuric and pigmentary, and atrophic. The early lesions are minute red puncta. These slowly increase in size by peripheral extension, producing lesions from split-pea- to dime-sized and larger. The color may be removed from the early lesions by pressure, but later, in the purpuric stage, the color is permanent. As the lesions spread peripherally, they clear in the center, producing in some cases slight atrophy. After several months, the lesions disappear, leaving pigmentation. While the eruption is usually situated below the knees, on the anterior and lateral surfaces of the limb, at certain times lesions may be found above the knees, on the arms, and rarely in other situations. In occasional instances, the eruption is preceded by some constitutional disturbance, indicated by neuralgia or rheumatic pains.

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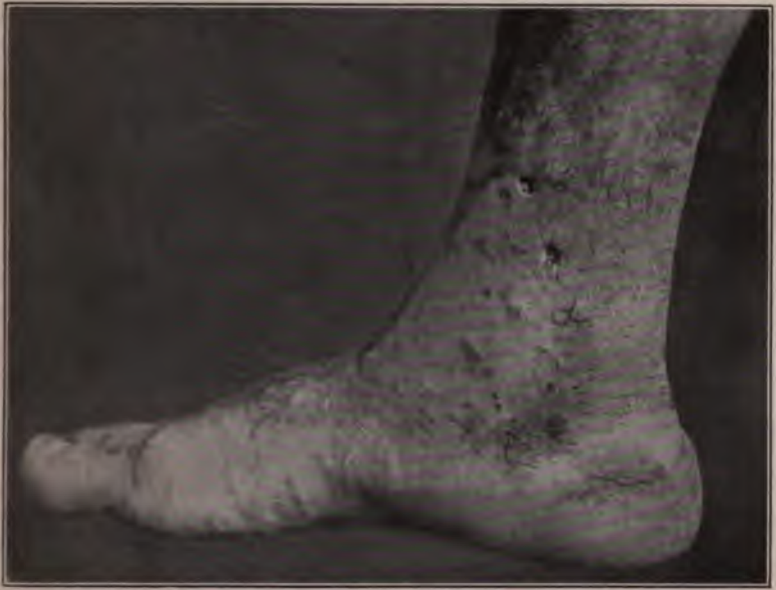
[1] Brit. Jour. Derm., 1903, xv, p. 390.

[2] Majocchi, Archiv, 1898, xliii, p. 447 (report of three cases not hitherto described, with histological study); Brandweiner, Monatshefte, 1906, xliii, p. 529 (three cases reported, with histological study in one); Rodoeli, Giorn. ital., 1911. ii (abstr. Archiv, 1911. cx, p. 320); Lipschütz, Archiv, 1912, cxii, p. 1017; Brandweiner, Derm. Wochenschr., 1912, lv, p. 1292 (two additional cases reported, with bibliography); Vignolo-Lutati, Archiv, 1912, cxiv, p. 303 (case report, with histological study); Lindenheim. Archiv, 1913, cxiii, p. 689 (case report, with histological study); Pasini, Giorn. ital., March, 1913 (abstr. Archiv, 1913, cxv, p. 1051; a clinical and histological study).

[3] MacKee, Jour. Cut. Dis., 1915, xxxiii, pp. 129, 186 and 280. "Purpura Annularis Telangiectodes;" paper read before the Amer. Derm. Assoc., at its thirty-eighth annual meeting, May, 1914, Chicago (a clinical and histological study, with a description of the disease as recorded in the literature. To this article the author is much indebted). Ibid., 1917, xxxv, p. 705, case presentation. Histology typical.

According to Majocchi, it should have the following characteristics: (1) rose-colored or livid spots formed from capillary dilatation, followed by hemorrhage, without previous hyperemia or any infiltration of the skin, and usually in distinct connection with the hair-follicle; (2) slow development and increase in size; (3) constant eccentric growth of the spots, resulting in definite ring forms; (4) symmetrical arrangement; (5) the primary location always on the extremities, usually the lower; (6) the usual absence of subjective symptoms; and (7) commonly leading to slight atrophy and achromia of the skin, together with alopecia. Instead of rings, the lesions may form half-circles, as described by Lipschütz. The course of the disease is slow, but with a tendency to clear up after several months. Brandweiner states that the lesions disappear spontaneously after a few months' duration without scar-formation, and according to this author's obser-

FIG. 74



Purpura annularis telangiectodes, showing annular lesions. Some of the margins depict purpuric spots and others show necrosis. (MacKee.)

vations new lesions not infrequently occur through trauma induced by scratching.

Etiology.—The disease usually attacks young male adults. Majocchi states that the etiology is obscure, but that the disease is probably a vasomotor phenomenon, having some resemblance to erythema pernio. Most authors believe the disease to be either of toxic origin or a neurosis. While usually occurring in adults, it has been seen in children.

Pathology.—Histologically, the capillaries are found to be dilated, and there occurs a perivascular cellular infiltration. In certain cases diapedesis of red-blood corpuscles occurs, and hemorrhagic areas are noted, with deposits of pigment. The lymph-spaces are also dilated, and there appears to be an increase in the number of small capillaries. Phlebitis and obliterating endarteritis are noted. In the final stage, the epidermis becomes atrophic and the papillæ are obliterated. Rodoeli suggested a connection between the disease and tuberculosis. Some cases have given a positive tuberculin reaction, but inoculation experiments have been negative. Most authors agree that there is no connection between the two diseases. MacKee found in an early lesion endarteritis, with increase in the number of small capillaries, edema in the corium, and hyalin degeneration of the arterial walls. In some places aneurysmal sacculation was noted. Hemorrhagic areas also occurred, and in addition a moderate amount of perivascular cellular infiltration, the cells being small lymphocytes. In a chronic lesion the epidermis was atrophic; the corium was edematous and contained a few vessels; there was endarteritis, with partial obliteration of the lumina; and hemorrhagic and pigmented areas occurred throughout the corium, with some perivascular cellular infiltration. The appendages were normal.

Diagnosis.—The disease is to be distinguished from syphilis and from ordinary purpura, but by noting the classical symptoms as described above little difficulty should be experienced.

Treatment.—As the cases tend to clear up of themselves, little treatment is necessary. Rest in bed, with elevation of the limbs, is recommended. In case this cannot be carried out, suitable bandaging would be of value. In rare instances, where necrosis occurs, as in MacKee's case, these areas should be treated with mild antiseptic lotions or ointments.

Prognosis.—As indicated above, the prognosis is good, always remembering the possibility of recurrence.

A PECULIAR PROGRESSIVE PIGMENTARY DISEASE OF THE SKIN.

Synonym.—Schamberg's disease.

An affection was described under the above title by Schamberg[1] in 1900. A number of similar cases have since been recorded.

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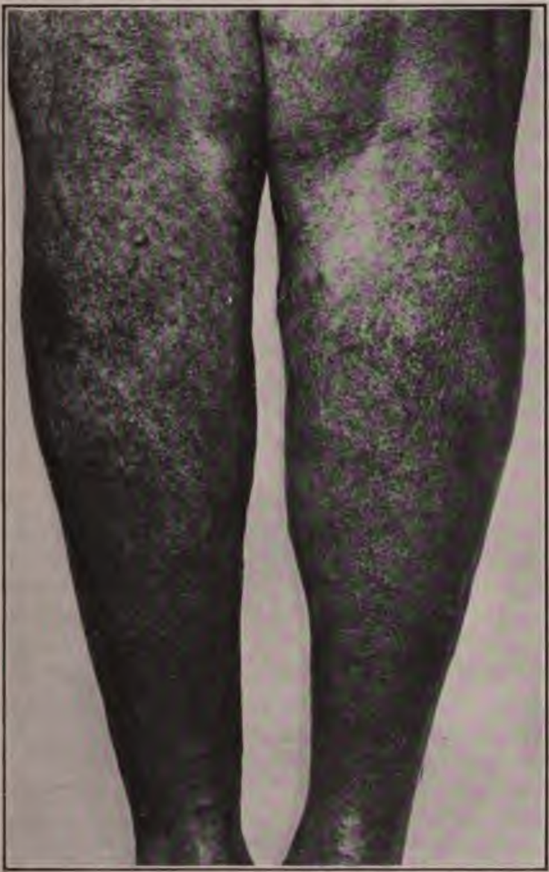
[1] Trans. Amer. Derm. Assoc., 1900; and idem, Brit. Jour. Derm., 1901, xiii, p. 1.

e disorder progresses slowly and occurs in the form of irregular tches. In Schamberg's first case the patches are described as lows: They are sharply defined, of a reddish-brown color, vary in e, and at the border are visible a number of small outlying macules the same color and of the size of pins' heads. The borders of other tches are made up of pinpoint- to pinhead-sized, reddish puncta, sely resembling grains of cayenne pepper, though darker in tint.

FIG. 75



Schamberg's disease.

me present a somewhat telangiectatic appearance. As the lesions dergo involution, a slight, diffuse, brownish-yellow staining is left. ere are no subjective sensations.

The disorder began over the shins and spread over the ankles and rsum of the feet. There were also lesions about the knees and on e flexor aspects of the wrists.

The disease apparently begins as pinhead-sized, reddish puncta or dots, forming irregular patches, which slowly extend by the formation of new lesions upon the periphery. The puncta in time disappear, leaving behind a brownish, brownish-yellow, or reddish-brown pigmentation, which slowly fades (Schamberg).

A *histological* examination reveals a subacute inflammatory process. The most marked changes are noted in the papillary and subpapillary layers, where there occurs a dense cellular infiltration, composed of lymphoid cells and polymorphonuclear leukocytes, with a few epithelioid cells. In addition, there are stellate, fusiform connective-tissue cells and a few mast-cells. Dilated bloodvessels and lymph spaces are present. Pigment-cells and granules are absent as a rule. In a recent histological study Kingery[1] demonstrated pigmentary deposits. Infiltration occurs particularly about the sweat-ducts, in some places sufficient to produce obstruction.

The disease is to be differentiated from purpura and angioma serpiginosum.

ECZEMA.

Synonyms.—Fr., *Eczêma;* Ger., *Ekzem.*

Definition.—Eczema is an acute, subacute or chronic inflammation of the skin characterized by erythema or by the appearance of isolated or grouped papules, vesicles, or pustules, occurring in uniform, multiform, or modified types upon a reddened infiltrated base, accompanied by itching or burning sensations and resulting in weeping and crusting, in infiltration and scaling caused by varied and complex internal and local factors.

Eczema is decidedly a protean disease. It is not only protean in its clinical manifestations, but its causes are varied, numerous, and usually complex. Although a dozen successive cases of eczema may present wholly different clinical pictures, nevertheless all of them have some characteristics in common and the diagnosis in most cases is not difficult.

A vexed and unsettled question among dermatologists is the relation of eczema to other forms of dermatitis.[2] In its historical evolution many separate and distinct diseases have been grouped under this title. The present tendency is to exclude as rapidly as possible all forms of dermatitis which have a definite cause, and which upon the removal of this cause, disappear. Eczema is a dermatitis and it is not possible to say in every case which title is the more appropriate. A convenient division, which is followed in these pages, classes under dermatitis those forms of involvement of the skin which result from recognized external causes and which subside upon removal of the cause.

Symptoms.—Eczema is characterized by heat, redness, itching or burning sensations, infiltration of the skin, weeping or moisture at some stage and multiform lesions. Redness varying in shade from the bright red of the acute to the dull red of the chronic forms is present in all cases. Itching is practically always present and may vary from slight annoyance to intolerable distress. It is commonly intermittent or paroxysmal in character and is usually worse at night. In some instances, especially in acute and erythematous types, the sensation of burning or smarting may be more marked than that of itching. Occasionally the skin is hyperesthetic and hypersensitive to contact with even the blandest substances. The degree and character of the subjective sensations in eczema depend largely upon the location, type and severity of the disease, but also, to some extent, upon the general condition or peculiarities of the individual. In acute eczema there is often some edematous swelling and always slight infiltration of the skin. In the chronic forms the infiltration and thickening of the skin are more pronounced and may be excessive. The serous discharge, which is present during at least part of the course of most cases of eczema, is characteristic and stiffens articles of clothing on which it dries. It commonly escapes from a denuded surface or from minute excoriated points which represent abortive or ruptured vesicles.

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[1] Jour. Cut. Dis., 1918, xxxvi, p. 166.

[2] For a discussion of this subject see recent papers by Heimann, Jour. Cut. Dis., 1916, xxxiv, p. 259; and Trans. Sec. on Derm., Amer. Med. Assoc., 1916, p. 242, and Knowles, ibid., p. 279.

Aside from some cases of erythematous and papular eczema which may persist throughout without change of type, eczema is notably a polymorphic disease, presenting in irregular succession, or in varying combinations, erythema, papules, vesicles, pustules, crusts, scales, fissures, excoriations, or denuded and oozing surfaces.

The course of the disease is capricious. Not only does the severity of the process change frequently and rapidly, but often the type of lesion as well. This is conspicuous in children and in others having delicate skin and in those cases in which the affected areas are not protected from atmospheric and other external influences. Frequent variations in severity with or without modification or change of type are not uncommon. Apparent recovery is frequently followed by a relapse which may develop fully within a few hours without apparent cause.

Furthermore, as to course, eczema may be divided into acute and chronic types, the latter being the rule, but modified from time to time by acute exacerbations or marked increase in the intensity of the symptoms. As a rule, eczema does not undergo spontaneous recovery but tends, rather, to remain indefinitely and to extend either by involving contiguous surfaces or by developing in new areas. It may attack any part of the cutaneous surface and the manifestations vary in different anatomical situations. The flexor surfaces, the hands and face are points of predilection.

The disease is commonly more or less local, appearing in one or several irregular and usually ill-defined areas, but it may be general or even universal. No constitutional symptoms accompany the disorder except in instances of acute eczema of severe grade and wide distribution.

Clinically several types of eczema can be recognized. In the major portion of cases mixed types occur or are seen in succession. Though several forms of eczema frequently coexist, it is usual for one type to predominate either throughout the course of the disease or for certain periods. For purposes of description it is usual to divide eczema into primary and consecutive types.

Eczema Erythematosum is most common on the face, especially in an individual exposed to wind and weather, the rays of the sun or to direct heat, but it may occur on any part of the body and is frequently seen on the palms or soles, the genital region and in areas where two cutaneous surfaces come in contact. It begins usually as a diffuse, ill-defined area of redness; less frequently as localized, coin-sized or larger macules or erythematous patches which may coalesce or remain more or less distinct. Swelling and infiltration are present in varying degrees. In acute cases the edema may be excessive, sometimes closing the eyes. In the subacute and chronic forms, which are the more common, there is less swelling and more infiltration and thickening of the skin. The sensation of itching which is characteristic of most forms of eczema is usually excessive but at times it may be largely or wholly replaced by one of heat or burning. This occurs particularly in the acute types. The color varies from a bright to a dull or purplish red, depending upon the severity of the disease, its location and individual peculiarities. In middle-aged adults, in whom the disease is common, the normal hue of the integument is darker than in early life and the color of the part is often noted to be of a dull crimson shade. At times the redness is irregularly distributed, producing a mottled appearance, having different shades over different areas. Occasionally on the face a yellowish tinge is seen, which is probably induced by the sebaceous glands abundant in this region. Scaling occurs over the surface after a few days and persists to the end of the disease. In pure types of cases there is no discharge, but it is not uncommon for the type to change to the moist form. When the disease occurs on apposed surfaces, as in the axilla, under the breasts, or on the interdigital surface of the feet, or about the genitals, the superficial epidermis may be destroyed by maceration and friction and leave a denuded, oozing surface. Occasionally oval or round, fairly well circumscribed, erythematous, scaling patches occur to which the title **eczema orbiculare** has been given.

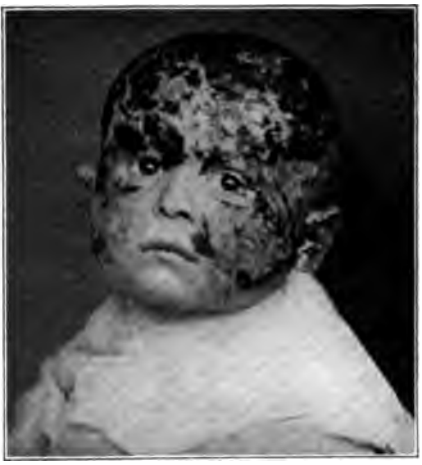
The disease may pursue an acute course, terminating in exfoliation and gradual resolution, or changing to papular, vesicular, pustular, or mixed types. More frequently, the process persists and becomes chronic. The skin then becomes more infiltrated and thickened and may present numerous folds produced by the exaggeration of the normal folds of the skin. This may be a conspicuous and deforming feature. The area involved is frequently better defined than in other forms, except the papular, and though the condition may remain limited to its original site for months or years, it has a tendency to extend either contiguously or by the formation of new areas. The intensity of a process may change very rapidly. It is usually aggravated by exposure to heat, cold, wind, or other external irritants.

Scratch lines are rarely seen, but instead minute, superficial, denuded spots may be observed. Like other varieties of eczema, this form is extremely liable to recrudescence and relapse. In advanced life the disease may continue over a period of years.

Eczema Papulosum.—Under this title are classed the forms which have been described as lichen simplex, lichen eczematodes and eczema lichenoides. The papules are acuminate, pinhead-sized or larger, colored in various shades of red, and are usually situated upon a reddened and infiltrated base. They are generally discrete, though often set closely together, are accompanied by intense itching, and of all eczematous lesions are most likely to be irritated by scratching. The summits of the papules are torn, often to such an extent as to produce bleeding, the blood drying in minute crusts on the apices of the individual lesion. The papules may persist for weeks, or disappear to be replaced by others. They may coalesce completely to form irregular, thickened, elevated, pea-sized or larger patches covered with scales. The involved areas are usually well defined in outline. The extent of the affected surface varies, the disease being in some cases largely diffused over several portions of the body, but it is commonly limited to single patches from coin to palm size. Such patches may continue for long periods of time, or, being at one time relieved, may recur. Papular eczema is a dry manifestation of the disease and is more common in adult life, or in advanced years. The papules here described often exhibit pinpoint-sized vesicular summits, which may develop into minute or larger pustules. A patch of papular eczema, where no vesiculation or pustulation has been observed will, if sufficiently scratched, ooze with moisture, allowing the serum to escape from the abraded surface. There are, in fact, few scratched eczematous surfaces which will not moisten a handkerchief applied to the part. A follicular type was described by Morris, under the title **eczema folliculorum.** In this type each inflamed follicle projects from the surface in the form of reddened papules, about which the skin becomes hyperemic. As the process spread centrifugally by the involvement of adjacent follicles, the center undergoes involution with desquamation and a gradual change in color from red to yellow. This condition is found most frequently on the extensor surfaces of the legs and arms in multiple scattered patches. The condition is obstinate, usually recurrent, and may be accompanied by an intense itching. As resolution of papular eczema occurs the papules flatten and the patches become less infiltrated and finally disappear.

Eczema Vesiculosum.—The clinical features of vesicular eczema are chiefly due to the acuity of the inflammatory process present and to the consequent free exudation of the serum of the blood from the dilated vessels of the corium. The involved surface usually feels at the outset hot, itchy, or unusually sensitive. It soon after becomes intensely reddened, as a result of the hyperemia and consequent exudation, which may last for one or several hours. Upon this base poppy-seed to grape-seed sized vesicles become visible. The lesions may be closely packed together, or discrete, or may be abundant and coalesce. Each vesicle is filled with a droplet of clear serum and is located superficially in the epidermis. The vesicle is readily ruptured or opens spontaneously, and its limpid contents are then poured out upon the surface of the integument. The quantity of the fluid thus exuded is in excess of that contained in the small vesicles, owing to the fact that the excoriated, macerated, and broken epidermis no longer confines the outflow of serum from the engorged vessel beneath. Minute and even large drops of clear fluid of syrupy consistency can be seen collecting at the points where a solution of continuity has occurred. When a droplet is removed its place is immediately taken by a second. Crops of new vesicles succeed the first, each being followed by the train of symptoms described. The vesicles are usually short lived and have often disappeared before the patient has been seen by a physician. In some instances the destruction of the epidermis by rubbing or scratching, or by an abundant and rapidly formed exudate allows the escape of the fluid without previous vesicle formation. The discharge dries rapidly when exposed to the air, forming light yellowish crusts.

FIG. 76

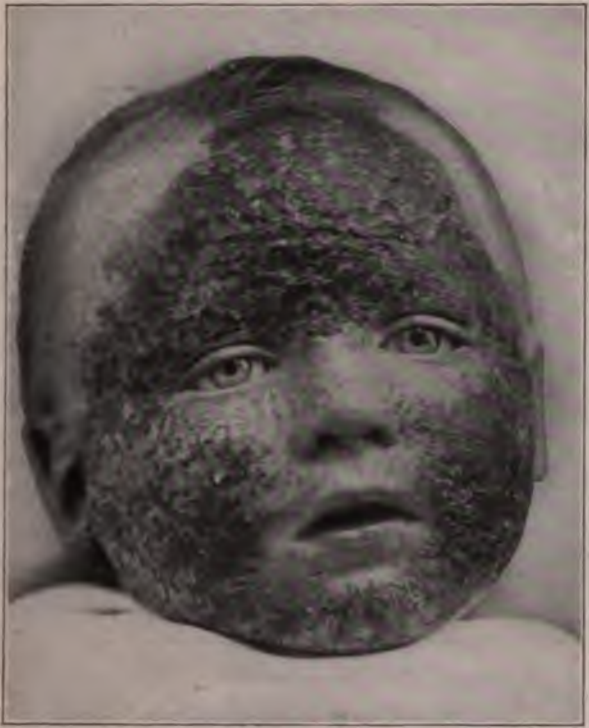


Eczema pustulosum (infantile).

The contour of the affected patch or patches is seldom well defined, the diseased portion imperceptibly shading into the normal skin. The color of the area varies according to the stage of the process, being of a vivid red early, later yellowish-red when covered with crusts or scales during the process of involution. Edema and infiltration of the skin are characteristic and when a portion of the affected integument is pinched up between the finger and thumb it is found to be thicker and less elastic than normal. This form of eczema may persist or recur in a single small area, or it may become diffused or even generalized. It appears commonly on the flexor and other surfaces where the skin is thin.

The subjective symptoms of vesicular eczema are more or less intense itching and burning. In very acute forms there is considerable tenderness. Exceptional cases, more frequently observed in children, are accompanied by mild febrile symptoms. As resolution occurs all the symptoms described above gradually decline in severity; the serous discharge diminishes, the redness fades, the limits of the involved areas become less distinct, the crusts loosen and fall, and beneath the scales which have taken the place of the oozing epidermis, a new and tender epithelial covering is produced. As a rule, for weeks after the process has completely ceased the newly formed epidermis is tender and has a slightly reddened appearance, although resolution is followed by no permanent sequels. Instead of undergoing resolutions the condition may terminate in one of the consecutive forms, eczema rubrum, or eczema squamosum, or be transformed into eczema pustulosum.

FIG. 77

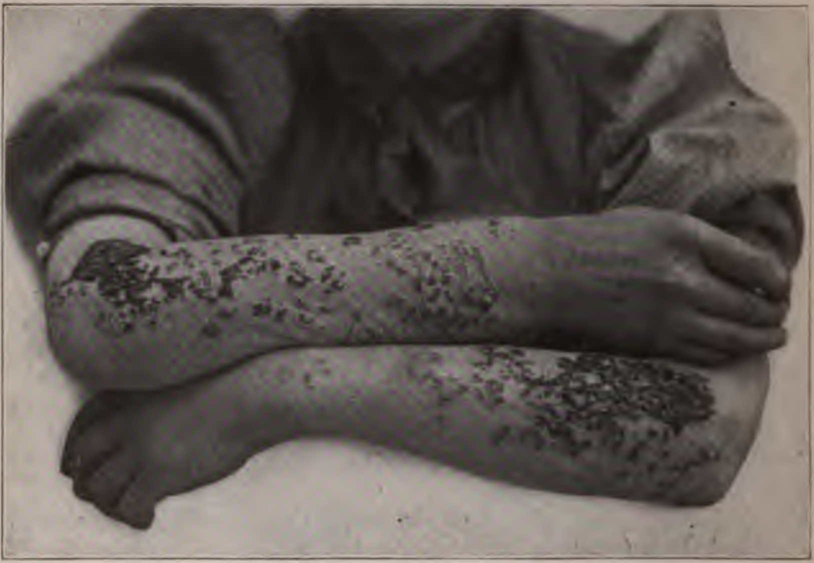


Eczema impetiginosum. (Fox.)

Eczema Pustulosum.—(*Eczema impetiginoides: Impetigo eczematodes*).—This type may originate in one of the other forms of eczema as a result of secondary pus infection, or pustular lesions may rapidly form at the outset. Usually, there is first seen a crop of minute vesicles which enlarge and become extended with puriform contents.

These pustules rupture either spontaneously or through trauma and the fluid with which they are distended dries into yellowish-green or darker colored friable crusts. In aggravated cases the purulent matter forms directly upon the involved surface. Itching and infiltration are characteristic, the former being less severe than in erythematous and papular types. Pustular eczema is more frequently encountered on the head and face, but also occurs at times on the limbs, particularly about the hair follicles (*eczema sycosiforme*). It attacks those who are debilitated or whose resistance to the invasion of pus-cocci is lowered. When existing on the face or scalp, the altered secretion from the sebaceous glands is added to the exudate, producing, when dry, yellowish-green or darker colored crusts which later fall, leaving a reddened and tender new epidermis beneath.

FIG. 78



Eczema pustulosum. (Fox.)

Many examples of pustular eczema belong to a group differentiated by Engman[1] and emphasized by Fordyce[2] designated as *dermatitis infectiosa eczematoides* (Cf. this section).

The four primary types above considered are sometimes encountered in practice as distinct and unmingled forms of cutaneous disease, some of them more commonly than others. It must be understood, however, that the several forms which are presented here as distinct types and which are useful in the analytic study of the disease often become, in actual observation, inextricably commingled. Other types consecutive to the above are met with.

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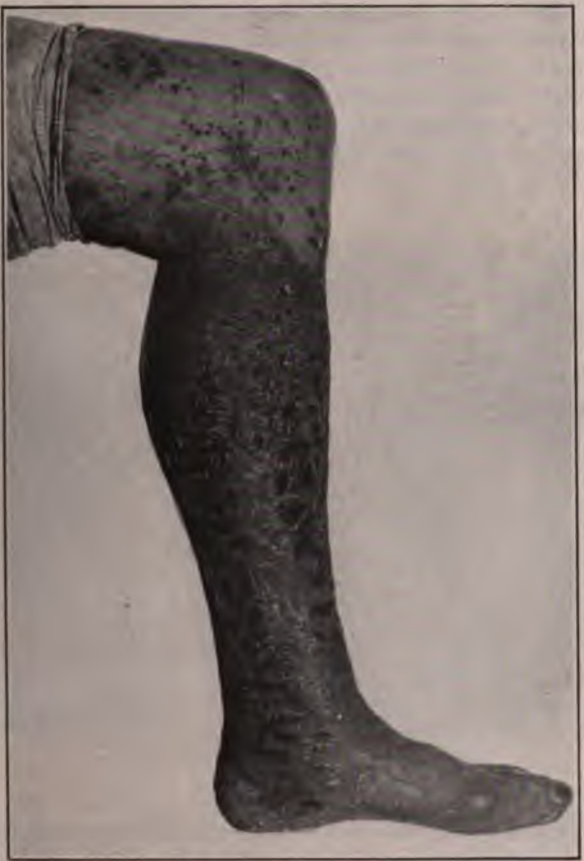
[1] Amer. Med., 1902–03, iv, p. 760.
[2] Jour. Cut. Dis., 1911, xxix, p. 129.

Eczema Rubrum.—This name has been given to the reddened, angry form of the disease, which because of the free exudation of the serum from the surface has also been termed *eczema madidans*. In this form there is presented an intensely red, raw and weeping, or crust covered surface. Through the rapid exudation of serum the horny layer is removed and a thick gummy or syrupy liquid collects on the surface which, if removed, reveals the inflamed and actively discharging skin. If the discharge dries, flakelike crusts are formed. The crusts may be thin and yellow, or thick and dark-colored, and often blood stained. The crusts may remain but a few hours before an excessive outpouring of fluid removes them. The successive appearance of denuded and crusty surface is thus produced. Eczema rubrum may occur on any part of the body, but is seen especially on the legs of elderly people or those who stand much of the time. It is also seen

Fig. 79

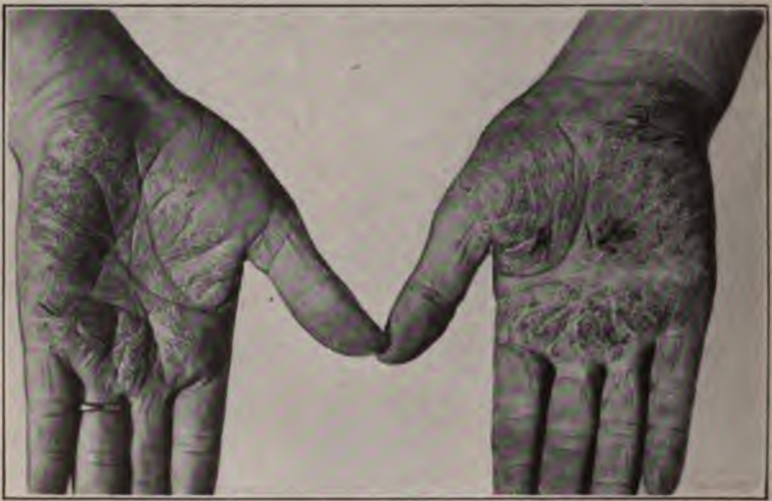


Eczema rubrum.

about the flexures of joints or where two surfaces are apposed. It occasionally occurs on the face of infants consecutive to a severe pustular eczema. On the legs of older people the disorder is exceedingly chronic and rebellious to treatment, eventually being accompanied by marked infiltration and thickening which may produce a condition simulating elephantiasis.

Eczema Squamosum (*Eczema exfoliativum*).—Squamous eczema represents a low grade of inflammation and is present as a transitory condition during a part of the evolution of all other types of the affection. This type is marked by more or less redness, infiltration and exfoliation of the skin. The scales are usually small, thin, whitish and adherent. They may be scattered or abundant. It frequently persists over a long period of time in the form of irregular, usually ill-defined, more or less infiltrated, dry, scaling patches. It is commonly seen on the face, at the border of the scalp, and on the limbs.

FIG. 80



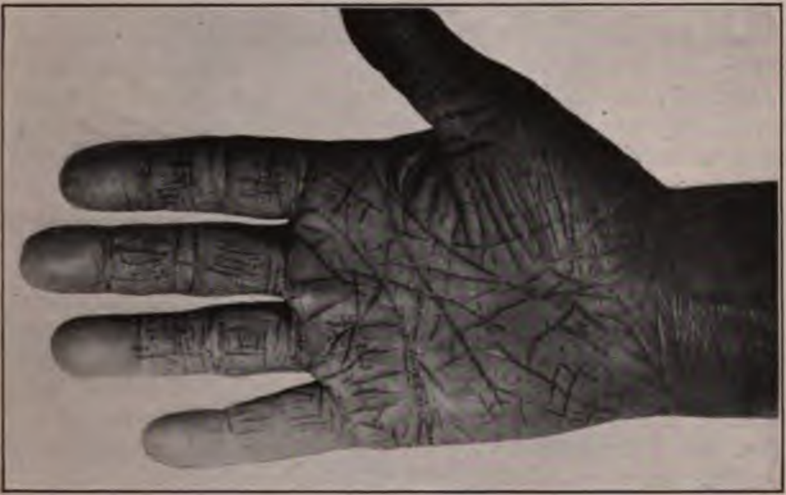
Squamous eczema. (Fordyce and MacKee.)

Eczema Fissum (*Eczema rhagadiforme*).—Fissures are linear tears and occur in eczema as the result of stretching the skin which has lost its elasticity through infiltration and thickening. They are observed most frequently where elasticity and extensibility are necessary to meet the requirements of movement of the parts. They are, therefore, most conspicuous about the hands and fingers, feet and toes, especially over the palms and soles, but they are also observed about the flexures of joints, the angles of the mouth, the nares, nipples, and about the ano-genital region. On the palms, soles and about the anus fissures are frequently deep and very painful. In the former situations they interfere with the occupation and walking and through local irritation may become severe. Chaps or chapping is a mild form of eczema fissum due to local irritants in a dry skin.

Eczéma Craquelé.—This is a rare form of eczema described by French writers, in which a reddened surface is covered with large, thin flakes or scales, separated and outlined in polygonal areas by superficial cracks or fissures. The condition usually involves a considerable surface of the skin and is accompanied by itching and burning, in most cases by hyperesthesia and extreme sensitiveness to temperature changes. It occurs chiefly in neurotic subjects.

Eczema Verrucosum.—Eczema verrucosum, or the wart-like form of the disease is occasionally observed, especially on the lower extremities in middle life or in advanced years, as the result of long-continued disease. These cases commonly occur in people whose return circulation is impaired. The integument becomes thickened and so hypertrophied as to suggest the appearance of warts closely packed together in circumscribed patches, or diffusely distributed over the limb.

FIG. 81



Eczema fissum.

Eczema Sclerosum.—This form is frequently observed upon the palmar or plantar surfaces, and the lower limbs about the ankles. There is presented a densely thickened, inelastic integument, suggesting the condition of tanned leather, without the occurrence of any of the other types of eczema described above. Movements of the joints in the vicinity of these lesions are impaired.

Eczema Nummularis (Devergie).—Eczema nummularis is a localized patchy variety, occurring chiefly on the surfaces of the limbs, particularly the upper but also on the trunk, and is characterized by small, poorly-defined, erythematous and vesicular patches of rapid development; and by itching and exudation. Under the title *Recurrent* *eczematoid affection of the hands,* Pollitzer[1] describes a similar condition limited to the dorsum of the hands, and rarely occurring on the extensor surface of the forearms. In these round, sharply-defined groups, closely aggregated vesicles occur, of an average size of 2 to 3 cm. in diameter. The patch rises suddenly, does not increase in size peripherally, is accompanied by moderately severe paroxysmal itching, and disappears in a few weeks under appropriate treatment. Relapses are common.

The above primary and consecutive types and chronic forms include all that is essential in the description of the disease. Unna described what he termed *Tuberculous eczema of nurslings,* which is not connected in any way with tuberculosis, but was merely a manifestation of the disease occurring about muco-cutaneous orifices induced for the most part by serous discharges from those parts. It has also been the rule to describe a special type of eczema occurring in patients having diabetes under the term *Eczema diabeticorum.* In these cases the eczematous process is increased and made more severe by the irritation by the sugar-charged urine. It is merely an eczema with intense symptoms.

Under the title **Parasitic eczema** a number of conditions have been described. As there is no variety other than that produced by the epidermophyton inguinale, and the infectious eczematoid dermatitis described by Engman, which are now separated from eczema, the term parasitic eczema should be obsolete. The ringworm fungus (*Epidermophyton inguinale*) is responsible for the so-called eczema marginatum (described in the section devoted to ringworm), and also for many cases of eczema intertrigo of the toes. In addition, some of the well-defined patches of so-called eczema occurring on other parts of the body are found to be caused by some member of the ringworm family.

Acute Eczema.—During the course of any eczema an acute exacerbation of symptoms may occur. This is exhibited by a marked increase in the local symptoms present, both in number of lesions and in severity. Acute eczema may, on the other hand, begin as such. In either case, the symptoms are similar. About the face, a commonly attacked area, there is marked swelling and the type of lesion is apt to be erythematous. On other parts of the body the lesions are likely to be vesicular or weeping in type. The involved surface may be limited or extensive, but only rarely are the attacks associated with general symptoms, and those chiefly in children.

Chronic Eczema.—Chronic eczema differs from the acute in several respects. The symptoms are less pronounced. There is much more thickening of the skin and the type of lesion is apt to be papular, erythematous or squamous. The lesions may be few in number, limited, for example, to a single patch for many years, or extensive areas of the cutaneous surface may be involved. In certain areas the thickening is of such degree that deformity of the part results. At any time during its course acute exacerbations may occur and all the signs of an acute eczema supervene.

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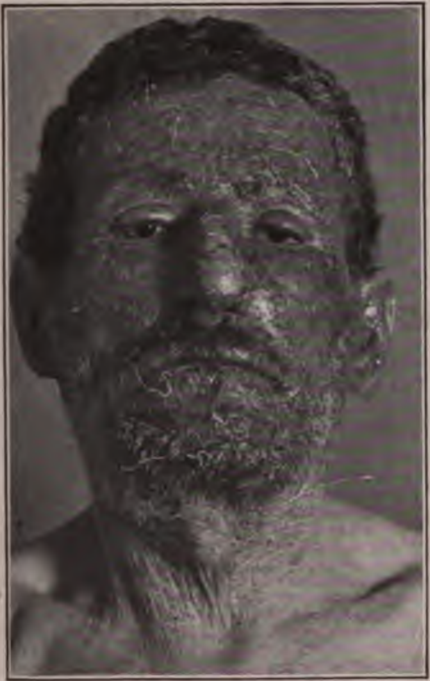
[1] Jour. Cut. Dis., 1912, xxx, p. 716.

Topical Varieties of Eczema.—Certain types of eczema select special regions and these are emphasized below, but it is to be remembered that the description of the various types given on the preceding pages applies irrespective of region.

Eczema of the Scalp.—(*Eczema capitis, Eczema capillitii*).—The erythematous and squamous varieties of the disease are most commonly seen on the scalp. The eruption is usually circumscribed and occurs in patches and the patches may be asymmetrically placed. The disorder may originate in a preceding pruritus which has been irritated through some application. The lesions are usually well defined in outline.

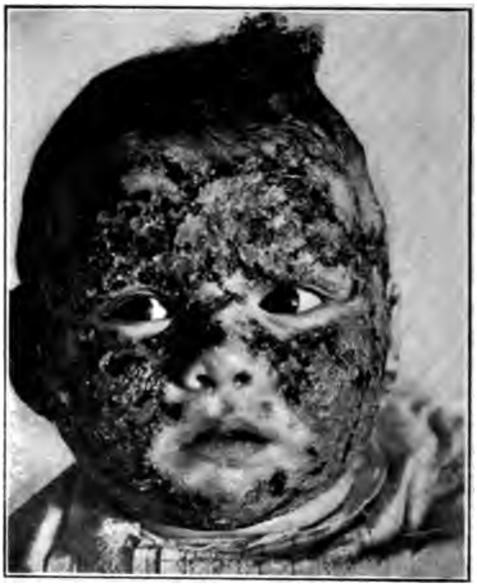
Fig. 82



Acute eczema. (Fordyce and MacKee.)

Eczema of the Face.—(*Eczema faciei*).—The most common type of eczema seen on the face of adults is the erythematous and it is usually uncomplicated. It may be seen during any of the decades of adult life. The skin is infiltrated in varying degrees, is dusky red, involved, as a rule, symmetrically, and the affected surface is elevated slightly above the normal cutaneous level. The forehead, cheeks, eyelids and nose are commonly involved. Occasionally slight oozing is seen after irritation. In the early stages the surface is smooth and reddened but later becomes covered with fine scales. The eyelids particularly the lower, are frequently swollen or puffy. The affected surface is usually well defined. The disorder often extends into the scalp and bearded regions. Itching is intense and the skin may show excoriations from scratching. In certain instances the eruption is asymmetrically disposed on different portions of the face. After the disorder has been present for some time the eyebrows are rubbed off and form a stubble. This type of eczema is seen in people whose faces are exposed to irritation, such as locomotive engineers, pilots of steamships, and laundresses.

FIG. 83



Eczema infantilis of the face. (Fordyce and MacKee.)

Eczema of the Lips.—(*Eczema labiorum*).—Eczema in this location is often chronic on account of its continued aggravation through local irritation. The local action of saliva and the irritation produced by the tongue are important factors. Erythematous, vesicular, pustular, and crusted or squamous lesions occur and through infiltration and thickening, fissures frequently result. The scaling may extend over the vermilion border. Through irritation from the nasal discharge the upper lip may become thickened and elephantiasic. Eczema of the lips is to be distinguished from sycosis, herpes labialis and syphilis.

Eczema of the Nostrils.—(*Eczema narium*).—The process here may involve the mucous membrane as well as the skin in the immediate neighborhood. The Schneiderian membrane is involved in the process and furnishes a secretion which is poured out on the skin. On inspection this membrane appears raw or succulent, or in a condition analogous to that seen in pharyngitis sicca; that is, dry, glazed and free from discharge. The nostrils are often thickened from the inflammatory infiltration, and in consequence present fissures, particularly at the lines of the nares laterally and inferiorly. The lip is usually coincidentally involved and not infrequently there is a complicating sycosis or furunculosis.

Eczema of the Ears.—(*Eczema aurium*).—In this location eczema may be acute or chronic and not infrequently originates as a seborrheic dermatitis, or it may be induced by discharge from the auditory meatus. The whole or only a part of the ear may be involved. The most common site is in the post-auricular region. Pustular and weeping forms are common at the superior, inferior and posterior boundaries of the pinna, where a linear fissure is apt to form in the line of the angle made by the auricle with the plane of the adjacent integument. The lobules may present the erythematous and scaling types of the disorder and become infiltrated, thickened, and deformed. They are dark red in color and the course of the disease is slow and persistent. Sometimes the auricles are uniformly dark red, infiltrated, present alternate weeping and scaling phases, and project to a noticeable extent from the side of the head in consequence of their increased size. Itching is present but usually not severe. There is described a sensation of fulness and tenseness of the part. The disorder is usually symmetrical. It is to be distinguished from seborrheic dermatitis, dermatitis venenata and pyogenic infections.

Eczema of the Eyelids.—(*Eczema palpebrarum*).—In eczema of the eyelids the free edges of the lids or the skin over the orbital margin of the tarsal cartilage may be principally affected. When the free edge of the lid is involved there is present a folliculitis and purulent discharge which may agglutinate the lids. The latter are thickened and swollen and exhibit minute crusts between or glued to the hairs. Fissures occasionally form at the commissure of the eyelids. The disorder is often accompanied by an inflammation of the Meibomian follicles, which is described by oculists under the title of *blepharitis* or *tinea tarsi*. The conjunctivæ may or may not be implicated. A very frequent type seen especially in adults is exhibited as a subacute, inflammatory reaction in which the skin presents a brownish hue. There is marked scaling, frequent fissuring and some thickening. Itching in this variety may be marked; in other types it is moderate.

Eczema of the Beard.—(*Eczema barbæ*).—Eczema in this locality is usually of the pustular type. The surface presents in varying degree redness, infiltration, moisture, pustules and yellow or greenish crusts. After continuing for some time there is much loss of hair. In some cases the erythematous and scaling type is present. In a number of these the pustular condition primarily affects the skin, and secondarily invades the hair follicles, producing a folliculitis. The disorder is likely to be symmetrical and is accompanied by marked itching. The latter is less severe than on the non-bearded portion of the face. Eczema of this region is to be distinguished from sycosis.

Eczema of the Genital Regions.—(*Eczema genitalium*).—The types commonly seen here are erythematous or papular and squamous. In men, on the scrotum, the common type presents a thickened, reddened, slightly scaling integument, which is frequently fissured and covered, with excoriations and bloody crusts. There may be torn papules, nodules and nodose swellings closely packed together exhibiting a dark red or purplish hue. In aggravated cases the infiltration is so great as to deform the parts, increasing the thickness and deepening the normal furrows of the skin to a marked degree, and in some instances producing an elephantiasic appearance.

In women moist forms are more frequently seen. The labia are reddened, edematous, agglutinated by crusts and excoriated severely by the finger nails. An intertriginous type is usually present at the labio-femoral angle. The entire area is frequently bathed in secretions from the mucous membrane in the immediate vicinity.

The subjective sensations in eczema of the genital region are remarkable for their severity. While itching may occur at any time it is more pronounced at night. The disorder in this location persists and recurs, being obstinate to a high degree. It is commonly seen in patients around middle life and more advanced years. Eczema of the genitals is to be distinguished from ringworm, essential pruritus, pediculosis and herpes progenitalis.

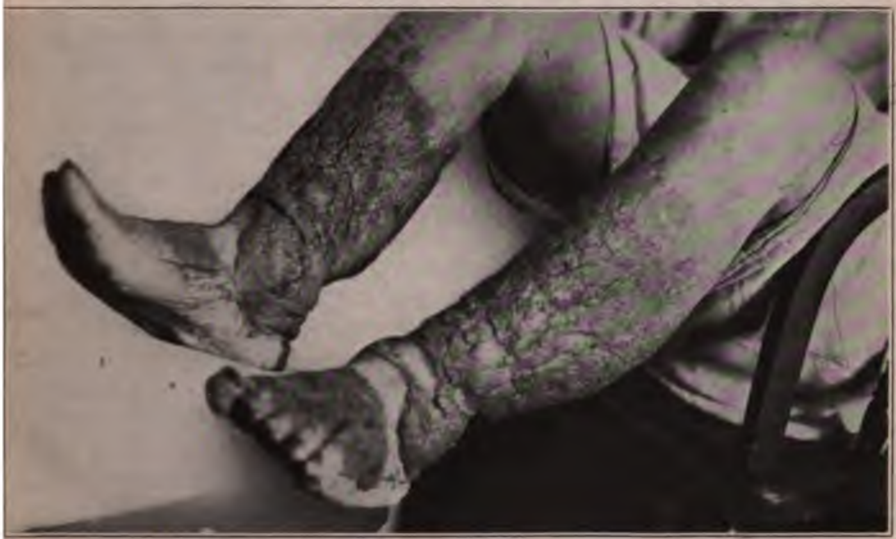
Eczema of the Anus and Anal Region.—(*Eczema ani*).—The common type here is the erythematous, squamous or papular. The redness, infiltration and itching may be limited to the edge of the anus, radiating from the latter in stellate lines, extending upward between the nates in the cleft, over the perineum to the genital region, or laterally over the inner face of each thigh. The buttocks are rarely involved in the process. Fissures and excoriations are usually present about the anal orifice. Itching is intense and not infrequently scratching is indulged in while the patient is asleep. In cases of long standing the skin may become markedly thickened, infiltrated and even present a warty or elephantiasic appearance, with occasional fissures and exaggeration of the normal lines and furrows. There is practically no disorder of the skin that produces so much suffering, both physical and mental.

Eczema of the Nipple and Breast in Women.—(*Eczema mammæ*).—The type seen in this region is the vesicular, erythematous or squamous, complicated by fissures, the latter occurring at the apex or side or the base of the nipple. There is frequently considerable crusting. One or both breasts may be involved. Occasionally an eczema intertrigo is seen in connection with this, but usually they are not associated. Itching and burning sensations are present to a moderate degree. Eczema about the nipple should always be examined with care as the early stage of Paget's disease is eczematous.

Eczema of the Umbilicus.—(*Eczema umbilici*).—In this location there is usually presented a reddish, infiltrated, more or less annular patch surrounding the umbilical depression, which may be filled with crusts. There is commonly a moderate oily discharge which has a disagreeable odor. Eczema in this region is persistent and difficult to treat.

Eczema Crurum.—(*Eczema crurale*).—On account of the feeble circulation in this region eczema is generally severe. The type most commonly seen is a consecutive one, eczema rubrum. In the early stages a common erythematous or pustular type may be present. At a later date when a more intense form is present edema with dense infiltration and induration occurs, or the skin may be covered from the knee to the ankle with large patches of eczema rubrum, presenting an intensely angry appearance, moist and crust-covered. Or, the surface may be dry and glazed and of a lurid, reddish hue, or dry, horny, or warty and reddish with irregular projections covered with scales, the whole resembling the bark of a tree; or the entire integument may be tense, inelastic, more or less covered with scars resulting from varicose ulcers, and pigmented in varying degrees.

Fig. 84



Eczema of the legs with verrucous lesions.

Eczema of the Hands and Feet.—(*Eczema manuum, Eczema pedum*).—All the types of eczema are seen in these situations, erythematous, vesicular, papular, pustular, and squamous. The entire surface may be involved, or the process may be limited to the interdigital surfaces, the palmar or plantar surface of one or more digits of either hand or foot. Fissures are common and often exceedingly painful. Vesicles are most frequently encountered upon the thin portions of the skin, as upon the dorsum and interdigital spaces. They also occur on the palm and sole but do not rupture spontaneously in these areas. Approximately one-third of all cases of eczema occur on the hands and the major portion are induced by the trades and professions. On the *palms* and *soles* eczema is usually asymmetrical, although it may be symmetrical. The palms are more frequently involved than the soles.

The condition is characterized by the appearance of irregular, ill-defined, more or less diffuse areas, of white or hyperemic, infiltrated and thickened integument, which may be fissured or which may be so tense and inelastic that the fingers are semiflexed into the palm or the toes on to the sole. Circumscribed patches of eczema with a fairly well-defined outline, reddish in color, covered with scales, subacute in course and accompanied by paroxysmal itching are common on the palm and sole. On the back of the hand circumscribed patches of vesicular, or erythemato-squamous types of eczema are most commonly seen.

Eczema in Infants and Children.—The most common type exhibited here is the vesicular, vesiculo-pustular, pustular, and consecutive to these eczema rubrum. The face and scalp are most frequently attacked. Crusting is a prominent feature in many instances. The surface beneath the crusts is reddened, infiltrated, moist and moderately swollen. The crusts may be light or heavy and are yellowish or darker colored and the surface exhibits excoriations of varying degree. In the scalp the crusts mat the hairs and not infrequently a foul smelling discharge occurs. When the crusted and pustular type is especially marked in the suboccipital region it is sometimes found to be complicated with pediculosis. Postcervical, preauricular, and occipital adenopathy frequently complicate the process. Hall[1] found that in 96 per cent. of infantile eczemas the face and scalp were the points of origin. Occasionally erythematous and scaling types are seen. In a certain proportion of cases other parts of the surface become involved in the process, especially the flexor surfaces of the elbows and knees. The hands, trunk and, in fact, any portion of the cutaneous surface may present patches of varying size and grade. The course is chronic as to duration, but most of the time presents acute symptoms. It usually begins a few weeks after birth and may continue more or less actively for four or five years, and in some rare instances indefinitely. After some years patches occur which present the characteristics of the subacute and chronic forms, being characterized by more or less infiltration and thickening.

Etiology.—Eczema is one of the diseases of the skin of most frequent occurrence. In the statistics gathered by medical men it would seem to rank first in the order of frequency, forming from 20 to 40 per cent. of dermatological cases reported. It is a disease of both sexes and all ages. It is not in itself hereditary, but a tendency to the disorder may be transmitted from parent to child, though not made manifest until adult life. Eczema may occur in individuals who are in every respect perfect examples of health, but in many instances it is associated with some disturbance of the general economy, and it often occurs in persons who are affected with some systemic disorder. Eczema has diverse causes, both internal and local. A large proportion of cases are due to the external irritation of a sensitized skin.

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[1] Brit. Jour. Derm., 1906, xvii, pp. 161-247 and 287, and Ibid., 1908, xx, p. 4.

Sensitization may be hereditary or acquired, transitory or permanent. The skin may be sensitized by a single application of an irritant, such as rhus. For example, in certain individuals the skin is so sensitized to certain irritants following an attack of dermatitis venenata from ivy, that many and diverse substances are capable of invoking an attack of eczema. In other instances prolonged exposure to the external irritant produces this sensitization. This is illustrated in cases due to formalin. For a long period of time these patients handle formalin without any appreciable bad results, but finally a dermatitis is induced by it and subsequently eczema occurs as a result of the action of many irritants in this sensitized skin. Internal factors inducing sensitization are errors of metabolism, digestive disturbances and faulty action of the glands of internal secretion. Schamberg and Raiziss[1] in a study of nitrogen metabolism in eczema, found true nitrogen retention in patients suffering with eczema of internal origin. Johnston[2] considered derangement of nitrogen metabolism of importance. The relation of digestive disturbance to eczema has been studied by many. Jacquet and Jordanet,[3] in an etiological study of occupational diseases of the hands, conclude that a close association exists between digestive troubles and occupational dermatoses, the internal irritation being the predisposing factor and the external irritation being the exciting factor in these cases. Towle and Talbot[4] in an investitation of infantile eczema and indigestion found a frequent association of indigestion of fats and sugar in the acute exudative types. White[5] in a study of food sensitization found excessive fat or starch in the stools of eczematous infants. The former in the moist, the latter in the dry types. In a further study of causes of chronic eczema[6] he concluded that a good many of the patients suffering with chronic eczema show sensitization to various types of food, and that a large proportion are unable wholly to digest all the forms of food they eat. Strickler[7] in a study of anaphylactic food reactions in forty-six cases of eczema demonstrated a causal connection between the diet and eczema. Fox and Fisher[8] in a study of protein sensitization in eczema conclude that "It seems difficult to draw definite conclusions at present regarding the true value of protein skin tests in eczema of adults . . . . . It would appear that they will ultimately prove to be of therapeutic assistance in a small proportion of cases . . . . ." Engman has demonstrated types of eczema that occur in hypothyroidism. Edelman[9] recorded an example of eczema induced by hypothyroidism which was completely relieved by the administration of thyroid. That many other changes than retention of nitrogen will be proved to be factors is beyond question, and also that glands other than the thyroid will be found upon further study to play an important role in its production is certain.

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[1] Jour. Cut. Dis., 1917, xxxv, p. 135.

[2] Ibid., 1913, xxxi, p. 3.

[3] Annales, January, 1911; abstr., Jour. Cut. Dis., 1911, xxix, p. 564.

[4] Amer. Jour. Dis. of Children, 1912, x; abstr., Jour. Cut. Dis., 1913, xxxi, p. 54.

[5] Jour. Cut. Dis., 1916, xxxiv, p. 57.

[6] Trans. Sec. of Derm., Amer. Med. Assoc., 1916, p. 258.

[7] New York Med. Jour., 1916, civ, p. 198.

[8] Protein Sensitization in Eczema of Adults, Jour. Amer. Med. Assoc., 1920, lxxv, 907.

[9] Ibid., March 9, 1918.

A long list of diseases are commonly enumerated as being the cause of eczema. Clinical experience demonstrates that in some of these eczema does occur, but they can only be considered as contributory factors. The toxemia active in their production or produced by them at times may be the factor that sensitizes the skin to external irritants. Prominent among these disorders are acute rheumatism, diabetes, nephritis, asthma, disorders of the liver, anemia, tuberculosis and syphilis. The victims of gout and chronic rheumatism not infrequently have an obstinate eczema, and the association of asthma and eczema is well known. A local eczema in the genital region in diabetes is also seen not infrequently, and occasionally a more generalized form occurs in this disease which is probably of infectious origin. Many observers believe the nervous system plays a part in the production of eczema. Nervous shock, or prolonged mental depression are considered important factors by Morris.[1] Johnston[2] states that demonstrable lesions of the central, peripheral or sympathetic nervous systems are rare in the eczematous person, but admits that shock, fright, worry and fatigue have to be considered etiologically. Reflex irritation is cited as the cause of eczematous patches occurring at a distance from the original focus. These cases are readily explained through external irritation of the skin in various areas, even though the irritation is not demonstrable. Csillag's[3] experiments show that irritants applied to the skin produce a dermatitis at the area of contact but in no other place if care be taken to prevent accidental conveyance of the irritant to other regions. He holds that in four-fifths of all cases of acute eczema the cause can be shown to be an external irritant acting upon a very sensitive skin and that lack of knowledge of this fact has led to the reflex theory. Hall[4] concludes after a thorough study of the etiology of infantile eczema, that eczema, whether occurring in infants or adults, is a form of reaction of response of the neuro-cutaneous apparatus to external irritation. An abnormally dry skin, such as occurs in mild ichthyosis, is particularly liable to eczema, especially during the cold weather.

External Causes.—The external causes of eczema are chemical, mechanical, thermal or actinic in their action. It is doubtful if any local cause of dermatitis acting for a limited period could produce a persistent eczema without coöperation of other conditions, either external or internal. The large majority of all external operating causes of dermatitis only effect a small proportion of people. The external causes, therefore, are merely the immediate exciting factors in the production of the disease, the underlying factor being the condition that produces sensitization. It is only necessary to mention a few of the chemicals that are actively concerned in the production of eczema. Among these are the various acids, alkalies, antimonial and mercurial compounds, mustard, sulphur, arnica, turpentine, alcohol, formalin, and a long list of other chemicals. Any of these when applied to the skin in a susceptible individual may produce an inflammation which persists after the removal of the cause and develops into a chronic eczema. The various articles handled in the trades and professions are responsible for a large proportion of the cases of eczema. Knowles[1] in an analysis of a large number of cases of eczema of external origin found that one-sixth were caused by the occupation of the individual. There are some forty such avocations that have been proved to be effective. Among these may be mentioned only a few: The confectioner, the baker, the dyer, the printer, the tobacconist, the grocer, the ink manufacturer, the mason, the painter, the gardener and the laundress.

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[1] Diseases of the Skin, 5th Ed., p. 291.

[2] Jour. Cut. Dis., 1913, xxxi, p. 3.

[3] Archiv, 1902, lxiii, p. 213; and Orvosa Hetilap, 1906, 36; abstr., Jour. de Pratic., 1906, No. 16; *Cf.* also Fordyce, Jour. Amer. Med. Assoc., June 13, 1903, p. 1621; and Pinkus, Med. Klinik, 1906, No. 9.

[4] Brit. Jour. Derm., 1905, xvii, pp. 161, 203, 247 and 287.

In susceptible individuals acute eczema may be caused by intense solar light or heat, particularly when aided by reflection from water. Artificial light also, as shown in furnace workers, may be causative. Other external factors are pressure or friction effects produced by contact with shoes, or edges of cuffs, trusses, crutches and corsets.

Scratching not only aggravates an existing eczema, but induces new areas. The experiments of Török[2] and Roma[3] indicate that mechanical irritation of the normal skin even in patients predisposed to eczema, will not produce a vesicular type, although in a very sensitive skin a dermatitis with an exudate may result, and if the irritation be sufficiently prolonged may cause a lichenification. Cold winds and sudden changes in temperature, especially from warm to cold, will often aggravate and prolong an existing eczema. There are many cases of eczema that occur only during cold weather.[4]

Among the *microörganisms*[5] described as the cause of eczema may be mentioned *Morococcus* of Unna.[1] More recently, this organism is being regarded as identical with *Staphylococcus epidermis albus.* Galloway and Eyre[2] describe cocci producing whitish cultures found in early and uncomplicated lesions of papulo-vesicular eczema. Whitfield[3] describes a peculiar diplococcus, which grew in the form of whitish or yellowish cultures on agar and did not liquefy gelatin, isolated from that variety of eczema which manifests itself in the form of small, dry disks on the cheeks of young children.

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[1] Trans. Sec. on Derm., 1916, p. 279.

[2] Archiv, 1902, lxiii, p. 27.

[3] Ibid., p. 39.

[4] Corlett, Jour. Cut. Dis., 1894, xii, p. 457, and Jour. Amer. Med. Assoc., December 20, 1902, p. 1583; Warde, Brit. Jour. Derm., 1903, xv, p. 349, and Corlett and Cole, Amer. Jour. Med. Sci., June, 1912, p. 710.

[5] For a full discussion of the parasitic and other causes of eczema consult the Transactions of the IV International Congress of Dermatology, Paris, 1900 (Compt. rendu, XIII, Congr. Internat. de Méd., p. 9 94, abstr. in British Jour. Derm., 1900, xii, p. 326); also papers by Morris, British Jour. Derm., 1898, x, p. 359; Roberts, ibid, 1899, xi, pp. 7 and 66; Török, Annales, 1898, s. iii, ix, p. 1073, and 1899, s. iii, x, p. 37; Sabouraud, ibid., 1899, s. iii, x, p. 305; Leredde, ibid., 1899, s. iii, x, pp. 30 and 438; Kromayer, Archiv, 1900, liii, p. 85; Scholtz et Raab, Annales, 1900, s. iv, i, p. 409; Whitfield, British Jour. Derm., 1900, xii, p. 406; Schwenter-Trachsler, Monatshefte, 1903, xxxvii, p. 233; Engman, American Medicine, 1902, iv, p. 769; see also chapters by Besnier, La Pratique Dermatologique; and Unna, Mracek's Handbuch. A brief summary is to be found in MacLeod's Pathology, p. 341.

The probability that some forms of eczema are due to toxins or different microörganisms seems to be established by the experiments of Bender and Gerlach.[4] In a long series of control experiments, they found that inoculation of the normal skin with cultures of staphylococci produced an impetigo or a simple pyoderma but when filtered bouillon cultures of the same organisms, which contained no cocci but only their toxins, were employed, the result was a papulo-vesicular eczema of ordinary type. The primary vesicles so produced were sterile, but later contained staphylococci. On the other hand, Cole,[5] in a series of well-controlled experiments, failed to corroborate the above findings. Bockart[6] believes that in individuals predisposed to eczema staphylococci may remain inert in the mouths of follicles until some cause from without or within arouses them into activity. They then produce toxins which are diffused through the epidermis and cause eczema. The lesions so produced are invaded subsequently by cocci and other organisms, so that the later changes in eczema are due largely to other agencies. Pathogenic microörganisms if not concerned in the production of eczema add to the symptoms by their growth, inducing secondary lesions.

Pathology.—Heimann[7] says the pathological changes seen in eczema vary with the type. Both the epidermis and corium are involved in the process; opinion differs as to which is primarily affected. The important primary conditions are edema, parakeratosis and acanthosis in the epidermis, and vascular dilatation, edema and cellular infiltration in the corium. The secondary changes include crust formation and scaling in the epidermis and hypertrophy of the papillæ and collagen in the corium. Ehrmann and Fick[8] find three conditions always present in eczema; viz., acanthosis, spongiosis and parakeratosis. Spongiosis (Unna) indicates a spongy appearance of the rete produced by interstitial edema. The fluid separates the cells, forming small canals which interlace and produce degeneration of some of the cells. Parakeratosis indicates a softened condition of the cells of the stratum corneum in which keratin is absent, and the cells contain nuclei. The normal cornification process is prevented through edema. This abnormal cornification produces the scaling and is a factor in the production of some forms of crusting.

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[1] Monatschefte, 1899, xxix, p. 106.

[2] Brit. Jour. Derm., 1900, xii, p. 307.

[3] Ibid., p.327.

[4] Monatshefte, 1901, xxxiii, p. 149.

[5] Archiv, 1913, cxiv, Sec. 3, p. 207; abstr., Jour. Cut. Dis., 1913, xxxi, p. 593.

[6] Monatshefte, 1901, xxxiii, p. 421.

[7] Jour. Cut. Dis., 1917, vol. xxxv, p. 369. A complete presentation of the histopathology from a personal study.

[8] Kompendium der Speziellen Histopathologie der Haut, Wien, 1906.

The rete, in addition to being edematous and presenting spongioid changes, is thickened (acanthotic), contains vesicles, individual degenerating cells, migrated leukocytes and pus cells. Vesicles are situated chiefly in the upper portion of the rete, but they may occur more deeply and occasionally are entirely within the stratum corneum. MacLeod states that vesicles form in the region of least resistance, which in eczema is commonly the superficial portion of the rete, but when the edema appears with unusual rapidity the greatest strain is put on the cells near the basal layer, where the vesicles then are found. At times cornification occurs beneath the vesicle and then its location is within the stratum corneum. The vesicles are formed by both interstitial and parenchymatous edema. In the former the prickle cells are mechanically separated, forming small spaces lined by elongated or spindle-shaped cells. Some of the cells disappear through pressure atrophy, others become swollen (balloon cells) and remain suspended in the fluid. In parenchymatous edema the cells become edematous around their nuclei, their protoplasm degenerates and by junction through rupture of the walls a vesicle is formed. The vesicles contain serum, fibrin, leukocytes and degenerated epithelial cells.

The corium exhibits vascular dilatation, cellular infiltration and edema. The cells lining the vessels are swollen and the infiltration is made up of leukocytes, young connective-tissue cells and a few mast-cells. The collagen bundles are separated by edema, especially in the papillary layer, but show little change otherwise. The process extends to the subcutaneous tissue, but is less marked in the deeper situation.

Crusts are formed by drying of the contents of ruptured vesicles or by their desiccation without rupture, or serum may come directly to the surface from the edematous rete, the soft cells of the stratum corneum not presenting a barrier to its escape. When this discharge is abundant the stratum corneum may be entirely removed and the rete directly exposed (Eczema rubrum), or more or less completely covered with crusts, dried serum and degenerated cells. This serous discharge is characteristic of eczema. It is a yellowish-white, syrupy liquid, feebly alkaline in reaction and contains a large quantity of albumin. When dry it forms light yellowish to brown friable crusts resembling honey or gum.

In chronic forms acanthosis is more pronounced. The papillæ are much enlarged and the vascular changes and cellular infiltration occur to a greater degree. In elephantiasic forms in addition to the above changes the collagen becomes hypertrophied and increased and the connective-tissue cells are greatly increased in number.

The verrucous forms are due largely to marked enlargement of the papillæ. In the tylotic forms there is marked acanthosis, spongiosis and particularly marked parakeratosis. Pigment is deposited in excess in these chronic forms on the legs, where the circulation is impeded.

Diagnosis.—The recognition of eczema is, as a rule, a simple matter but at times much difficulty is experienced, particularly in trying to distinguish eczema from other forms of dermatitis. There are several disorders in which a differential diagnosis is to be established. Only the most important of them are here enumerated.

Acne Rosacea.—This is distinguished from erythematous eczema of the face by the greater amount of infiltration, marked itching and more widespread distribution of the latter over the face. All the lesions of rosacea are, as a rule, limited to an oval beginning at the center of the forehead, extending down over the cheeks and terminating at the point of the chin. In erythematous eczema the skin is hot to the touch, the reverse being true in rosacea. The former is frequently seen in infancy while the latter never occurs during that period. The accompanying acneform lesions and telangiectasia, and in severe forms moderate scar formation are all distinguishing features of rosacea.

Dermatitis of artificial origin is to be distinguished from eczema rather by its history than by special differences in the appearance or evolution of the lesions. In many cases the two affections are indistinguishable. A history of the external application of irritating or toxic substances is important. Frequently the inflammation in dermatitis is limited to the area of application of the irritant. In cases of dermatitis the onset is sudden and the inflammatory reaction entirely subsides upon withdrawal of the irritant. The subsidence of the symptoms under these circumstances suggests the nature of the affection. Eczema is much more capricious in its distribution and career than dermatitis.

Erysipelas.—Recurring cases of erythematous eczema are not infrequently incorrectly considered to be examples of erysipelas. Erysipelas is usually accompanied by general symptoms, elevation of temperature, headache and nausea. In addition there is a leukocytosis of varying degree, all of which findings are absent in eczema. The affected surface is reddened and much more swollen than in eczema, owing to the involvement of the deeper structures, and the surface presents a peculiar shiny appearance characteristic of this disorder and never seen in erythematous eczema. The line of demarcation between the normal and affected skin is well defined in erysipelas, while poorly defined in eczema. Erysipelas is an acute infection spreading from area to area, each acute exacerbation being accompanied by constitutional symptoms. In eczema of the face that region alone is likely to be involved, while erysipelas may invade the scalp. The subjective symptoms in erysipelas are burning sensations, as well as moderate itching, while in eczema itching is usually severe.

Erythema.—Eczema is readily differentiated from the simple erythemata. From erythema intertrigo it is distinguished by the mild inflammatory reaction in the former. The discharge in erythema intertrigo results from sweat, which does not stiffen linen as does the serous exudation of vesicular eczema. The transformation of erythema intertrigo into eczema is accomplished without much difficulty. The various forms of erythema multiforme are readily distinguished from eczema by absence in the former of severe itching and moisture, or weeping, and by the recurrence of the latter at special seasons of the year. The papules, nodules, and other lesions found in multiform erythema are characteristic and entirely unlike the small lesions that occur in eczema.

Herpes.—Herpes simplex is distinguished from vesicular eczema by the difference in location of the disorders, the former occurring about the muco-cutaneous outlets of the body, by the difference in size of the vesicles, those of herpes being much larger and of more permanent nature than those of eczema. Furthermore, the grouping of several vesicles on a mildly inflammatory base, with normal skin intervening, is entirely unlike patches of vesicular eczema which occur on many parts of the body other than the parts of predilection in herpes. In herpes zoster, the limitation of the eruption to one side of the body, usually along the course of one or more nerves, the large size and deep situation of the vesicles, and the accompanying neuralgic pains, serve to distinguish this disorder from vesicular eczema.

Lichen Planus.—Papular eczema, when occurring in patches, may be confounded with lichen planus under similar conditions, but in the latter disorder typical papules having a flat top and irregular or polygonal base, covered with a thin, closely adherent, shining, scale and having a violaceous or dull crimson hue may be found at the periphery of the patch. The papules of eczema have round or oval bases, acuminate or rounded summits, and are brighter red in color. They form more rapidly and frequently undergo a change of type. The patches of lichen planus are more sharply defined than those of eczema. Upon the involution of the patches in lichen planus a characteristic brown or sepia-tinted pigmentation remains.

Lupus Erythematosus.—In certain instances lupus erythematosus resembles squamous eczema of the face. The great chronicity of the former and firm attachment of the scales, the symmetrical distribution of many patches upon the face, the evident involvement of the sebaceous glands, the well-defined border of each area, and the presence of a smooth delicate scar will distinguish it from eczema. In addition, in eczema itching is present, it may change in type, the scales are much more loosely attached, and never have attached to them the stalactiform plugs on the inferior surface.

Granuloma Fungoides.—Granuloma fungoides, in its early stages, may be indistinguishable from some forms of localized or even generalized eczema. As a rule, however, the early erythematous and eczematoid lesions of granuloma fungoides can be recognized by their characteristic gyrate outlines, assuming, as they do, the shape of a kidney, horseshoe, half-moon, and other fantastic, more or less circinate, forms. These lesions may change frequently in form and location, or and particularly marked parakeratosis. Pigment is deposited in excess in these chronic forms on the legs, where the circulation is impeded.

Diagnosis.—The recognition of eczema is, as a rule, a simple matter but at times much difficulty is experienced, particularly in trying to distinguish eczema from other forms of dermatitis. There are several disorders in which a differential diagnosis is to be established. Only the most important of them are here enumerated.

Acne Rosacea.—This is distinguished from erythematous eczema of the face by the greater amount of infiltration, marked itching and more widespread distribution of the latter over the face. All the lesions of rosacea are, as a rule, limited to an oval beginning at the center of the forehead, extending down over the cheeks and terminating at the point of the chin. In erythematous eczema the skin is hot to the touch, the reverse being true in rosacea. The former is frequently seen in infancy while the latter never occurs during that period. The accompanying acneform lesions and telangiectasia, and in severe forms moderate scar formation are all distinguishing features of rosacea.

Dermatitis of artificial origin is to be distinguished from eczema rather by its history than by special differences in the appearance or evolution of the lesions. In many cases the two affections are indistinguishable. A history of the external application of irritating or toxic substances is important. Frequently the inflammation in dermatitis is limited to the area of application of the irritant. In cases of dermatitis the onset is sudden and the inflammatory reaction entirely subsides upon withdrawal of the irritant. The subsidence of the symptoms under these circumstances suggests the nature of the affection. Eczema is much more capricious in its distribution and career than dermatitis.

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Internal Treatment.—In acute eczema and acute attacks in chronic eczema elimination is essential. In the beginning a mercurial purgative, such as the blue mass pill 5 grains (.33) or calomel 1 grain (0.05) may be given each night for several succeeding nights, followed in the morning by a saline laxative, or a single dose administered every fourth night may be given. Some prefer small doses of calomel, $\frac{1}{10}$ grain (.0064) hourly each day for three days, then four times daily for a period of two weeks. A saline laxative is given during this period each morning. Rhubarb, podophyllin or cascara may be preferable. The various salines and saline waters, such as Hathorn, Karlsbad, Hunyadi Janos, Friedrich and Pluto, are useful, and special combinations, such as the following, are frequently employed.

℞—Ferri sulphatis,		grs. x;	.66
Sodii chlorid.,		ʒj;	4
Acidi sulphuris dil.,		ʒij;	8
Tincturæ cardamoni comp.,		℥ss;	15
Magnesiæ sulphatis,		℥ij;	60
Aquæ dest.,	q. s.	℥-viii;	ad 240

Sig—A tablespoonful before breakfast in a tumblerful of cool or hot water.

℞—Olei menthæ pip.,		grs. iv;	20
Tincturæ colchici,		ʒss;	2
Magnesiæ carbonatis,		ʒj;	4
Magnesiæ sulphatis,		℥ss;	15
Aquæ dest.,	q. s.	℥vj;	ad 180

Sig—Two tablespoonfuls in a wine glass of water every three or four hours.

Castor oil is a valuable remedy in many cases and may be administered every second day. In hyperchlorhydria a powder containing sodium bicarbonate and calcined magnesia āā. 20 grains (1.30) two hours after each meal is valuable, or ammonium carbonate, liquor potassii or milk of magnesia may be employed. Diuretics are frequently necessary. Pure, distilled or alkaline spring water, potassium citrate, acetate or carbonate, and various lithium salts are recommended. In anemia iron in some form is indicated. Cod-liver oil is used in tuberculous subjects. Strychnin, the hypophosphites and arsenic are indicated in certain cases. Small doses of antimony in plethoric individuals is of service.

Diet.—The diet must be regulated to meet individual requirements. There should be a restricted diet in the overfed, and a nutritious diet in those undernourished. Cutaneous tests should be made to determine if there be a sensitization to any foreign protein, and if such be found to exist care should be exercised to prevent that protein from coming in contact with the patient either externally or through the food. A study of the stools may reveal an improper digestion of fats or carbohydrates and this should be corrected.

Such articles as shell fish, except oysters, cheese, pastry, excessive sweets, hot breads, pork, pickles, and pickled meats should be avoided. The ability properly to assimilate such food as meat, tea, coffee, and cocoa, varies so much that no rule can be formulated. In gouty and diabetic individuals a diet proper for such conditions is of much value.

In infants a careful study of the diet is essential and the results obtained are a sufficient reward for the time spent in such investigations. (Cf. Etiology.)

For direct relief of itching internal treatment offers little. Acetyl salicylic acid, quinin and phenacetin are valuable at times and not harmful as a rule. Opium should not be employed.

In certain chronic cases beginning in early childhood and lasting many years in which some metabolic disturbance seems probable, arsenic will at times be of service. It never should be used in acute cases and always with reserve and caution.

Local Treatment.—The local treatment of eczema is necessary in all cases and is all that is required in many instances. In view of the fact that the disease is so diverse in its manifestations, and, furthermore, that so many factors enter into its causation and continuance, and that so little is specifically known about the chief underlying factor (sensitization) the treatment necessarily is symptomatic, and covers a wide and almost endless field. In all cases a prime consideration is protection from external irritants.

The chief applications made use of in the local treatment of eczema are powders, lotions, ointments, glycogelatins, varnishes and paints, and the method of their application is important. It is usually essential that the dressing be constantly applied. Lotions may be applied as a wet dressing, as follows: Saturate a smooth piece of cotton cloth or gauze and lay it evenly over the surface. Place over this several thicknesses of gauze saturated with the lotion. These are removed, moistened and replaced at varying intervals, leaving the layer next to the skin in place during the entire day. The latter is kept wet by pouring the lotion on each time the outer dressings are removed. In all areas where practicable these dressings are kept in place with bandages. In extensive involvement only the most marked area may be thus dressed, the others being treated by frequently dabbing the lotion on the surface. Most lotions have an insoluble powder content which is deposited on the skin as the lotion dries. In case none is present a powder may be dusted over the surface following the application of the lotion.

Ointments and pastes are usually best applied as dressings, held in position by bandaging, and the surface cleansed with an oily or greasy substance at each change of dressing. At times a small amount of either the ointment or paste is thinly applied over the surface and dusted with an inert powder. This procedure prevents the preparation from being immediately removed through external contacts.

In acute phases lotions are preferable in most instances. When practicable the lotion should be employed as a wet dressing for a part of the day, at least. When extensive areas are involved this may not be possible and then its frequent application is necessary. In acute vesicular and erythematous eczema various modifications of the zinc oxid and aqua calcis lotion or calamine lotion are most commonly employed.

℞—Zinci oxidi,			
Amyli pulveris,	āā	℥ss;	15
Liquor calcis,			
Aquæ rosæ,	āā q. s. ad	℥viij;	240
Ft. ung.			

℞—Calaminæ (crude),		ʒij;	8
Zinci oxidi,		℥ss;	15
Liquor calcis,			
Aquæ rosæ,	āā q. s. ad	℥viij;	240
Ft. ung.			

When a greater antipruritic effect is desired phenol or dilute hydrocyanic acid in the strength of ¼ to 1 per cent. may be added. If the former is used glycerin, drams ij (8) must be added to insure proper solution. When the above preparations are too drying tragacanth mucilage, drams ij (8) or oatmeal water may be added at each application, or olive, almond or other oil may be added to form a liniment. These liniments are useful when large surfaces are involved. A useful formula that may often be used alternately with the above lotions is:

℞—Bismuth sub. nit.,		ʒj;	4
Zinci oxidi,		ʒij;	8
Olei olivæ,			
Liquor calcis,	āā q. s. ad	℥viij;	240

Black wash, pure or diluted, is occasionally serviceable. Mixed with an equal amount of olive oil it is soothing and protective. Other useful lotions are the following:

℞—Zinci oxidi,			
Calaminæ (crude),	āā	ʒij;	8
Liquor calcis,			
Aquæ sambuci,	āā q. s. ad	℥viij;	240

℞—Bismuth subnitratis,		ʒj;	4
Tragacanthæ,		℈ij;	2 66
Liquor calcis,	q s ad	℥viij;	240

Phenol and glycerin or dilute hydrocyanic acid may be added here as above noted.

In similar cases the lead and opium wash is valuable:

℞—Tinct. opii,		℥ss;	15
Liquor plumbi,			
subacetatis diluti,	q. s. ad	℥viij;	240

Zinc oxid, talcum, boric acid, or other powder may be added to this lotion. A good soothing lotion is composed of sodium bicarbonate, or biborate 1 to 2 drams (4–8) to a quart (1000) of thin oatmeal gruel or of marshmallow decoction.

When the lesions are of the pustular type boric acid, drams ijss (10) or biborate of sodium, drams ijss. (10) may be added to the zinc oxid and aqua calcis or calamine lotion, or a saturated solution of boric acid into which 2 per cent. or more of glycerin or tragacanth mucilage is added, may be employed. In certain instances a weak solution

(1–4000) of potassium permanganate may be used over small areas. It is both antiseptic and antipruritic. Ichthyol and thiol in aqueous solution (2 to 10 per cent.) may be similarly used by painting over the surface.

While lotions are usually indicated in acute moist forms of eczema, at times ointments may be of service. A valuable ointment that may be employed in any moist or weeping case with excellent results is composed of naftalan, 50 parts; zinc oxid, 25 parts; and amylum, 25 parts. This is applied on a dressing and held in position with a bandage. The dressing is renewed morning and evening and the parts cleansed at each dressing with olive or other oil. White[1] recommends crude coal tar in all moist, non-pustular phases in the following formula: Crude coal tar, 2 grams; zinc oxid, 2 grams; amylum, 16 grams; petrolatum, 16 grams. This is applied twice daily under one layer of thin compress until the lesion disappears.

At times the combined use of lotions or ointments is preferable. The lotion then is applied during the day or for varying periods of time during the day, and the ointment during the remainder of the time. When the combined lotion and ointment method is employed in pustular varieties it is usual to add salicylic acid 1 per cent., ammoniated mercury or sulphur 1 to 3 per cent. to the ointment selected.

After the subsidence of the acute symptoms, indicated by disappearance of the vesicles in the vesicular type and the swelling and acute inflammatory reaction in the erythematous type, ointments and pastes should be employed until resolution is complete. At this time any of the following will be found serviceable:

℞—Ichthyolis,		℈ij;	2 40
Zinci oxidi,		℥j;	30
Amyli,		℥j;	30
Ungt. petrolati,	q. s. ad	℥iv;	120

In the above naftalan may be added in the strength of 10 to 25 per cent., or ichthyol may be replaced by salicylic acid, ammoniated mercury or sulphur in the strength of 1 to 2 per cent.

℞—Ungt. diachylon alb., (Hebra)	℥j;	30
Ungt. zinci oxidi,	℥j;	30

℞—Bismuth oxidi,		ʒj;	4
Ceræ albæ,		ʒiij;	12
Ungt. petrolati,		℥j;	30
Ol. olivæ		℥j;	30
Ol. Rosæ,	q. s.		

It is essential to continue the application until all evidence of the inflammatory process has disappeared.

In subacute and chronic eczema more stimulating preparations are necessary. Ointments and pastes are more frequently used, though lotions are valuable. The latter are commonly employed here for limited periods of time during the day with an ointment or paste in the interim. Some useful lotions follow:

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[1] Jour. Cut. Dis., 1916, xxxiv, p. 497.

℞—Liquor carbonis detergens,		ʒss;	2
Liquor plumbi subacetatis,		ʒss;	2
Aqua dest.	q. s. ad	℥viij;	240

℞—Mentholis,		℈ij;	2 40
Alcoholis,	q. s.		
Glycerini,		ʒj;	8
Aqua. dest.,	q. s. ad	℥viij;	240

Camphor my be substituted for menthol in the above, and either may be employed in the strength of 1 to 3 per cent. A powder, such as zinc oxid or talcum may be added for greater protection.

℞—Liquor picis alkalinis,		ʒss–ʒij;	2–8
Glycerini,		ʒij;	8
Aqua dest.,	q. s. ad	℥viij;	240

For dry, irritable and itching patches Boeck recommends:

℞—Talci,	℥ij;	60
Amyli	℥ij;	60
Glycerini,	ʒvj;	24
Liquor plumbi subacetatis dil.,	℥iv;	120

This is diluted with 2 parts of water and applied with a swab or brush.

Ointments and pastes usually contain salicylic acid, ammoniated mercury, calomel, resorcin, sulphur, tar, ichthyol, boric acid, menthol, and occasionally phenol, chrysarobin or pyrogallic acid. These ingredients are used in varying strength, depending upon the amount of inflammatory reaction, infiltration, thickening and irritability. In dry, scaling types, glycogelatin, salve and plaster mulls, paints and varnishes are frequently serviceable, whereas in sluggish and thickened areas plasters, and mild caustics are at times necessary.

Ointment bases include cold cream salve, lanolin, eucerin, lard, and simple cerate, alone or in varying combinations. Cerates require one to two drams (4 to 8) of glycerin to the ounce (30) of cerate. They may be stiffened and rendered more protective and astringent by the addition of a dram (4) or more of zinc oxid, calamine, bismuth subnitrate or subcarbonate to the ounce (30). A thin base is made up of equal parts of lanolin, olive oil and glycerin. This is serviceable on hairy surfaces. Unna's refrigerant ointment contains lanolin, 10 parts; lard, 20 parts; and rose water, 30 to 60 parts. To any of these simple bases may be added the active medicament above mentioned. Pastes are thicker and more protective and usually contain equal quantities of powder and ointment base. Lassar's serves as an example:

℞—Zinci oxidi,	ʒij;	8
Amyli,	ʒij;	8
Petrolati,	℥ss;	16

The four base paste is at times preferable:

℞—Zinci oxidi,	ʒij;	8
Amyli,	ʒij;	8
Lanolini,	ʒij;	8
Petrolati,	ʒij;	8

As with ointment bases, any selected active medicament may be added.

In cases presenting subacute symptoms with moderate infiltration and thickening, some of the following ointments are useful:

℞—Acidi salicylici,	grs. xv;	1
Ungt. zinci oxidi,	℥iij-ʒj;	100

Ammoniated mercury, calomel, or resorcin may be substituted for the salicylic acid.

In many of these cases a paste answers as well as an ointment. The alternate use of a mildly stimulative lotion with an ointment serves a better purpose than either alone.

In certain of these cases Pick's[1] linimentum exsiccans, variously medicated, is useful. This contains tragacanth 5, glycerin 2, boiling water to make 100. To this may be added phenol ½ to 2 per cent., or boric acid 2 per cent. Stelwagon[2] suggests zinc oxid 2 parts, glycerin 1 part, and mucilage of acacia 5 to 8 parts, as it dries more easily. Bassorin paste[3] suggested by Elliot may be used similarly. Bassorin 48 parts, dextrin 25 parts, glycerin 10 parts and water to make 100 parts. This is prepared cold. When there is no discharge and complete protection is desired, glycogelatin dressing is indicated. Several formulæ are employed. A satisfactory combination suggested by Fraser follows:

℞—Zinci oxidi,	℥ij-grs. v;	67 50
Glycerini,	℥iv;	120
Gelatini (flake),	℥iv-gr. xv;	135
Aqua destill.,	℥vj;	180

The ingredients are mixed on a hot water-bath and when cool and solidified may be cut in pieces of convenient size for use. Before application a sufficient quantity is melted in a dish placed in a receptacle containing water, which is heated to a suitable degree; the liquefied material is then applied with a brush, care being taken that it is not uncomfortably hot for the patient. It dries somewhat slowly, and it is well after two or three minutes to pat the surface with cotton or to cover it completely with gauze. By increasing the quantity of glycerin a softer and more slowly drying preparation is formed. By lessening the quantity of glycerin and increasing that of the zinc oxid or gelatin a firmer and more rapidly drying product is obtained. Though these glycogelatins serve their best purpose in the dry forms of the disease, there are few forms of eczema in which they may not at times be used with benefit.

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[1] Archives, 1891, p. 633.

[2] Diseases of the Skin, 8th Ed., p. 321.

[3] Elliot, Jour. Cut. Dis., 1891, ix, p. 480; 1892, x, p. 184.

When infiltration is marked and a more chronic type is present, the above remedial agents are employed in greater strength. In these tar is of much service. In fact, tar is valuable in all except acute cases. In some of its forms it probably is more valuable than any other drug, but at the same time may change a subacute or chronic type into the acute form. It always should be used with caution. Crude coal tar[1] is a most valuable preparation. It may be employed in varying strengths in ointments or pastes, or painted on the surface in pure state. It is of value not only in chronic, dry, infiltrated types, but in weeping phases as well. Other most commonly used preparations are pix liquida (pine tar), oleum rusci (oil of white birch), oleum cadinum (oil of cade) and terebinthina canadensis (balsam of fir). The tars are usually employed in the strength of ½ to 2 drams (2 to 8) in one ounce (30) of ointment base. Occasionally they are painted on in a liquid state with a camel's-hair brush.

In beginning the use of tar with any individual, weak preparations should be employed, and the strength be gradually increased until tolerance of the skin is determined, as an acute dermatitis not infrequently follows the application of stronger preparations. A convenient method is to order one jar of a fairly strong tar ointment and another of the zinc oxid, the Hebra, or other simple salve. Before the first application the patient takes a sufficient quantity of the simple ointment and mixes it with a very small proportion of the tarry preparation. If no irritation follows this application, the amount of tar can be gradually increased with each dressing until enough is used to relieve the itching and to reduce the infiltration, after which a simple paste or powder may be employed until the skin has regained its normal strength and resistance. If the application at any time causes an acute dermatitis, simpler remedies for a time must be substituted. To accomplish the best results, tar ointments should be rubbed well into the skin or liquid preparations painted on. Sometimes it is well to permit the application to accumulate until thrown off by exfoliation, but it is better to cleanse the skin with oil or with soap and water according to indications, before each application.

Some combinations follow:

℞—Ol. cadini (olei rusci),		ʒss–ʒiij;	2–12
Potass. bicarbonatis,		grs. xx–ʒss;	1.33–2
Ungt. aq. rosae,	q. s. ad	℥j;	30

For the potassium bicarbonate ½ to 1 dram (2 to 4) of zinc oxid may be substituted, or from 2 to 4 grains (0.133 to 0.266) of red mercuric oxid, or ½ scruple (0.66) of mild chlorid. The vehicle, also, of such ointments may be vaseline, lanolin, simple cerate, or ½ ounce (15) of either in combination with an equal quantity of diachylon ointment.

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[1] Sutton, Jour. Amer. Med. Assoc., August 8, 1908, p. 497; Rygier and Miller, Archiv, October, 1912; Alste, Brit. Jour. Derm., 1913, xxv, p. 73; White, loc. cit.

A modified Wilkinson ointment is also valuable:

℞—Cretæ preparat.,		℈iiss;	3	33
Sulphuris sublimat.,				
Ol. rusci,	āā	ʒij;	8	
Saponis mollis,				
Adipis,	āā	℥ss;	15	
M. ft. ungt.				

Another valuable formula containing sulphur and salicylic acid in addition follows:

℞—Acidi salicylici,		grs. iij;		20
Mentholis,		grs. iiss;		16
Sulphuris ppt.,		ʒss;	2	
Zinci oxidi,		ʒj;	4	
Olei cadini (Muth & Co.),		ʒj;	4	
Amyli pulvis,		ʒij;	8	
Ungt. petrolati,	q. s. ad	℥j;	30	
M. et. ft. ungt.				

When greater stimulation is required chrysarobin or pyrogallol may be employed in ointment form in the strength of 5 per cent. The staining of the skin must be remembered when ordering these preparations, and it is usually best to advise their use in limited areas only where there is much infiltration, and to limit their use to five daily applications, following this with a soothing ointment until the reaction has subsided. Should reaction occur before the fifth day suspend the treatment at its appearance and employ the soothing application.

At times favorable results may be obtained in these chronic, circumscribed infiltrated patches by daily washing with sapo mollis, followed by the application of diachylon ointment or one of the above mentioned tar combinations.

If more energetic measures become necessary a salicylic acid plaster (Unna) may be applied for a few days, followed by a bland ointment.

Liquor picis alkalinis (Bulkley) is at times valuable in this type. This may be employed as a lotion diluted with 4 to 8 parts of water, or one dram (4) or more may be incorporated in an ointment base—its formula follows:

℞—Potassii hydroxidi,	℥j;	30
Picis liquidæ,	℥ij;	60
Aq. dest.,	℥v;	150

Radiotherapy is valuable in these cases as well as the more subacute and less infiltrated types. It furthermore furnishes a most valuable method in the whole group of so-called trade or professional eczemas occurring on the hands.

The treatment of eczema in particular locations, such as the scalp, face, ears, genital and anal regions is usually described by repeating what has already been stated in discussing the subject of its various phases. It is only necessary to determine the type presented in these various situations and employ the treatment recommended for that type. In hairy regions lotions should not contain powders in suspension, and ointment bases should not contain lard; chrysarobin and pyrogallol should not be employed on the face nor in the scalp. It is rarely necessary to cut the hair in eczema of the scalp.

Treatment in Infants.—It is essential that the diet should be studied and carefully regulated to fit the age and other findings. Reduction in the amount of fats or starches given is indicated when these are found in excess in the feces. The importance of a correct diet has been definitely proved in the work of Towle, White and others mentioned in the section on etiology.

In the local management of infantile eczema of the face, the points of importance are avoidance of all external irritating applications, including soap and water; the removal of crusts and débris with olive or other oil or white vaseline; and the application of soothing preparations held in position with a mask. The dressings are best retained in position by using a skull-cap, made of firm, old cotton or linen cloth, which is closely fitted to the calvarium, and a mask of the same material is shaped to the face, with exactly placed apertures for the eyes, nose, mouth, and ears. This mask is gathered in beneath the chin, and laps over two inches at the back of the head; it may be used only during sleep, or, in aggravated cases, also during the day.

The lotions, oils, or ointments required are placed on pieces of soft cloth, applied to the face, and the above described device used to hold them in place. To prevent scratching of the face, splints should be applied extending from the middle of the forearm to the middle of the arm on the flexor surface. The application of most value in these cases is naftalan, used as before mentioned in acute eczema. The zinc oxid and lime water lotion or the calamine lotion are valuable, also the following oily cream: Zinc oxid, 8 parts; bismuth subnitrate, 4 parts; olive oil, 120 parts; and liquor calcis, 120 parts. In addition the black-wash and zinc salve treatment, the diachylon salve, Lassar paste, boric acid ointment, lead lotions, glycerol of starch, and other preparations and methods described in full in the treatment of acute eczema are often useful. These cases are often very capricious in their course, and treatment may have to be changed frequently to meet the varying conditions.

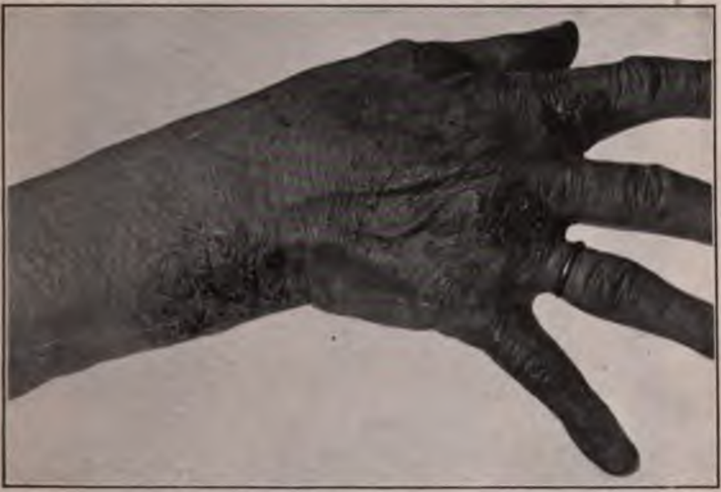
Prognosis.—While many patients recover entirely from eczema, the duration of any particular case cannot be predicted. Acute attacks usually subside in a reasonable time, but the question of relapse has always to be considered. The disorder is not contagious nor hereditary, and these are important questions with many afflicted with the disease. As a rule, acute eczema is much more readily relieved by proper treatment than are the chronic forms of the disease. Some of the latter, particularly in the old, persist indefinitely. Relief from symptoms can in most instances be promised within a reasonable time, but the possibility of recurrence should never be forgotten.

INFECTIOUS ECZEMATOID DERMATITIS.[1]

In 1902 Engman isolated a group of cases which had been hitherto considered eczema and described them under the above title.

Symptoms.—The disorder is characterized by areas of dry, scaling dermatitis, or large weeping patches, or a combination of moist and crusted lesions in addition to lesions which had the characteristics of an acute, vesicular eczema or eczema rubrum. Engman, in a close study of the development of the affection, describes the primary lesion as an erythematous, scaling or crusted, weeping spot. From these lesions, patches are formed by peripheral extension of the initial points or by coalescence of many individual lesions. When fully developed the patches are crusted, weeping or scaling and they enlarge at the periphery by the formation of pustules or vesicles or, more commonly, by the splitting up or undermining of the epidermis. There is never any attempt at central involution of the lesions. In aggravated forms of this dermatitis there occurs a rapidly spreading discharging surface, presenting a sharply defined irregular border. The peripheral epidermis is raised by the serous discharge and in a comparatively short time the epidermis may be removed over large surfaces. Over the face and some other areas marked edema is present. When the disorder occurs about the limbs, and also in some other situations the superficial epidermis is removed and the surface is covered by a sticky seropurulent discharge which oozes from various points and frequently dries into thin crusts, producing a condition indistinguishable from

FIG. 85



Eczematoid dermatitis of a senile skin.

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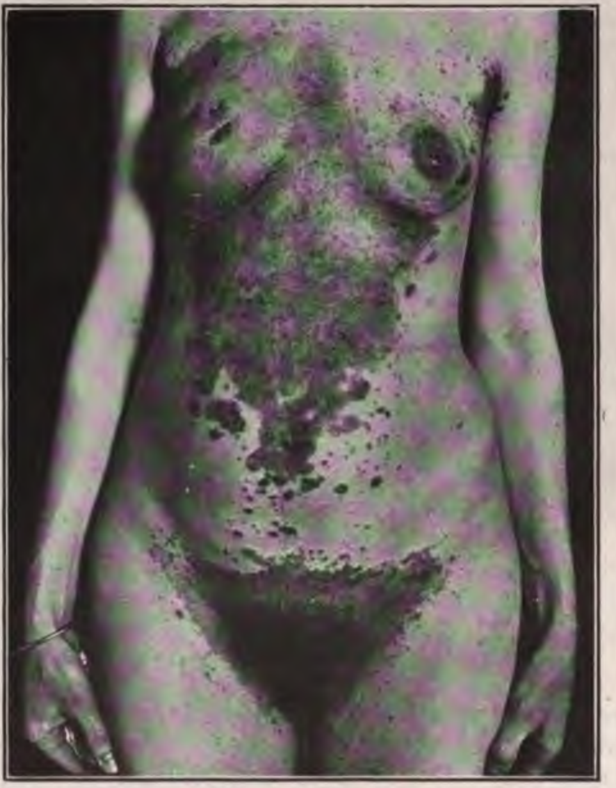
[1] Engman, Amer. Med., 1902, lv, p. 769; Fordyce, Jour. Cut. Dis., 1911, xxix, p. 129, Sutton, Jour. Amer. Med. Assoc., 1920, lxxv, p. 976.

eczema rubrum. The subjective symptoms in this type of dermatitis are much more mild than in eczema, although the disease is undoubtedly spread by scratching.

Etiology.—The disorder is produced by staphylococci, chiefly *Staphylococcus albus.* It is induced in the major portion of cases by an antecedent pus infection, such as discharges from abscessed sinuses, ulcers and other pus secreting surfaces. A simple pustule following trauma, insect bites, chemical or thermal irritation is not infrequently the starting point of this affection.

Fig. 86

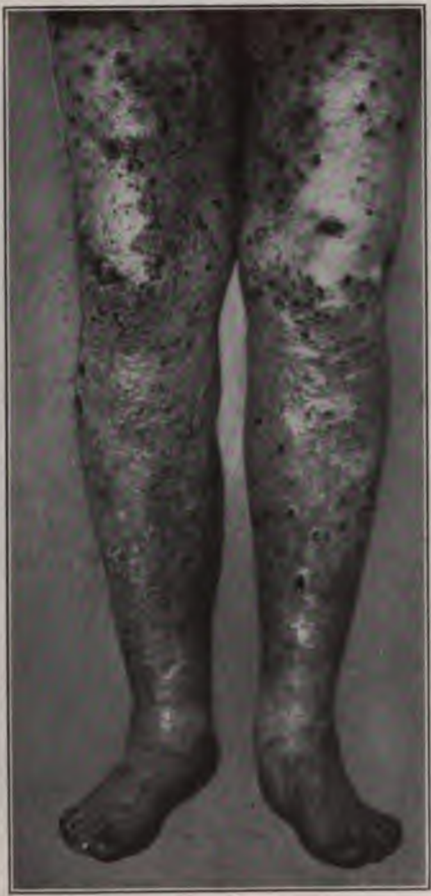


Infectious eczematoid dermatitis following abscess of the breast. (Fordyce and MacKee.)

Diagnosis.—Eczema is the only disorder from which it is to be distinguished. The important distinctive points are the well-defined lesions, more mild subjective symptoms, as a rule, the larger size of the vesicles, the development of the disease following a definite local infective process, the evident peripheral extension of the lesions, and the not infrequent involvement of the nearby glands.

Treatment.—Local antiseptic preparations have been employed with success, although many of these cases resist treatment the same as those of ordinary eczema. Vaccine therapy has been of service to Engman and others. Ointments containing 1 to 3 per cent. salicylic acid or ammoniated mercury, or sulphur, have been serviceable. In extensive cases it is sometimes necessary to use boric acid baths to remove the crusts, and in these cases lotions such as the zinc oxid and calamine, to which has been added boric acid or biborate of sodium, are of service.

FIG. 87



Infectious eczematoid dermatitis, following ecthyma. (Fordyce.)

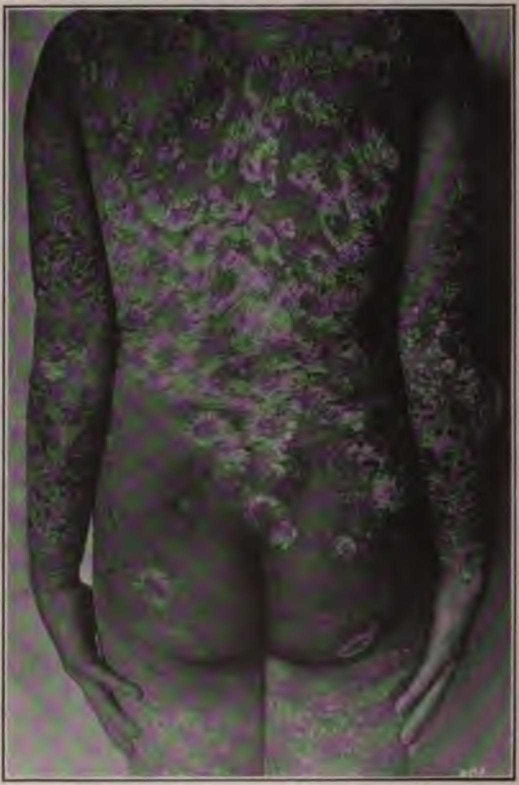
PSORIASIS.

Synonyms.—Lepra, Alphos, Psora. Ger., *Schuppenflechte.*

Definition.—Psoriasis is a chronic, occasionally acute, inflammatory disease characterized by reddish-brown, flat papules, or circumscribed plaques or areas of varying size, covered with silvery-white, imbricated scales.

Symptoms.—In typical evolution, the papules and plaques of psoriasis always are sharply defined from the surrounding skin, somewhat infiltrated, slightly elevated, and covered more or less completely with silvery-white or mother-of-pearl-colored scales, which are arranged in thin layers like mica. On removal of the scales, there is exposed, in recent lesions, a bright-red surface; in older lesions the color is of a duller hue. If the deepest scale, which is often thin, translucent, and closely adherent, is pulled or scraped off, there can be seen several minute bleeding points, which correspond to the apices of papillæ beneath. The lesions vary greatly in number, size, shape, and distribution, but the type (that of the dry papule or plaque covered with scales) always remains the same; so that in uncomplicated cases psoriasis is a distinctly dry disease, without vesicles, pustules, or other moist lesions.

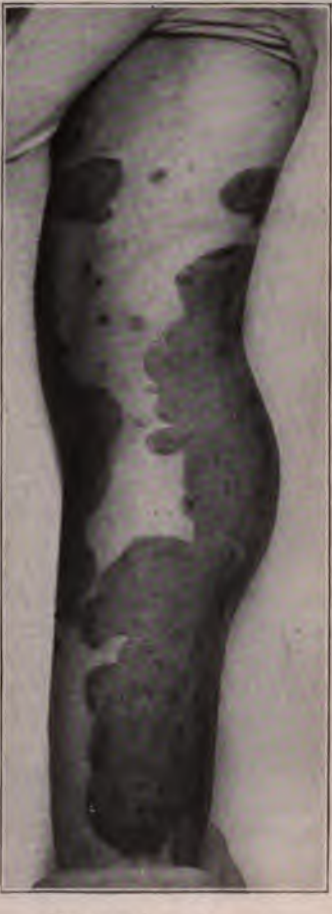
Fig. 88



Psoriasis with dermatitis exfoliativa beginning on the arms as a result of treatment with chrysarobin.

The primary lesion of psoriasis is a pinpoint- or pinhead-sized, flat, round or oval, sharply defined, slightly elevated, red papule, which always at the earliest moment of observation is covered either entirely or all but a narrow rim at the border with delicate silvery-white or mica-white scales. The bleeding points produced by forcibly removing the scales may be so minute that they are only visible with the aid of a lens. As the lesion grows peripherally, it may become somewhat more infiltrated, slightly more elevated, and covered with more abundant imbricated scales; but otherwise it retains its original characteristics. Larger plaques and areas are all formed either by the gradual increase in size of the original papules, or by the coalescence of a number of papules or smaller plaques. The small plaques formed by the peripheral growth of single papules are usually round or oval, but areas formed by the coalescence of smaller plaques are irregular in outline. As a matter of convenience, descriptive terms have been applied to the lesions of psoriasis to denote their size and arrangement.

Fig. 89



Psoriasis (large plaques).

Psoriasis punctata describes the disease which occurs in the form of small, scale-covered points. *Psoriasis guttata* indicates that form of the disease with lesions approximating the size of drops of water. When the patches become the size of small coins, they are termed *psoriasis nummularis* or *discoidea*. *Psoriasis circinata* or *orbicularis* is characterized by patches exhibiting activity at the periphery of the circle, the center of which is free from disease; a condition due usually to the involution of the center as the disease extends peripherally. The coalescence of spreading circinate patches produces *psoriasis gyrata* and *figurata*, in which case fantastic figures are frequently produced. *Psoriasis diffusa* indicates that form where the cutaneous surface is affected in large areas. When the coil-glands and hair-follicles are chiefly invaded, the disease is termed *psoriasis follicularis*. Areas of long persistence, in which the skin is infiltrated deeply and often fissured and covered with heavy scales, are designated as *psoriasis inveterata*.

Psoriasis rupioides (*Cf.* Parakeratosis scutularis) indicates a variety of psoriasis in which large, conical crusts, marked by concentric rings, occur on many patches.[1]

In a given case the lesions may be of fairly uniform size, but more commonly, if at all numerous, they exhibit different stages of development, and therefore vary in size. They may be arrested at any stage of growth, and persist for months or years as guttate, nummular, or larger plaques; or, by continued extension and coalescence, form areas covering an entire region of the body. Though cases are reported in which the surface of the entire body is covered, it is rare that areas of normal skin cannot be detected.

In number and distribution of its lesions and in its course psoriasis varies greatly. The disease commonly begins with one or two small papules, which increase slowly in size. In ordinary cases new lesions appear during the course of weeks, months, or years, until there are often from ten to one hundred or more patches of varying size scattered over the body. It is not unusual, however, for the disease to remain for years limited to two or three coin-sized areas, situated commonly over the elbows and knees. Occasionally, a single patch may persist indefinitely without the appearance of others. In other instances, but chiefly in recurrences of the disease, a large number of punctate papules may appear within a few days; and at times even a generalized, acute attack occurs. In the same individual the number, size, and distribution of the patches vary from time to time. With many patients the psoriatic areas partially or wholly disappear in summer, only to return in cold weather. In a smaller number of cases the disease is worse in summer and better or entirely absent in winter. Without the influence of climate or any other known cause, the disease may disappear, partially or wholly, for months or years and then return. In recurrences of the disease, the lesions do not necessarily correspond in number, size or distribution with those of earlier attacks. In acute febrile and other intercurrent diseases, patches of psoriasis may fade or disappear temporarily.

Involution of a patch of psoriasis begins in the center, and is recognized by diminution in the hyperemia and in the scaling. The process progresses slowly until no trace of the disorder is left. Temporary pigmentation may remain for weeks (on the lower extremities for months) after the scaling and infiltration have completely disappeared.

In distribution psoriasis is, as a rule, symmetrical, but exceptions to the rule occur. The sites of preference of the disease are the extensor surfaces of the extremities, especially about the elbow and knee, in which situation it is decidedly most common. After these locations should be named in order the scalp, the region of the sacrum, the upper surface of the chest, the face, the abdomen, and the genitals; more rarely the hands and feet.

Upon the scalp plaques of well-defined contour, covered with thick, whitish scales, may mat the hairs, but alopecia rarely results. Often a fillet or band one or two inches in width projects beyond the border line of the scalp over the forehead. When the vertex is bald from physiological loss of hair, the patch of psoriasis usually lingers near the fringe of hair left at the sides of the head, projecting thence to the regions of baldness. On the face the lesions are usually indistinct and small in size, being displayed over the cheeks, chin, and nose, avoiding parts near the mucous orifices. In the genital region, also, the lesions are usually small and indistinct, and over the scrotum psoriasis is usually complicated by fissures, moisture, and other evidence of acute inflammation.

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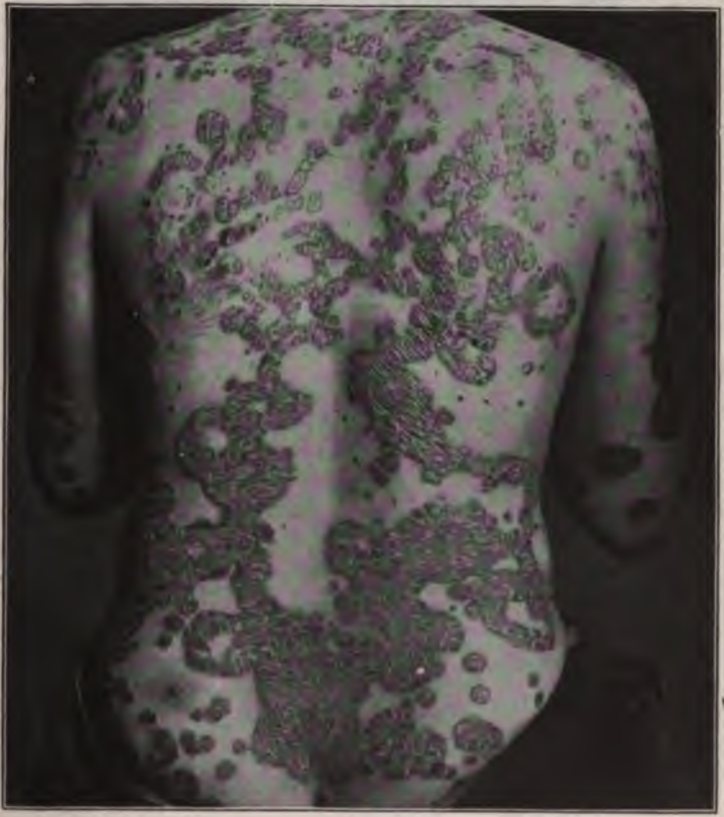
[1] Anderson, Treatise on Dis. of the Skin, London, 1887, p. 310.

FIG. 90

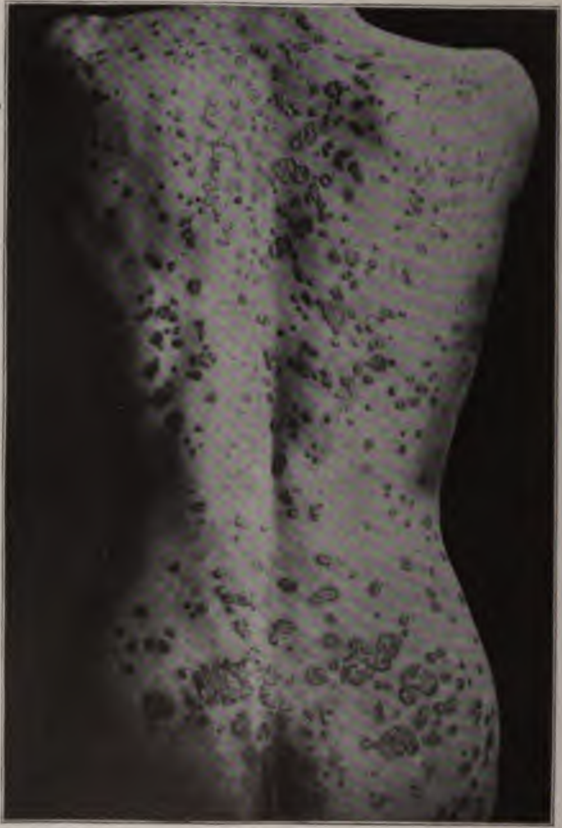


Generalized psoriasis. Lesions coalescing in places to form large plaques.

The hands, feet, fingers, and toes are not often involved, and the palms and soles only rarely. The author has had two cases in which the disease was limited to the palm for considerable periods of time before the appearance of characteristic lesions on other parts of the body.

Other writers report similar instances. In many cases the nails are attacked, being thickened, eroded in points, irregularly laminated, rigid, brittle, and yellowish-white or dirty-whitish in color. In certain cases the nails alone are attacked (*Cf.* the section on Diseases of the Nails). On the palms and soles the lesions may show, instead of scaling, sharply circumscribed areas, in which the horny layer is much thickened. Occasionally, bullous lesions develop in these regions.[1] Through cracking and partial destruction of horny masses, the patches may assume a worm-eaten appearance.

FIG. 91



Generalized psoriasis. Guttate and nummular lesions.

Psoriasis is not known to affect the mucous surfaces. The lesions of so-called *psoriasis linguæ* are those of leukoplakia buccalis, or "smokers' patches," of syphilitic disease of the mouth, or flat epitheliomata (Hyde). This opinion is corroborated by other writers.[1] Schütz[2] reports two cases and refers to others in which psoriasis was associated with mucous-membrane lesions. These lesions, however, occur with other cutaneous and systemic disorders, and their relation to psoriasis is not demonstrable.

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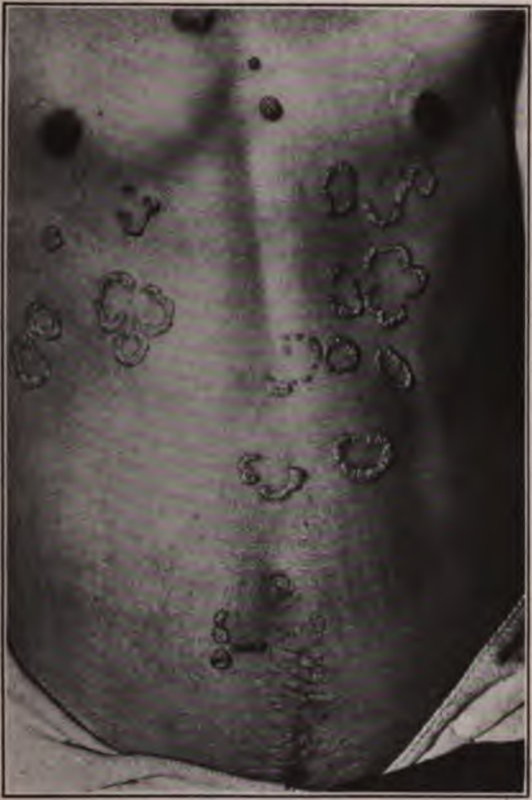
[1] MM. Hallopeau et P. Salmon: Psoriasis palmaire avec soulèvements d'apparence bulleuse, Bull. de la Soc. Fran., 1908, p. 243.

In a patient subject to psoriasis, a local irritation, such as a pin-scratch or a mustard plaster, may cause new lesions to appear at the site of the irritation. Crocker[3] describes a form of psoriasis punctata in which the lesions, though numerous, are limited to the sweat-ducts; and another form of punctate psoriasis in which the papules are situated about the hair-follicles.

Fig. 92



Annular lesions of psoriasis.

The amount of scaling varies greatly in different persons and in the same individual. Ordinarily, the scales are abundant and thickly heaped up over even small areas; sometimes they are sparse over large areas. Free perspiration, friction by the clothing, or frequent bathing may prevent the accumulation of scales on areas where they would otherwise be abundant. Where the epidermis is thin, the scaling is less; therefore, over flexor surfaces, near the mucous orifices, and on the back of the hand the scaling is less than over the extensor surfaces and other regions. The scaling is more pronounced in advanced years. The scales may adhere with considerable firmness to the patch, or may be shed freely from the surface, in pronounced cases powdering the clothing of the patient or the sheets of the bed upon which he reposes at night. As a rule, the scales are disposed over the entire patch, extending slightly beyond the margin.

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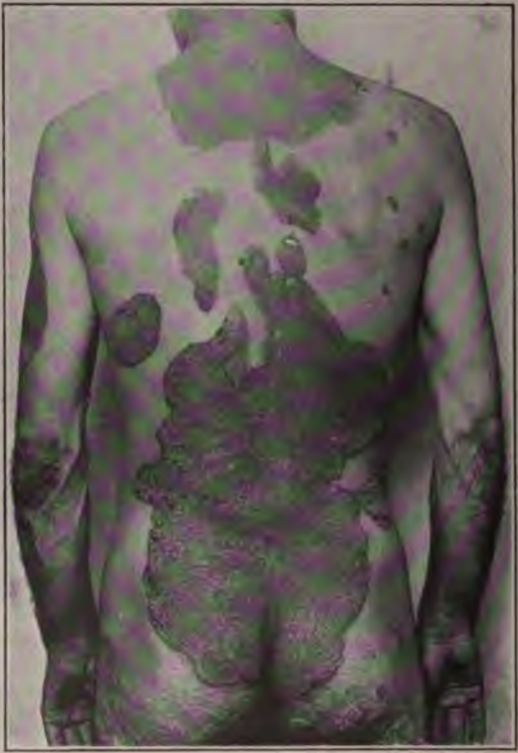
[1] Sée, Annales, 1903, iv, 219; and Oppenheim, ibid., 1905, s. iv, vi, p. 379.

[2] Archiv, 1899, xlvi, p. 433.

[3] Diseases of the Skin, 3d ed., p. 361.

FIG. 93



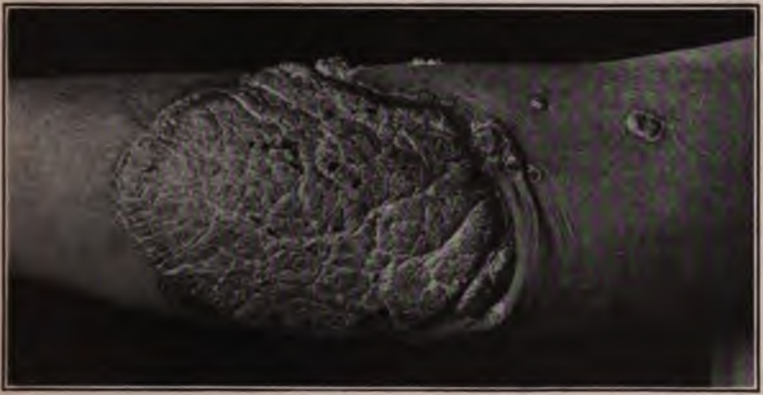
Psoriasis in large plaques.

Instead of a lustrous white, the scales may display a deep yellowish shade; and instead of being imbricated they may form a continuous sheet of exfoliated epidermis. When the eruption is disappearing, the scales fall, leaving a pigmented or slightly discolored patch of integument.

Psoriasis is essentially a chronic disease, but may present at times acute exacerbation, and occasionally begin as an acute process. In the acute stages the inflammatory symptoms are more marked, and the lesions are of a brighter red color, and not so sharply defined as in the ordinary forms of the disease. The scales are fewer in number, thin and easily detached, and the sensations of burning and itching may be severe. When acute, the papules are usually numerous and punctate, and may appear on the face. In other instances, the patches may be as large as a small saucer; are dark- or lurid-red over the whole area; and are covered with a more uniformly constituted, thin, squamous film or sheet of semitransparent, delicate membrane, through which the red glare of the patch beneath is visible. This condition may be seen also in young persons to whom arsenic has been administered for the relief of the disease, with the production of irritative effects. An acute attack may come and go as such, but usually it terminates in a chronic form of the disease.

Subjective sensations may be entirely absent in psoriasis, even when it is extensive. There is, however, usually slight, but occasionally severe, itching. In acute cases burning and smarting are often present. In exceptional cases the subjective sensations interfere with sleep and rest; otherwise, the disease does not affect the general health of the patient.

FIG. 94



Rupioid psoriasis.

Atypical and complicated forms of psoriasis[1] occur in which the character of the lesions is modified considerably. Rarely the scales may be heaped up in the center in the form of an oyster shell, producing what is termed *psoriasis rupioides* or *psoriasis ostreacea*. In a few instances, the accumulated scales have assumed the appearance of a cutaneous horn.[2] Occasionally, sufficient thickening occurs to produce a wart-like appearance. These cases are termed *psoriasis verrucosa* (Besnier, Kaposi, and Crocker). The scales may be slightly greasy and the surface beneath exhibit a trace of moisture, making the diagnosis between psoriasis and dermatitis seborrhoica difficult if not impossible. Indeed, the two conditions may be associated. Occasionally, in moist situations, on the sensitive skin of children, or as an effect of local irritation or infection, the patches may be acutely inflamed and indistinguishable from ordinary eczema.

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[1] Beyer, Wien. klin. Wochenschrift, 1901, xiv, p. 805; a review of the subject, with classification of reported cases.

[2] Gassmann, Archiv, 1897, xli, p. 357.

There can be no question that intermediate forms between eczema and psoriasis occur, in which forms it is difficult to determine whether the two disorders coexist, or the one has assumed the features of the other. In these cases there may be itching and infiltration of the skin, with vesicular and other lesions foreign to psoriasis, and a catarrhal discharge.[1]

Cavafy,[2] Kuznitsky,[3] and others report cases in which psoriatic lesions, though numerous, were limited to one side of the body.

The sequelæ of psoriasis are, as a rule, nothing more than transitory pigmentation, but cases are reported in which involution of the lesions has been followed by superficial scars (Crocker, Hutchinson), keloid formations (Anderson, Purdon, Crocker), persistent, deep pigmentation (Crocker), or permanent achromia (Hallopeau et Gasne,[4] and Rille[5]). In some cases these unusual sequelæ were due, undoubtedly, to the treatment. A few instances have been reported by J. C. White,[6] Hartzell,[7] Schamberg,[8] and others in which epithelioma has followed verrucous lesions which had developed upon psoriatic patches. Some, possibly all, of such changes were due, as suggested by Hartzell, to previous long-continued use of arsenic for psoriasis. The author recently studied a case of this type, in which several epitheliomata occurred on verrucous lesions in a patient suffering with psoriasis, who had taken arsenic for a long period of time.

When extensive, and especially after persisting for a number of years without amelioration, psoriasis may lose its distinguishing features and assume all the characteristics, both clinical and pathological, of dermatitis exfoliativa.

Etiology.—The cause of psoriasis is not known. Sex, social condition, and occupation evidently play little or no part in the etiology. The disease is common, comprising about 4 per cent. of all cutaneous affections reported in America. It occurs most frequently in the second and third decades of life, but no age is exempt. It is unusual for the first attack to appear after forty-five, and the disease is uncommon under ten, and rare under three, years of age. Rille[9] reported a case in which the disease appeared in an infant six days old. Other cases in infants less than one year old have been reported by Neumann, Kaposi, and others.

Heredity is seemingly a factor in a considerable number of cases, in so far as inherited predisposition is concerned; but direct transmission of the disease itself by inheritance has not been demonstrated. Several careful observers believe that the disease is often hereditary. We have seen several families in which the disorder was present in three generations. A family history of psoriasis, however, is the exception rather than the rule. Engman's[1] report of a psoriasis family-tree is interesting in this connection. Knowles[2] found only six family cases in hundreds of cases examined, and concludes that psoriasis is not hereditary. It is of great rarity in the dark-skinned races. Such cases are reported by Dade,[3] Winfield, H. Fox[4] and others.

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[1] Benassi: A description of the causes of moist forms of psoriasis. Giorn. ital., 1901, xxxvi, p. 427; abstr. in Monatshefte, 1901, xxxiii, p. 460.

[2] Cited by Crocker.

[3] Archiv, 1897, xxxviii, p. 405.

[4] Annales, 1898, s. iii, ix, p. 690.

[5] Ibid., 1901, s. iv, ii, p.80.

[6] Amer. Jour. Med. Sci., 1885, lxxxix, p. 163.

[7] Ibid., 1899, cxviii, p. 265.

[8] Jour. Cut. Dis., 1907, xxv, p. 26; ibid., 1909, xxvii, p. 130.

[9] Jour. Mal. Cut., 1890, xi, p. 385.

An early conception of the cause of psoriasis was that it was a cutaneous manifestation of some humor. Gout and rheumatism played an important part in this hypothesis. But modern observers, in view of the rarity of the association of psoriasis with these disorders, give them a small place in the etiology.

The disease apparently bears no definite relation to any one systemic condition. It appears in individuals who are apparently in perfect health, as well as in the delicate and those ill of other disorders. Defective assimilation and elimination, such as exist in gout, rheumatism, and other arthritic disorders, as well as in plethoric and over-fed individuals, exercise an unfavorable influence on psoriasis. Associated with such conditions, psoriasis is usually indolent in type, but exceedingly persistent, unless the systemic condition is improved.

Metabolic disturbances have been attributed as a cause of psoriasis by a number of observers. Johnston and Schwartz,[5] in a careful study, found no recognizable disturbances in the nitrogen metabolism in cases of psoriasis. Brocq and Ayrignac,[6] in a careful study of the urine in psoriatic subjects, found no constant results. Schamberg,[7] on the contrary, reported a remarkable instance of nitrogen retention in the studies of metabolism carried out in his experimental work. So striking was this example that further investigation is being made.

In the neurotic and poorly nourished, psoriasis is also persistent, but usually with more acute symptoms. The disease has been attributed to fright, shock, and other neurotic conditions. Acute toxemias of various origins have been followed by an outbreak in individuals predisposed to the disease. Winfield[8] reported a group of six cases following tonsillar infections. Pollitzer,[9] in his study of the etiology of psoriasis (an excellent review of existing theories), concludes that rheumatism, gout, neuroses, and heredity are not direct etiological factors in the production of psoriasis, but in the present state of our knowledge it can neither be denied nor affirmed that they may have some bearing on the obscure conditions of the system which render it more or less susceptible to this especial infection.

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[1] Jour. Cut. Dis., 1913, xxxi, p. 559.

[2] Jour. Amer. Med. Assoc., 1912, lix, No. 6, p. 415; abstr. Jour. Cut. Dis., 1913, xxxi, p. 57.

[3] Jour. Cut. Dis., 1909, xxvii, p. 207.

[4] Ibid., 1916, xxxiv, p. 847.

[5] Trans. VI. Internat. Derm. Congress, 1907, p. 862.

[6] Annales, 1906, s. iv, vii, pp. 433-460.

[7] Research Studies in Psoriasis: A Preliminary Report, by J. F. Schamberg, John A. Kolmer, A. I. Ringer and G. W. Raiziss, Ph.D., Jour. Cut. Dis., 1913, xxxi, pp. 698, 802.

[8] Jour. Cut. Dis., 1916, xxxiv, p. 441.

[9] Ibid., 1909, xxvii, p. 483.

Polotebnoff[1] is a strong supporter of the nervous origin of psoriasis. He believes that psoriasis is one of the multiple symptoms of a vasomotor neurosis, in which the disturbances in the circulation, just as they occur in various organs of the body, sometimes extend to the skin. The neuropathic hypothesis has many supporters.[2] Weyl[3] believed that psoriasis was due to inherited weakness of the nerve-centers that regulate the nutrition of the skin, and that the cutaneous lesions were the superficial expressions of a central disturbance.

The parasitic theory of psoriasis is strongly suggested in many ways. Syphilis and tuberculosis have both been held responsible for the production of psoriasis. Erasmus Wilson, followed later by R. W. Taylor, believed psoriasis to be due to an attenuated syphilis, and recently Ravogli[4] states his belief that psoriasis is of nervous origin, the underlying cause of the nervous changes being an extinguished syphilis. Menzer[5] believes psoriasis to be a cutaneous manifestation of a latent tuberculosis. Schoenfeld[6] and Huebner[7] made studies similar to that of Menzer, but could find no connection whatever between tuberculosis and psoriasis. Sellei[8] describes sharply outlined, circular or ovoid bodies found deeply in the skin in cases of psoriasis. He regards these bodies as the probable exciting cause of psoriasis.

Pollitzer[9] believes that psoriasis is most probably due to an external microbic infectious agent. Schamberg,[10] in his study of the question of the parasitism of psoriasis, arrives at no definite conclusion as to the parasitic cause of the disease. In his research studies in psoriasis[11] he states that an unidentified diplococcus *X* was found in five psoriatic lesions and in one blood-culture, and is deserving of further study; also, that an ultramicroscopic, mobile, bacillary body was discovered in seventeen out of nineteen cases of psoriasis. These bodies, also, are being further studied. Aside from these two, the other parasites cultivated from psoriatic lesions were discarded. Probably the earliest writer to discuss this phase of the subject was Lang,[12] who described a fungus, which he named *epidermophyton*, that he believed to be the cause of psoriasis. His findings were confirmed by Wolff[13] and Ecklund, but were later rejected by Neisser and others, including Ries,[1] who found the bodies described by Lang to be artificial products and not spores.

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[1] Derm. Studien (edited by Unna), Hamburg, 1891, Series 2, vol. v, p. 347.

[2] Weyl and others, including Besnier, Polotebnoff, Bourdillon, and Kuznitsky, Archiv, 1897, xxxviii, p. 405.

[3] Ziemssen's Handbook, 1885, p. 247.

[4] Jour. Cut. Dis., 1913, xxxi, p. 250.

[5] Deutsche med. Wochenschrift, 1912, xxviii, No. 45, p. 2119; abstr. Jour. Cut. Dis., 1913, xxxi, p. 127; and ibid., 1913, xxxix, No. 33, p. 1599, abstr. ibid., p. 1055.

[6] Deutsche med. Wochenschrift, 1913, xxxix, No. 30, p. 1446; abstr. Jour. Cut. Dis., 1913, xxxi, p. 1054.

[7] Ibid., 1913, xxxix, No. 11, p. 505; abstr. ibid., p. 528.

[8] Wien. klin. Wochenschrift, 1910, No. 29, p. 1075; abstr. Jour. Cut. Dis., 1911, xxix, p. 363.

[9] Loc. cit.

[10] Jour. Cut. Dis., 1909, xxvii, pp. 496–512.

[11] Loc. cit.

[12] Vierteljahr, 1879, p. 257.

[13] Vierteljahr, 1884, p. 337; abst. Annales, 1885, vol. vi, p. 305.

Many attempts to transmit the disease by direct inoculation have failed, but Bestot[2] apparently succeeded in inoculating himself from an infant who had vaccinal psoriasis.

The fact that psoriasis frequently has followed vaccination[3] and other local injuries of the skin has been held by some as an argument in favor of the parasitic origin of the disease. Serkowski and Wisnewski[4] have recently described ultramicroscopic organisms, resembling the Paschen corpuscles of variola, in psoriasis. Successful inoculations with these organisms are recorded. It has long been known that in psoriatic patients lesions may be developed artificially along the lines of mechanical irritation. In this way, figures in the shape of anchors, crosses, hearts, and many others, have been produced on the skin of psoriatic patients, one of whom has been ingeniously photographed by Fox,[5] of New York.

The distribution of psoriatic lesions suggests that the disease may be influenced by the exclusion of sunlight from those portions of the body covered with the clothing and the hair. Certain it is that in exceptional cases only are the hands involved or is the face attacked at a distance from the line of the hairs upon the brow and bearded region. It is likewise true that after exposure of the affected areas to abundant sunlight, not only when patients are treated intentionally by such exposures of the nude body to light in hospitals and in private practice, but in occupations which necessitate the same, beneficial results often are marked.

In winter and in cold countries psoriasis is much more prevalent than in warmer seasons and climates. Kayser[6] reports that in the tropics the subjects of psoriasis are few, and the symptoms of the disease, when they develop at all, are rudimentary, the typical eruption being scarcely ever produced.

Pathology.[7]—The pathogenesis of psoriasis is an unsettled question, and while the many observers of the histological changes agree as to findings, their interpretation of these differs. Some believe the initial changes occur in the epidermis, while others are of the opinion that the corium is first involved. The chief changes noted in the epidermis are hyperkeratosis and parakeratosis in the stratum corneum, and acanthosis in the rete. In the early lesions the scales are convex and thickest in the center, contain air spaces and are arranged in layers. Migratory leukocytes are found which frequently collect and form microscopic abscesses (Munroe). The rete-pegs are elongated and thickened and there occurs a moderate amount of intracellular edema. The portion of the rete overlying the papillæ is not thickened, and may be thinner than normal, this accounting for the ease with which bleeding occurs in early lesions when the scale is removed.

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[1] Vierteljahr, 1888, xv, pp. 521, 685, 871 (a review of previous reports on the pathology of psoriasis, with bibliography).

[2] Annales, 1901, s. iv, ii, p. 337 (review of the case by Hallopeau, with discussion).

[3] Weinstein, Brit. Med. Jour., 1902, i, p. 271 (*résumé* of 24 cases); also Rioblanc, Monatshefte, February, 1896, xxii, p. 195. Lane, Jour. Cut. Dis., 1916, xxxiv, p. 201; A Case of Postvaccinal Psoriasis.
Monatshefte, February, 1896, xxii, p. 195.

[4] Nowinylek, 1913, Bd. xxv; abstr. Archiv, 1914, cxvii, p. 673.

[5] Photographic Illustrations of Cutaneous Diseases, New York.

[6] Geneeskumdig Tijdschrift vor Nederlandsch-Indie, 1907, xlvii, fasc. 5.

[7] Heimann: Jour. Cut. Dis., 1916, xxxiv, p. 204. A histological study and review of the work of others, with bibliography.

In the corium there occurs vascular dilatation with swelling of the endothelial lining of the vessels, and perivascular cellular infiltration consisting of leukocytes and fibroblasts. The papillæ are elongated, broadened and at times clubbed, fitting the interspaces of the rete-pegs. The collagen is edematous, but the elastin is not affected.

Diagnosis.—The recognition of a pronounced case of psoriasis is made with ease. As usual, it is the atypical form of the eruption that awakens doubt. The diagnostic features of the common type are summarized in the first paragraph, under the heading of symptoms.

Eczema.—Eczema elects the anterior surfaces of the body, the neighborhood of the mucous outlets, the flexor surfaces of the joints and limbs, the crevices, folds, pockets, depressions, and protected angles of the skin. Psoriasis elects the posterior surfaces of the body, avoids the vicinity of mucous outlets, and occurs over the extensor aspects of the joints and extremities, especially about the knee and elbow. Both disorders occur in the scalp. Eczema commonly spreads downward over the face, involving the nose, lips, chin, and region back of the ear. Psoriasis ordinarily involves only the upper part of the forehead.

In individual patches eczema will be recognized by its severe itching; by the scratching it excites; by the history of moisture, discharge, and crusting; by its ill-defined outline; by its asymmetrical disposition, except upon the similarly irritated hands and feet; and by the fewer, more yellowish, smaller, and less lustrous scales which characterize its squamous varieties. In squamous eczema, moreover, the areas are, as a rule, larger, more irregular in shape, fewer in number, and the less perfectly defined outline does not show the small, round plaques which unite to form the larger psoriatic areas.

Dermatitis Seborrhoica.—In seborrhoic dermatitis, the scales are smaller, greasy, and less abundant; the surface beneath is moist or oily, shows no bleeding points, and is less reddened than in psoriasis. The lesions are most numerous on the scalp, over the sternum, and between the scapulæ, and are rarely found on the elbows and knees. From the scalp the disease spreads by choice down behind the ears and over the forehead. A fringe may be formed similar to that which occurs in psoriasis. The individual lesions and differences in scaling are indicated above.

Syphilis.—Psoriasis in many cases greatly resembles the squamous and papulo-squamous syphilides. In syphilis the greatest aid will be obtained by a history of infection, adenopathy, and mucous patches; and in women abortions, miscarriages, and stillbirths. Psoriasis is a singularly uniform disease; syphilis is decidedly multiform in its manifestations. Syphilitic patches are less symmetrical, more elevated at the edge, and the scales with which they are covered are fewer, smaller, dirty-yellowish rather than lustrous in color, and are apt to form a collarette about the base of the lesion instead of occurring in an imbricated manner over the margin. Their circular outline is often abruptly broken by gaps, thus producing semilunar and small arc-shaped segments. In syphilis the eruption is less generalized, and shares with other syphilodermata the brownish and purplish hues of the skin beneath; and the base of the syphilitic lesion is indurated. The scales of many of the syphilides which resemble psoriasis partake of the character of crusts, being agglutinated by exudations from the patch. They are only occasionally squamous as in psoriasis. The squamous syphiloderm of the palms and soles often occurs only in these localities. Psoriasis is extremely rare in such situations, and is seldom limited to these regions exclusively. A psoriasiform circle limited to the region of the mouth, nose or chin will generally prove to be syphilitic. The disease which has for a long time persisted in the production of squamous patches can generally be demonstrated to be psoriasis, as syphilis changes its type in the course of months.

Pityriasis Rosea.—In this disease the patches occur most commonly on the trunk, rarely on the arms, and practically never in the scalp. The patches are more oval than circular, the scales are finer, and on their removal no bleeding points are seen. The center of the patch is usually tawny or salmon-colored. The disease is much more superficial, less inflammatory, and more rapid in its career than psoriasis. Complete involution is accomplished usually in a few weeks, and recurrences are rare.

Lichen Planus.—The primary lesions in lichen planus are minute, flat, angular papules, which as individuals rarely become as large as the cross-section of a small pea. The larger areas are formed always by grouping and coalescence of small papules, whereas similar areas in psoriasis are produced by peripheral extension of the early papule. Instead of presenting distinct scales, the lichen planus papule is covered with a thin, horny layer, giving the papule a glazed or varnished appearance. There is a tendency to linear arrangement of the lesions, and when these coalesce to form larger areas the latter are commonly linear or angular in outline. The larger papules and patches in lichen planus have a characteristic purplish or violaceous hue, which never is seen perfectly in psoriasis. The favorite sites of lichen planus are the flexor surfaces of the wrists and forearm and the legs above the ankle. It is rarely conspicuous on the elbows and knees and other sites of predilection of psoriasis.

Pityriasis Rubra Pilaris (*Lichen ruber acuminatus*) is a comparatively rare disorder, and has for primary lesions fine, pointed, scale-capped papules, which do not enlarge peripherally, but form larger areas slowly by the coalescence of many small papules, some of which can be demonstrated at the borders of large areas. The characteristic circular areas and typical scales of psoriasis are wanting, and there is thickest in the center, contain air spaces and are arranged in layers. Migratory leukocytes are found which frequently collect and form microscopic abscesses (Munroe). The rete-pegs are elongated and thickened and there occurs a moderate amount of intracellular edema. The portion of the rete overlying the papillæ is not thickened, and may be thinner than normal, this accounting for the ease with which bleeding occurs in early lesions when the scale is removed.

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When defective elimination is demonstrable, or in plethoric or rheumatic patients, the administration of alkalies, such as liquor potassæ, potassium citrate or acetate or sodium bicarbonate, taken with large quantities of water three times a day, after meals, is recommended. When associated with gout, Robinson advises:

℞—Vin. colchici,	ʒij;	8	
Spts. ether. nit.,	℥ss;	15	
Potass. acetat.,	℥j;	30	
Syr. aurantii,	℥iss;	45	M.

Sig.—A dessertspoonful three times daily in water after meals.

Winfield[1] reported the successful treatment of psoriasis with colonic irrigation and the internal administration of lactic acid.

Local Treatment.—The successful operation of various medicinal substances employed requires the removal of the scales. This may be accomplished by applying oil, glycerin, or vaseline, after which the scales may be washed off with soap and water. In some instances a general alkaline bath may be used for similar purposes. The preparation of most value in the local treatment of psoriasis is chrysarobin, first recommended for this disorder by Squire of London in 1878. The drug may be applied in strengths varying from 2 to 40 grains (0.13 to 2.60) to the ounce (30.) of ointment, paste, collodion or traumaticin, or it may be incorporated in plaster. It is occasionally used in greater strength for localized inveterate areas. It commonly produces a dermatitis which subsides within a few days. The plan followed by us is to have the chrysarobin applied daily until a slight erythema is detected at the edge of the brownish discoloration produced by the application. Its use is then suspended until the reaction has subsided. As a rule, the application may be repeated on five successive days. In some instances a single application produces a reaction, while in others ten or more fail to do so. When the areas are numerous or in large areas chrysarobin is best used in the form of a soft ointment well rubbed into the patches daily. The surplus ointment may be wiped off and the skin covered with a dusting powder. For circumscribed areas chrysarobin may be applied in collodion or traumaticin in a strength of 5 to 10 per cent. After the scales have been thoroughly removed a film of this preparation is applied with a brush or swab and allowed to dry. The application may be removed daily, as above suggested.

An effective combination suggested by Fox is 10 parts each of chrysarobin and salicylic acid, 15 parts of sulphuric ether, and 100 parts of flexible collodion. Besnier suggested making a solution of chrysarobin and chloroform, 20 to 40 grains (1.33 to 2.60) to the ounce (30) which is applied to the patches and after evaporation of the chloroform the adhering powder is covered with a layer of traumaticin or collodion.

Chrysarobin may be mixed with water and applied as a paste, or incorporated in a 50 per cent. aqueous solution of ichthyol and applied as a varnish. The combination suggested by Unna and Dreuw is effective for local, inveterate patches. This formula follows:

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[1] Jour. Amer. Med. Assoc., August 10, 1912, p. 416.

℞—Acid. salicylic.,	ʒiiss;	10	
Chrysarobin,			
Ol. rusci,	āā ʒv;	20	
Saponis mollis,			
Vaseline,	āā ʒvj;	24	M.

The effect of chrysarobin is greatly enhanced by the use of autoserum injections.[1] In 1914 many favorable reports were made concerning this method. In the following year adverse reports came from some of the previous observers as well as others. After five years' constant use we believe the method to be of distinct value in many cases. (For technic Cf. section devoted to General Therapeutics.)

When the patient can be kept in a hospital under direct supervision for a period of three weeks Mook[2] recommends the use of an ointment containing 10 per cent. of chrysarobin and 2 per cent. of phenol. This treatment is only applicable to hospital patients.

The patch of psoriasis undergoing resolution under chrysarobin becomes less scaly and assumes a whitish and normal aspect which contrasts strongly with the deep brown discoloration of the skin at the periphery of the patch. Novorobin, a derivative of chrysarobin which was brought forward by Schamberg, has the advantage of being more active and less disagreeable to use because of the fact that the staining qualities are reduced. Chrysarobin should not be used on the hands, face, or in the scalp except under unusual circumstances.

The tars probably rank next to chrysarobin in the treatment of psoriasis. Owing to their ability to produce undue reactions in susceptible skin it is well to employ them first on a relatively small portion of the affected surface. It is also necessary to leave the preparation on for several hours, as the tars do not produce prompt reactions.

Pix liquida, oleum cadinum or oleum rusci may be used in the form of a salve, 1 dram (4) of either to the ounce (30) of lard, lanolin or petrolatum, or a combination of these. A thin layer of this ointment should be well rubbed into the patches which have been denuded of their scales twice daily. In some European clinics tar is applied after a soft soap bath and the patient is returned to the bath, where he remains for several hours. Green soap, salicylic acid and sulphur are sometimes combined with tar, as follows:

℞—Ol. rosmarin.,	ʒiss;	6	
Glycerin.,	℥j;	30	
Ol. rusci,	℥j;	30	
Saponis mollis,	℥iv;	120	
Alcoholis,	℥viij;	240	M.

Sig.—For external use.

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[1] Gottheil and Satenstein, Trans. Sec. on Derm., Amer. Med. Assoc., 1914, p. 124; Howard Fox, Jour. Cut. Dis., 1915, xxxiii, p. 616; Trimble, Rothwell, Ibid., p. 621; Willock, Jour. Amer. Med. Assoc., July 3, 1915; Ravitch, Ibid., April 10, 1915.

[2] Archiv. of Derm. and Syph., 1920, ii, p. 447. See this article for details.

Wilkinson's salve as modified by Hebra is also used with good effect:

℞—Sulphur sublimat.,				
Ol. rusci (crud. vel. rectif.),	āā	℥ss;	15	
Saponis mollis,				
Adipis,	āā	℥j;	30	
Cret. preparat.,		℈iiss;	3	33 M.

Sig.—Wilkinson's salve, modified.

When applied over the general surface absorption of the tar may take place with the production of general toxic symptoms, which include fever, vomiting diarrhea, strangury, or the elimination of the toxic agent in secretions which are blackened by its presence. The symptoms are usually relieved in from twenty-four to forty-eight hours after the discontinuance of the drug.

Salicylic acid, ammoniated mercury, sulphur and resorcin are employed in cases or areas where chrysarobin or the tars are contraindicated. For the scalp lesions a 10 per cent. ammoniated mercury or mild chlorid of mercury or a 3 to 5 per cent. salicylic acid ointment is recommended. Chrysarobin and tar cannot be employed, as a rule, in this situation. Occasionally sulphur or resorcin may be substituted, especially when the lesions are of the seborrheic type.

In many cases on the glabrous skin it may be necessary to employ some agent other than chrysarobin or tar. An effective combination for moderate sized areas consists of salicylic acid 3 per cent., ammoniated mercury 5 per cent. in petrolatum. Frequently the use of a salicylic acid ointment for a few days in connection with the alkaline baths above suggested is valuable as a preliminary measure before beginning chrysarobin.

Pyrogallol, first suggested as a remedy for psoriasis by Jarisch, is inferior to chrysarobin. It is used in a strength of 10 per cent. in petrolatum. It is less rapid in its action than chrysarobin and does not produced so much discoloration. It is toxic when absorbed from the skin surface.

Beta-naphthol was first used in psoriasis by Kaposi.[1] It may be applied in an ointment in a strength of 15 per cent., or in alcoholic solution. Kaposi originally reported good results following its use. Unlike the above remedial agents, it does not stain the skin, hair or nails.

Crocker advised thymol in ointment, 10 to 30 grains (0.66 to 2.) to the ounce (30.).

For localized patches that have resisted chrysarobin, Blaschko[2] recommends the formula of Rochard:

℞—Iodi pur.,	grs. x;		66
Hydrarg. chlorid. mitis,	grs. xxxj;	1	7
Vaseline, vel adipis,	℥iii–ʒj;	100	M.

A large number of other remedial agents have been suggested in the local treatment, but most of them are inferior to those above enumerated.

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[1] Wien. med. Wochenschrift, xxxi, pp. 617, 641, 681.

[2] Archiv, 1901, lvi, p. 253.

Heimann[1] recommended the use of the Uviol lamp in the treatment of psoriasis.

Radiotherapy is a clean, efficient and most valuable method of local treatment. In the majority of instances psoriatic lesions disappear with more certainty and with much greater rapidity under *x*-rays than with any other local measure. The rays should be employed with great caution, and a dermatitis should not be induced. A few exposures of moderate intensity suffice. A dermatitis may be produced in an area which is the seat of psoriatic lesions by an amount of *x*-rays which, if applied to the normal skin, would produce little or no reaction. Telangiectasia is prone to develop in areas in which an active dermatitis has been induced. Recurrence of lesions takes place after radiotherapy as after other methods of treatment. Great caution is necessary in treating a series of recurrences, especially if the recurrent lesions occupy areas formerly involved. Radiotherapy is not recommended in psoriasis of the scalp on account of the resultant alopecia, and should be the method of choice only in selected cases.

Prognosis.—The permanent relief of a grave case of psoriasis is not insured by any treatment, though hundreds of patients are permanently relieved by even the simplest measures. The disease often recurs, and may do so repeatedly for the greater part of a lifetime. Permanent relief, therefore, should be neither promised nor predicted in any case. Once relieved, it should be the aim to guard against all possible recurrences. It is important to insist that treatment shall be followed until the last lesion has been cleared away. If even a few small areas are left, recurrence will follow more surely and quickly. After relief of any obstinate or recurrent attack, as also in inveterate cases, the prognosis is greatly improved by the removal to a climate suitable for the psoriatic patient.

Parakeratosis Scutularis.—Under this title Unna[2] described an affection characterized by abnormal cornification, affecting the body in areas, and accompanied by superficial inflammation. The lesions are described as occurring on the leg in patches the size of a shilling to a half-crown or larger, as being livid-red in color, and showing special involvement of the hair-follicles. In the follicles are described horny balls, which are early yellowish-red in color, later chalky-white, and which finally run together and form with the interfollicular scales peculiar large, bent shields, one or more of which are placed in the middle of the brownish areas. The under surface of the shield is dotted with a large number of thorn-like horny balls, representing the horny balls drawn from the follicles. On the scalp the hairs are enclosed in bundles of yellowish-white, waxy, horny frills, which run together and form a yellowish-white cap, closely adherent to the head. Weiss,[3] under the title *Parakeratosis ostracea* (*scutularis*), described a case with lesions closely simulating those above described. In this there were scattered over the body-surface discrete, variously shaped, larger and smaller, whitish and yellowish, laminated and raised masses. The resemblance between the rupioid psoriasis of McCall Anderson and this case was striking (see Psoriasis). It is believed by many that the two cases above mentioned are rare and unusual forms of psoriasis.

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[1] Jour. Cut. Dis., 1911, xxix, p. 635.

[2] Internat. Atlas, 1890, Part 3, No. 8; Histopathology, p. 286.

[3] Jour. Amer. Med. Assoc., 1912, lix, p. 343.

PARAPSORIASIS.

Synonym.—Resistant maculo-papular scaly erythroderma.

Fox and MacLeod,[1] in a careful study of a case of parakeratosis variegata and a survey of the literature at that time, classed under the second title above the following disorders: erythrodermie pityriasique en plaques disséminées (Brocq); dermatitis psoriasiformis nodularis (Jadassohn); pityriasis lichenoides chronica (Juliusberg); lichenoid psoriasiform exanthem (Neisser); and parakeratosis variegata (Unna, Pollitzer, Santi). At a somewhat later date, Brocq[2] introduced the term parapsoriasis for this group of diseases, and proposed the three following divisions: *parapsoriasis en gouttes*, *parapsoriasis lichénoïde*, and *parapsoriasis en plaques*. This classification includes the various manifestations, and presents a clear conception of the group.

Definition.—Parapsoriasis is a rare disease of the skin characterized by persistent, red, scaling patches or lichen-planus-like lesions, devoid of subjective sensations, and resistant to therapeutic measures. The disease, as described in individual cases, varies as to type. A particular study of the disorder has been made by J. C. White,[3] C. J. White,[4] Corlett and Schultz,[5] Anthony,[6] Sir Malcolm Morris and Dore,[7] Sutton,[8] and others in addition to the authors above noted.[9]

Symptoms.—In all varieties the persistence of the lesions in spite of treatment is characteristic. The lesions occur chiefly on the trunk and limbs. The inflammatory process is superficial and devoid of infiltration and of subjective sensations. The primary lesion is a macule or maculo-papule, often scale-covered, which spreads peripherally. New lesions slowly but surely appear, until larger areas become involved. In this way the guttate, retiform, and patchy varieties are produced. A true conception of the disease cannot be had from observation of a single case. The individual cases differ from each other in a striking manner. The particular features of each must therefore be depicted. At present it cannot be stated which variety occurs most commonly.

Guttate Variety (*Parapsoriasis en gouttes* (*Brocq*), *Dermatitis psoriasiformis nodularis* (*Jadassohn*), *Pityriasis lichenoides chronica* (*Julius-*

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[1] Brit. Jour. Derm., 1901, xiii, pp. 319–346, inc.

[2] Annales, 1902, s. iv, iii, p. 433.

[3] Jour. Cut. Dis., 1900, xviii, p. 536.

[4] Ibid., 1903, xxi, p. 153.

[5] Ibid., 1909, xxvii, p. 49 (literature).

[6] Ibid., 1906, xxiv, p. 455.

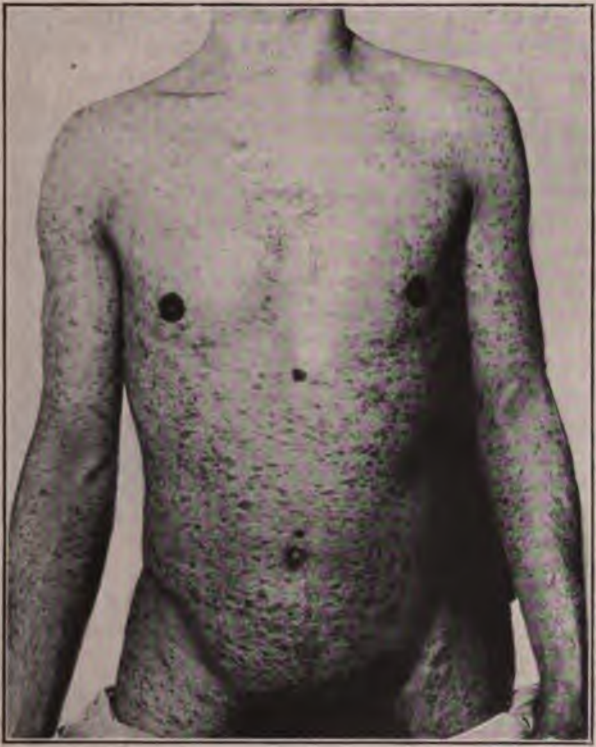
[7] Brit. Jour. Derm., 1910, xxii, p. 249.

[8] Ibid., 1913, xxv, p. 115.

[9] A number of cases representing the different types of the disorder have been shown before the various dermatological societies of this country and in England during the past few years.

berg).[1]—In this variety a close resemblance to psoriasis is noted. Again, at times, a scaling syphiloderm is simulated. The eruption is very superficial, consisting of pinhead- to pea-sized papules, round or oval in form, and of an intense, clear-red color. The larger are paler, well-defined, and flat, with an occasional central depression. The smaller are slightly pointed. Scaling may be quite perceptible, or the papules may be devoid of scales. The lesions are somewhat firm, and some are follicular. The scratched lesion is red and bleeds but little. The scale, when removed, is found to be thicker in the center than at the periphery. New papules or nodules appear here and there as the eruption gradually increases. In the beginning there is an areola of redness. No subjective sensations are present, and involution of the lesions is uncommon. The hands, face, and scalp are usually free from attack.

FIG. 95



Parapsoriasis lichenoides.

In a patient presenting this type, studied for some years by the author, the primary lesion was a red papule, scale-covered at all points. As the lesion enlarged, in this particular instance, it assumed a darker hue. The sites of election were the trunk and limbs. The patches were from pea- to dime-sized, and remained unchanged for long periods of time. At varying intervals, new lesions would develop, being light-red at first, later assuming the above mentioned characteristics. The lesions were but little influenced by treatment.

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[1] Stokes, Jour. Cut. Dis., 1916, xxxiv, p. 343, Clinical and Experimental Observations on a Case of Pityriasis Lichenoides Chronica (Juliusberg); Wallhauser, Ibid., 1919, xxxvii, p. 763, Parapsoriasis Lichenoides Limaris, report of an unusual case.

Pick,[1] Civatte,[2] and Milian and Pinard[3] have reported cases of this disorder which, on microscopical examination, were found to be tuberculides. Civatte surmises that they are all of this nature.

Retiform Variety (*Parapsoriasis lichénoïde* (*Brocq*), *Parakeratosis variegata* (*Unna, Pollitzer, Santi*), *Lichen variegatus* (*Crocker*).—In this variety the eruption is more generalized than in either of the other forms, and is represented by lesions that may be described as intermediate between those of lichen planus and psoriasis. The subjects of this disorder have been adults in the third and fourth decades of life. The eruption is usually generally distributed over the trunk and extremities and is retiform in character, almost as though the patient were covered with a net. This peculiar appearance is induced by hyperemia occurring in the form of a patchy network, enclosing areas of a less intense hue. The primary lesion is a reddish-yellow or darker colored, flat-topped, scale-covered papule. The lesions on coalescing produce the peculiar picture above described. The extremities may be the seat of a more marked dermatitis, with increased scaling, and also with more distinct papulation, the papules being flat-topped, shining, lichen-planus-like. In a few recorded cases, the premycotic stage of mycosis fungoides has been mistaken for this eruption.[4] One such case came under the observation of the author. This patient for some years was considered by experts to be suffering with parapsoriasis of the retiform type, but subsequently developed true mycosis fungoides, to which he later succumbed. Wise and Rosen[5] recently recorded a similar case. This form of parapsoriasis is resistant to treatment.

Parapsoriasis en Plaques (Brocq).—The cases described under the title of *Erythrodermie pityriasique en plaques disséminées* and *Xanthoerythrodermie perstans* by J. C. White, Crocker and others belong to this group. In this variety plaques or patches of varying size and irregular shape occur on the trunk and limbs. The patches are well defined, range in size from that of a dime to a walnut or larger, and present varying shades of red, reddish- or yellowish-brown or fawn-color, and at times a darker brown. In certain cases a seborrhoic dermatitis is simulated. Moderate scaling is usually present, and the scales are small and adherent. In certain instances visible scaling is absent. On pressure the color may all be removed. There is no elevation of the lesion, no infiltration of the skin, and subjective sensations are absent. The lesions are very persistent and show practically no tendency to undergo spontaneous involution, but are clearly resistant to treatment. The patches sometimes are so delicate as to appear like a mere stain.

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[1] Archiv, lxix, p. 411.

[2] Annales, 1906, p. 209 (Brocq's clinic).

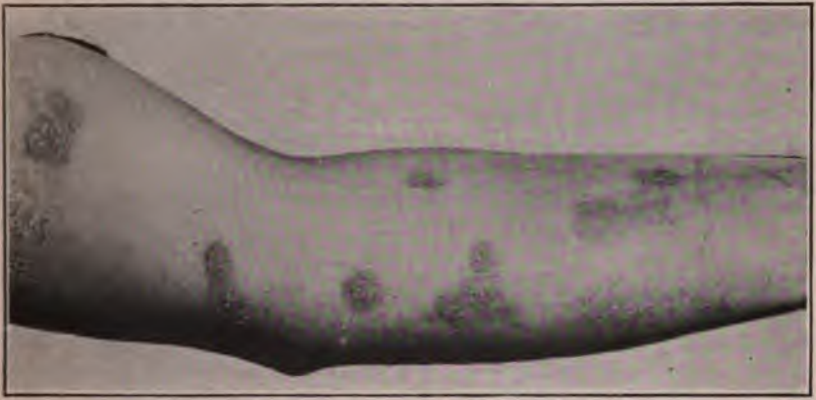
[3] Annales, 1907, s. iv, viii, p. 477.

[4] Jamieson, Jour. Cut. Dis., 1901, p. 440; Hudelot and Gastou, Annales, 1904, s. iv, v, p. 1090; and also the first case of Unna.

[5] Jour. Cut. Dis., 1916, xxxiv, p. 95; A Case of Early Mycosis Fungoides Clinically Indistinguishable from Parapsoriasis of Brocq.

Etiology.—The cause of parapsoriasis is unknown. It is essentially a disease of adult life, though one patient studied for some years by the author, developed the disease at the age of twelve years, and at the present time is suffering with active manifestations of the disorder, with but little change after sixteen years' duration. Exposure to excessive heat has been given as a factor in some cases. Men are more frequently attacked than women. There appears to be no connection between psoriasis or lichen planus and this disorder.

Fig. 96



Parapsoriasis in plaques. (Brocq.)

Diagnosis.—Parapsoriasis is to be differentiated from dermatitis seborrhoica, psoriasis, lichen planus, the maculo-papular syphiloderm, and the premycotic stage of mycosis fungoides. By taking into consideration the chief characteristics of this disorder as described heretofore—the persistence of its lesions in spite of treatment, their superficial character (being devoid of infiltration and subjective sensations), their gradual evolution, their slow spread, and their location—it may be recognized.

Pathology.—The pathogenesis of the disorder is unknown. C. J. White[1] suggests that the disease may be due to involvement of the deeper arterial twigs, producing a local ischemia, in consequence of which the other changes described occur. The histology, as summarized by Fox and MacLeod, is as follows: In the corium is found a superficial inflammatory process, confined to the papillary and subpapillary layers, with dilatation of capillaries, edema, and cellular infiltration.

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[1] Loc. cit.

The epidermis shows interepithelial edema and defective cornification. In the prickle-cells is found a moderate parenchymatous edema. Nuclei are present in the stratum corneum. No necrosis is noted, and the prickle-cells appear healthy. Corlett and Schultz,[1] in their careful histological study, call special attention to the vascular involvement, which they consider the essential change and the cause of the other abnormal histological findings.

Treatment.—A few cases have undergone spontaneous involution, the remainder have been resistant to treatment. All sorts of local and internal treatment have been tried, but no method has been proved successful. In the plaque variety we have obtained temporary results with radiotherapy. Internally, arsenic has been given a good trial, and the salicylates and salicin have been used. Locally, the stimulating treatment recommended for psoriasis may be used in the guttate and small-patch varieties. In the widely distributed cases, stimulating preparations are not recommended. Milder measures, such as are recommended for a slight degree of dermatitis, should be employed.

Prognosis.—As indicated above, the prognosis is unfavorable as to relief of the lesions, but good so far as the general health of the patient is concerned. In a few instances the plaque variety has been relieved, but as a rule the eruption is unaffected by any method of treatment.

PITYRIASIS ROSEA.

Synonyms.—Pityriasis maculata et circinata, Herpes tonsurans maculosus, Pityriasis circinata. Fr., *Pityriasis rosé de Gibert, Pityriasis circiñé et marginé, Pityriasis disséminé, Pityriasis rubra aigu, Roseole squameuse* (Chapard).

Definition.—Pityriasis rosea is an acute, rarely chronic, self-limited, eruptive disease, characterized by superficial scaling patches of varying size, usually round, oval, or circinate in outline, having a pale-red hue, with often a fawn-colored center, and situated chiefly on the trunk.

This disorder was first recognized and described by Gibert,[2] and later by Bazin,[3] Horand, Duhring,[4] and others.[5]

The subjects are commonly young adults, but the disease is seen in children and those in middle life, of both sexes. The outbreak of the malady, in exceptional cases, may be preceded for a variable time by languor, lassitude, inappetence, or a feeling of chilliness. Occasionally, the first noticeable symptom is the occurrence of mild fever, the body temperature rarely rising above 102° F. There may be slight swelling of the submaxillary or cervical glands. General adenopathy has been reported. In acute cases there may be distinct congestion of the fauces. In large numbers of cases, however, no constitutional symptoms are present.

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[1] Loc. cit.

[2] Traité pratique des Maladies de la Peau, Paris, 1860, i, p. 402.

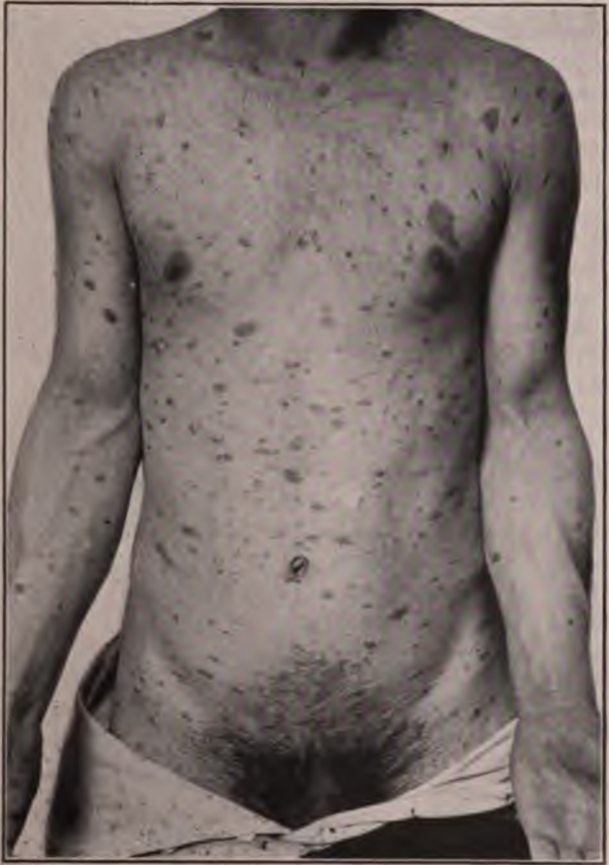
[3] Affections génériques de la Peau, Paris, 1862, p. 365.

[4] Am. Jour. Med. Sc., 1880, lxxx, p. 359.

[5] Moingeard, Thèse de Paris, 1889; Chapard, T., ibid., 1885; C. Colcott Fox, Lancet, London, 1884, ii, p. 485; Thibierge, La Pratique dermatologique, 1902, iii, p. 894 (with colored plate).

In some cases, Brocq[1] believes in all, the general outbreak is preceded for a week or ten days by a single lesion, situated usually at the side of the trunk. This "primary lesion" may often be recognized as the largest, most conspicuous, and most brilliant in hue of all the patches which later develop. The eruption often escapes recognition for a time after its appearance, on account of its sparseness or the trifling degree of itching it arouses. When fully developed, it is characterized by the conspicuous appearance over large surfaces of the trunk, especially upon the integument covering the clavicles, the ribs, and the scapulæ, less often on the exposed face and hands, of numerous pinhead-

FIG. 97



Pityriasis rosea showing the "herald spot" above the left nipple.

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[1] Annales, 1887, s. ii, viii, p. 615.

to small-coin-sized or larger, circumscribed, roundish or oval-shaped, slightly elevated, macular or maculo-papular lesions, which are fitly designated by Thibierge as "médaillons." These lesions may be discrete, closely set, or confluent, and instead of being elevated may be either on a level with the general surface or slightly depressed, with an annular border. They are dry, covered with furfuraceous, rather adherent scales, and vary in color from a yellow or tawny (chamois-skin shade) to a deep red. The infiltration is slight, and the patch is situated superficially. Itching is commonly inconspicuous among the symptoms, but in acute and extensive cases may be severe.

The fully-formed disks vary in long diameter from the width of a finger-nail to three or four centimeters or more. G. H. Fox[1] emphasizes the fact that extensive marginate patches may be formed by confluence of smaller areas. The oval contour is that more often recognized as characteristic of a well-developed lesion, the long axis of the disk usually corresponding with the lines of cleavage, and the terminal extremities of the oval being slightly frayed by the irregularity with which the fine, branny scales are there disposed. The patch frequently has a tawny salmon shade, which is characteristic of the disease, and enlarges by peripheral extension, leaving a relatively clear center. The scales often have a silvery-grayish color. The eruption may be fairly well generalized, but the face and other exposed parts of the body commonly escape, though the scalp may be involved. In the latter event, the hairs are unaffected. The evolution of the eruption may be by successive development of the eruptive elements at intervals for one to ten weeks or longer.

The variations exhibited by the exanthem in this affection are distinct, but are scarcely ever sufficient to mask the characteristic appearance of the oval or circular plaques over the neck, the arms, the abdomen, or the extremities; sometimes first appearing over the latter and extending thence to the trunk.

Fox[2] calls attention to the close similarity between pityriasis rosea and eczema marginatum of the axilla and groin. Scholtz[3] reports three cases illustrating this point. At times a retiform expression is given to the picture by coalescence of the patches. The disease occasionally occurs in the negro.[4] Wile[5] reports pseudo-vesicles associated with other lesions in a case of pityriasis rosea.

There may be moderate itching, with nocturnal exacerbation, but the usual type of the disease is mild. The affection runs its course ordinarily in from four to eight weeks, but may last for several months, if new lesions continue to appear. Fox[6] believes that there is an acute (the common) and a chronic (the rare) type, and quotes a case of Hallopeau's which lasted for four years. Recurrences are rare, though we have noted several such instances. Towle[7] and others have reported recurrent cases.

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[1] Jour. Amer. Med. Assoc., August 17, 1912, lix, p. 493.

[2] Loc. cit.

[3] Lancet Clinic, 1912, cviii, No. 21; abstr. Jour. Cut. Dis., 1913, xxxi, p. 375.

[4] Howard Fox, Jour. Cut. Dis., 1908, xxvi, p. 67; Schamberg, ibid., 1909, xxvii, p. 267

[5] New York Med. Jour., November 13, 1909.

[6] Loc. cit.

[7] Jour. Cut. Dis., 1909, xxvii, p. 364.

Etiology.[1]—The causes of this disease are obscure. It is without question more common in the spring and in autumn than in the other seasons. Bazin believed it occurred chiefly in lymphatic and scrofulous patients. Most of the patients are young (fifteen to forty years of age); many are of the female sex, have light hair and delicate skins, and have been enfeebled by physical fatigue or overtaxation in school. Alderson[2] noted definite severe nervous stress from prolonged excessive mental work, worry or actual shock in ten out of eleven cases studied. Profuse perspiration has been assigned as a cause by Horand.

Though no true epidemics are reported and positive evidences of contagion are wanting, it occasionally happens that the disease is so unusually prevalent during a few weeks in a given locality as to suggest an epidemic. There are also instances in which two members of the same family were affected.[3] It is possible that the disorder is feebly infectious and allied to the exanthemata. Szóaboky,[4] in 50 per cent. of 119 cases of this disease, recognized that there was but slight febrile movement before the development of the eruption. Of the entire number of patients only one had a return of symptoms. After microscopic examination, he failed to recognize a parasitic etiology for the disease; but in 66 per cent. of cases discovered that there were functional troubles of different character connected with the nervous system (sweating, trembling, pallor and redness, headache, and exaggerated reflexes). A large number of observers believe the disorder to be parasitic, among the earliest of these being Vidal. Oppenheim[5] recognized double-contoured organisms in the lesions, suggesting an oïdium, which in one instance appeared to transmit the disease from cultures. Mewborn[6] found a fungus with septate mycelium, staining with a central granular portion and clear, unstained margins. Dubois[7] describes a cryptogamic parasite, represented by masses of round spores up to 5 microns in size, found within the follicular and glandular orifices, which he believed to be etiological in pityriasis rosea.

Pathology.—The histopathology of the disease has been studied by Darier, Unna, Hollmann,[8] and Sabouraud.[9] The changes begin apparently in the papillary body and the subpapillary layer of the cutis, and include a dilatation of the vessels, perivascular cell-infiltration, and edema. As the disorder progresses, these changes are more marked, especially in the perivascular cell-infiltrate. The rete shows decided intracellular edema and proliferation of the prickle-cells, especially in the interpapillary portions. As the disease approaches its acme, minute vesicles (not visible on macroscopic examination) form beneath the horny layer, which later is exfoliated. Sabouraud states that these vesicles are found in the outer layers of the epidermis, much as the "dry abscesses" described by Munro are formed in psoriasis. The absence of polymorphonuclears (phagocytes) in the vesicles leads him to believe that the disease is not parasitic, but a vesicular erythema of toxic origin.

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[1] Owens, Jour. Cut. Dis., 1914, xxxii, p. 347; Observations on Pityriasis Rosea.

[2] Jour. Cut. Dis., 1914, xxxii, p. 353; Pityriasis Rosea: Clinical Observations..

[3] Crocker; Zeisler, Jour. Cut. Dis., 1893, p. 494; Fordyce, ibid., p. 497; G. H. Fox, and others.

[4] Monatshefte, 1906, xlii, p. 495.

[5] Verhandlung. der 79 deutsch Naturforscher und Aertze, September, 1907.

[6] Jour. Cut. Dis., 1906, xxiv, p. 431.

[7] Annales, January, 1912, s. v, iii, p. 32; abstr. Jour. Cut. Dis., 1912, xxx, p. 382.

[8] Archiv, 1900, li, p. 229.

[9] Revue praktique des Maladies cutanées, syph. et ven., June, 1902; abstr., Jour. Cut. Dis., 1903, xxi, p. 55.

Diagnosis.—The diagnosis is simple, especially if a number of oval patches show the usual arrangement, with long axes in the lines of cleavage. When the lesions are numerous but less perfectly developed, and are of the smaller, maculo-papular and less inflammatory type, the disease may resemble a maculo-papular syphiloderm so closely as to deceive even the expert. In the absence of all other evidences of syphilis, the delay of a few days will usually permit the development of either the typical oval lesions of pityriasis rosea or of other signs of syphilis. In syphilis the elementary macules are uniformly smaller and much less disposed to scale. Ordinarily, the lesions of pityriasis rosea are less infiltrated, are of a brighter but paler tint, and are usually more rapid in evolution than those of syphilis. The congestion of the fauces in the former is of a bright-red color and diffuse, while that of syphilis is dull-red and circumscribed. Other diseases to be differentiated are the following.

Dermatitis Seborrhoica.—In this disease, the slow development of the lesions; their distribution over the scalp, sternum, and between the scapulæ, rather than on the trunk along the lines of cleavage; the coarser and more abundant scales; the fine papules on the one hand or large areas on the other; and the absence of the oval lesions of pityriasis rosea, will establish the diagnosis. There are cases in which the differential diagnosis is exceedingly difficult or almost impossible, and which suggest an intermediate stage between the two disorders.[1]

Psoriasis.—In psoriasis the patches are infiltrated, elevated, and more sharply defined. The abundant imbricated and silvery-white scales, the bleeding points beneath, and the distribution of the lesions, are points of value in the diagnosis.

Ringworm.—In ringworm of the glabrous skin the lesions are rarely so numerous or so symmetrically distributed. The areas are more definitely circular, more circumscribed, and often display minute vesicles at the periphery. The areas show clearing centers and are usually larger than those of pityriasis rosea. Finally, the trichophyton fungus can be demonstrated in the scales.

Treatment.—Pityriasis rosea, as a rule, is a self-limited disease, in which the duration and career vary greatly in different cases. Consequently, it is difficult to judge of the value of treatment in a given case. Systemic treatment should be varied to meet the indications in each instance. General symptoms, if present, should receive appropriate attention. In the major portion of cases no internal treatment is required. Crocker believed the course of the disease is shortened by giving salicin in 15 grain (1.) doses three times a day. Locally, mild sulphur or other antiseptic ointments appear to shorten the duration of the disease in many instances. A convenient and simple treatment, which we have employed with apparently good results in many cases, is as follows: The patient takes a bath at night before retiring, and after drying the skin applies to the areas a weak vinegar or dilute solution of acetic acid, and before this dries follows with a 10 to 15 per cent. solution of sodium hyposulphite. In a few moments, after the surface is dry, a simple dusting-powder may be applied. In the few instances in which itching or burning is annoying, the underclothing should be of silk or cotton, and the surface of the body should be kept constantly covered with some adherent powder, like zinc stearate. Occasionally, it is necessary to use soothing, mildly antipruritic lotions or ointments, such as are recommended for the early stages of eczema. In unusually extensive cases, in which itching is a pronounced feature, mild exposures to *x*-rays are followed by prompt cessation of subjective sensations and by more rapid involution of the lesions.

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[1] Besnier, Annales, 1889, s. ii, x, p. 108.

LICHEN RUBER.

Under the term *Lichen ruber*, Hebra[1] was the first to describe the disease which corresponds closely to the disorder described in these pages as pityriasis rubra pilaris. Most of Hebra's cases, however, were associated with grave systemic conditions, and twelve out of fourteen terminated fatally. Kaposi later described a lichen ruber acuminatus, which he stated is identical with the lichen ruber of Hebra, though in his cases the general health of the patient was not seriously affected. He divided these cases into two groups, the acuminate and plane. Confused with these cases were those described by Wilson as lichen planus.

The relationship of the various diseases under consideration has been the subject of much discussion. At a dermatological congress held in Vienna, in 1892, a patient was exhibited who, Kaposi stated, was suffering with lichen ruber acuminatus, while Vidal, Hallopeau and other Frenchmen declared it to be a case of pityriasis rubra pilaris. Critical comparison of the literature and illustrations of the subject remove all doubt that pityriasis rubra pilaris (Devergie) and lichen ruber acuminatus (Kaposi) are one and the same disease. Hebra's lichen ruber, judging from Kaposi's statements and from two plates published by Hebra (to which Crocker calls attention), was probably a severe form of the disease. A few German authorities still teach that pityriasis rubra pilaris is wholly distinct from lichen ruber, which they subdivide into lichen ruber acuminatus and lichen ruber planus. Instances are cited by Kaposi, Neumann, and others in which the acuminate and the plane papules coexisted in the same individual. These few cases are probably coincidences or modifications of usual types, and lichen planus is generally held to be an entirely independent disease.

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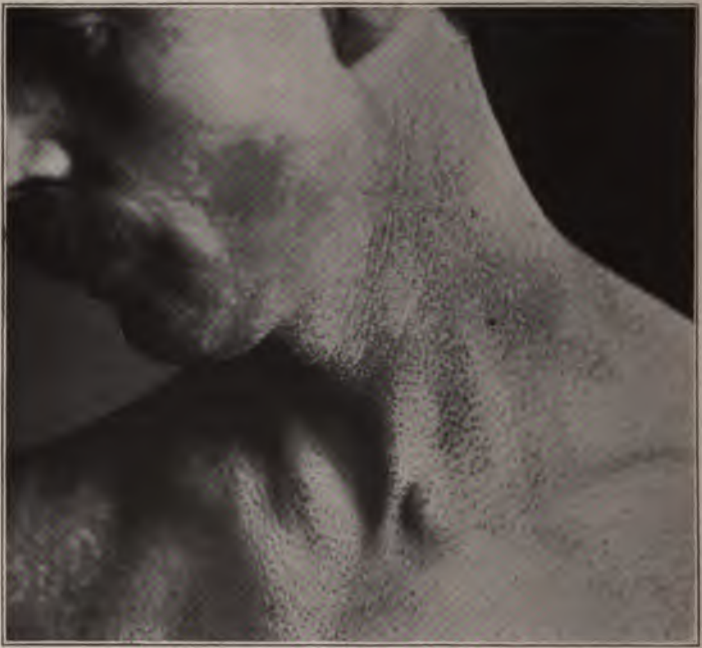
[1] Diseases of the Skin, New Sydenham Society translation, 1868, p. 57.

PITYRIASIS RUBRA PILARIS.

Synonyms.—Lichen ruber (Hebra); Lichen ruber acuminatus (Kaposi); Lichen-Psoriasis (Hutchinson); Pityriasis pilaris (Devergie). Fr., *Pityriasis rubra pilaire.*

Definition.—Pityriasis rubra pilaris is a chronic, mildly inflammatory, exfoliating disease of the skin, in which the characteristic lesions are fine, acuminate, firm papules situated at the mouths of hair-follicles, and displaying at the apex a horny plug or scale, which dips into the follicle. By coalescence the papules form reddened, scaling areas, which may spread and cover the entire surface of the body. This affection has been described chiefly in France by Devergie,[1] Besnier,[2] Richaud,[3] Brocq, and others. The Museum of the St. Louis Hospital, Paris, is provided with illustrations in wax of every phase of the malady. Numerous examples of the disease have come under the observation of experts in America. The malady is undoubtedly identical with lichen ruber acuminatus of Kaposi.

FIG. 98



Pityriasis rubra pilaris

Symptoms.—The disease usually begins insidiously, but may appear more or less suddenly, with or without mild systemic disturbance.

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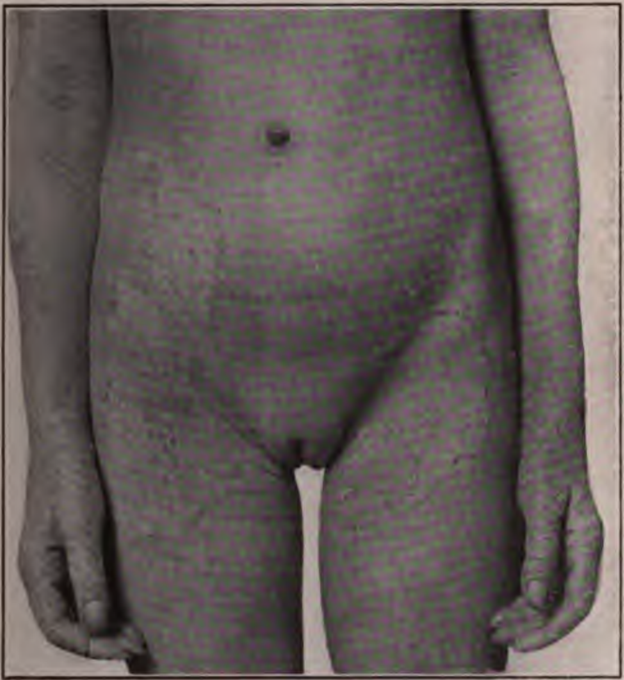
[1] Traité pratique des Maladies de la Peau, 1857, 2d Ed., p. 454.
[2] Annales, 1889, s. ii, x, pp. 253, 398, 485.
[3] Thèse de Paris, 1877.

a rule, the characteristic papules ("projecting cones") are not seen til after a period in which the disease appears as a seborrhea sicca the scalp, with or without palmar and plantar scaling patches. The order may appear first on the face (nose, brow, lips, chin), as a e pityriasis, or as a condition simulating seborrhea sicca. A similar e desquamation may be present on the ears, neck, and other parts the body before the appearance of papules, but as a rule the latter pear on one or more regions soon after the first evidence of the disler and gradually extend to other portions of the body. The scales seated at the follicular orifices; are thin, whitish, grayish, or heaped

FIG. 99

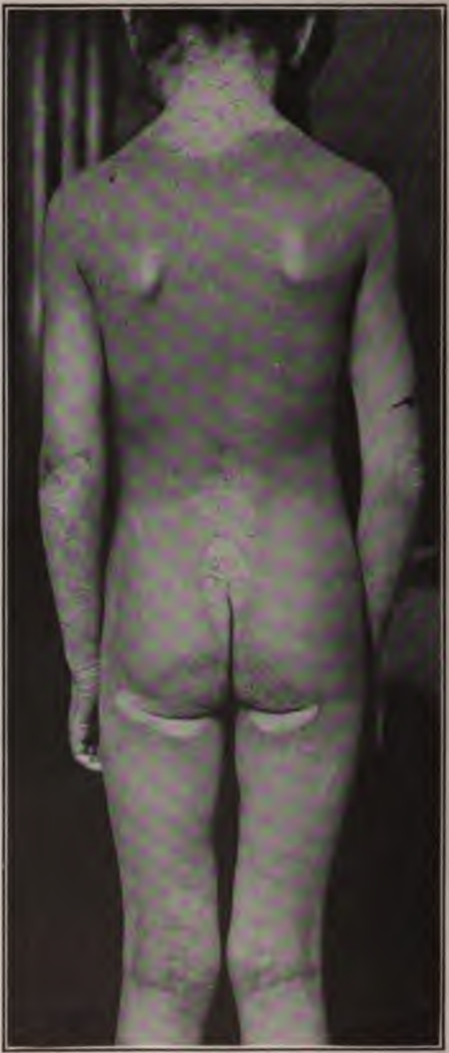


ityriasis rubra pilaris. Duration seven months. Russian child aged seven years.

in large discoid masses; are dry, firmly attached, friable, and in es suggest the "crackle-ware" of the potteries. The disease is ually well marked over the extremities and on the back of the neck, t may involve any or all portions of the body. Occasionally, in acute type of the disorder, a large number of isolated papules pear somewhat suddenly over several regions, producing a condition ulating goose-flesh.

The characteristic papules are minute, acuminate, hard, dry, and of color varying from that of the normal skin to the different shades pink, rosy-yellow, or duller hues. They are situated at the hair-follicles and each is pierced by a hair. At the apex of the papule, a surrounding the hair, is a horny sheath, which penetrates the ha follicle for a short distance. Fine lanugo-hairs which pierce the papu

FIG. 100

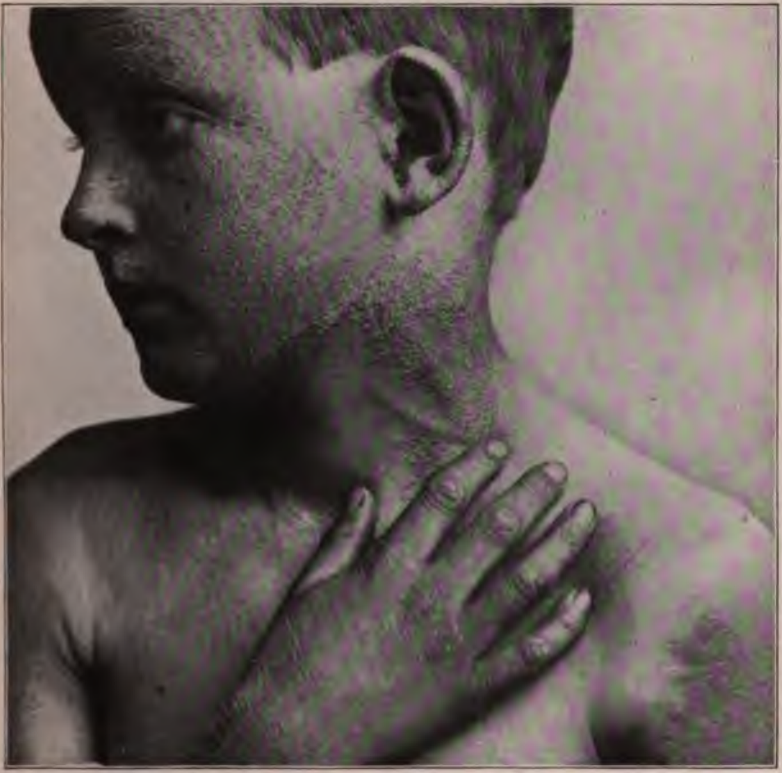


Pityriasis rubra pilaris.

may be recognized on close inspection; the whitish, horny plugs t giving the lesions a scale-capped appearance.

The papules become more and more numerous, and appear at ti to coalesce, but may form patches, at times symmetrically dispos covered with fine elevations, conical and discrete; or they may bec round, flatter, and coalesce so completely as to be lost in the general scaling, exfoliating, erythematous, and lucent area. The yellowish-red or deep-reddish patches may be the seat of pityriasic scaling, or may exhibit separation of the epidermis in large, adherent flakes, which, especially over the elbows and the knees, present the appearance of psoriasis. When the infiltration is moderate, the intensifying of the natural lines of the skin is a conspicuous feature. The areas are irregular in size and shape, but frequently have an angular or oblong outline. Commonly at the borders of these patches are found the initial papules of the affection, still isolated and surrounding characteristic stumps, filaments, or black points of hairs, enabling one thus to make the diagnosis with ease.

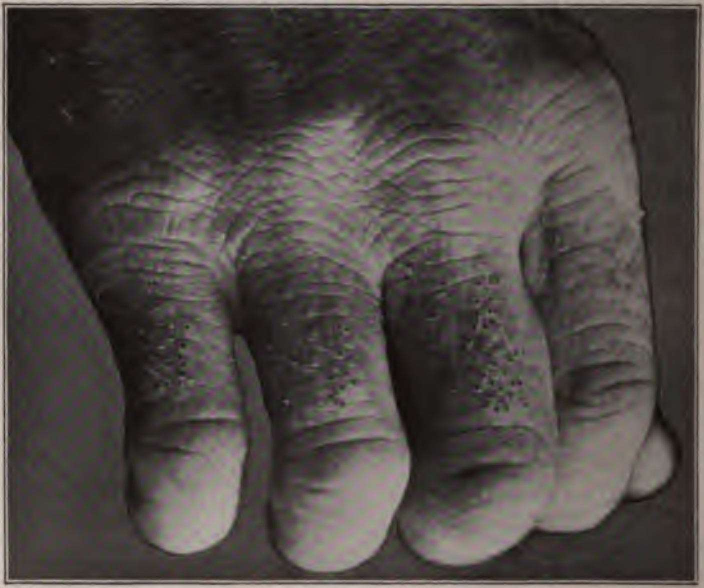
Fig. 101



Pityriasis rubra pilaris.

When discrete papules are grouped closely, and in areas formed by aggregation rather than by complete coalescence of the papules, a "nutmeg-grater" effect is produced when the finger is passed over them. At times the eruption is generalized; when the face chiefly is involved, the slight crusts formed are decidedly of the type of those described under dermatitis seborrhoica. In many cases the tension of the dry, infiltrated skin produces ectropion of the lower eyelid. Occurring over the hairy scalp, the accumulated scales and crusts may form a dense and resisting cap, which is difficult to remove. The nails are usually grayish, yellowish, transversely striated, and roughened. There may also be a coincident polytrichia. Important for purposes of diagnosis are the little horny, blackish, conical papillæ occupying the site of the hair-follicles on the dorsal surfaces of the first and second phalanges of the fingers. These usually remain distinct even when, on all other parts of the body, their identity has been lost in the general exfoliative process. Sometimes an exceedingly characteristic feature of the disease is displayed in the face, which on inspection seems to be covered with a more or less firmly attached, irregularly creased, mortar-like plaster, the "cast" being conspicuously evident on the tip and root of the nose, the lower brow, the lips, and the chin. When the palms and soles are involved, they become the seat of a firm, thick, lamellar hyperkeratosis, reddish-yellow in hue, furnishing a "keratodermic sandal" (Besnier) for the sole.

Fig. 102



Pityriasis rubra pilaris.

The course of the disease is usually chronic, irregular, and subject to relapses and to unexpected exacerbations. The disease has a tendency to become generalized, and even universal, and to persist indefinitely. Periods of remission or of complete clearing of the skin are noted in a few instances, but the disorder usually returns. In the group of cases under the observation of the author improvement has occurred temporarily but relapses occurred. In two the disease was acute at its onset, becoming almost universal within ten weeks from its appearance. In one of these patients, who acquired syphilis soon after the appearance of pityriasis rubra pilaris, the latter disappeared entirely in five months from its onset and had not recurred at the end of nine years, when the patient died as the result of an accident. The other was entirely relieved at the end of nine months, but his subsequent history is unknown.

Subjective sensations may be entirely absent, though there is usually a sense of dryness and of contraction of the skin. There may be more or less itching, though, as a rule, this is not marked. In the earlier stages, at least, the general health appears to be unimpaired, even when the disorder is generalized. Eventually, however, in some cases there is more or less failure of general nutrition, leading in rare instances to a fatal result.

Etiology.—The cause of the disease is unknown. It commonly begins in the second decade of life, but has been observed in all ages, somewhat more often in men than in women. Cases are reported at the age of one and one-half years,[1] two and one-half years,[2] and at three years.[3]

Milian[4] regards tuberculosis as the etiological factor. De Beurman, Bith, and Heuyer[5] report four cases in one family, two males and two females, the ages ranging from twelve to twenty-eight years, three of them having apical phthisis.

Pathology.—The definite pathogenesis of the disorder is unknown. The theory of its being a toxic process due to the bacillus of tuberculosis or other such agent has been advanced. The histopathology, as given by Jacquet in Besnier's cases,[6] Taylor,[7] Heidingsfeld,[8] Hartzell,[9] Heller,[10] and others, shows that the papule which is the essential lesion of the disease is formed by a hyperkeratosis of the superior portion of the hair-follicle, and that there is a hyperkeratosis of the entire epidermis in addition, and a mild inflammatory process in the corium, probably secondary to the epithelial changes. German cases, under the name of *lichen ruber acuminatus*, have been studied by Hebra, Kaposi, Neumann, Biesiadecki, Joseph, and others. Different reports vary considerably, depending, probably, upon the age of the lesions examined. The pathological processes correspond closely to that described above, except for a more pronounced inflammation in the corium, as the result of which Kaposi and others believe the epithelial changes to be secondary to an inflammation of the corium.

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[1] Whitehouse, J., Jour. Cut. Dis., 1912, xxx, p. 482.

[2] Rasch, Centralb., 1899, i, p. 199.

[3] Heller, Zeitschrift, 1903, x, p. 153 (with histological study).

[4] Annales, 1906, s. iv, vii, pp. 1067–1075.

[5] Ibid., December, 1910, p. 609; abstr. Brit. Jour. Derm., 1911, xxiii, p. 165.

[6] Loc. cit.

[7] New York Med. Jour., January 5, 1889, p. 1.

[8] Jour. Cut. Dis., 1906, xxiv, p. 371

[9] Stelwagon's Diseases of the Skin, 7th Ed., p. 234.

[10] Loc. cit.

Diagnosis.—The disease is to be differentiated from all others by the characteristic papule pierced by the shaft, or segment of shaft, of a hair. In extensive cases of long standing the identity of the papules may be lost in the general desquamation over most of the body; but in nearly all cases lesions can be recognized on the backs of the fingers, as described above.

From lichen planus the diagnosis is not difficult in the early stages, or when individual papules are found bordering the larger areas. The dull-crimson or violaceous hue of patches of lichen planus is characteristic. Moreover, the disease is rarely so generalized as pityriasis rubra pilaris. Keratosis pilaris is limited, as a rule, to the regions which it chiefly affects, the extensor faces of the limbs. Ichthyosis is commonly congenital, the first lesions developing soon after birth. In psoriasis the characteristic silvery-white, imbricated scales, the bleeding points beneath, and the larger size of the primary lesions will usually establish the diagnosis. In pityriasis rubra (of Hebra) the history of the disease, the absence of distinct papules and of infiltration, and the appearance later of atrophy of the skin are distinctive features. It must be remembered that rarely pityriasis rubra pilaris may terminate in a generalized exfoliative dermatitis, which cannot be distinguished from the same process arising in psoriasis, eczema, or other scaling affections (see Dermatitis Exfoliativa).

Treatment.—Systemic treatment should be varied to meet the indications in each individual. In many cases tonics, cod-liver oil, and an especially nutritious diet are indicated. Crocker praises thyroid extract, beginning with 5 grains (0.33) and gradually and continuously increasing. Arsenic has given excellent results in some cases, but in a large number has failed; and apparently in a few instances has aggravated the disorder. We have had marked amelioration of the symptoms following the combined use of arsenous trioxid, grain $\frac{1}{20}$ (0.0033), and protiodid of mercury, grain $\frac{1}{6}$ (0.01), three times a day, together with external applications.

The local treatment corresponds closely to that of psoriasis, squamous eczema, and other exfoliative conditions. The daily use of an ointment containing from 5 to 20 grains (0.33–1.3) of salicylic acid to the ounce (30.) of vaseline, or of equal parts of vaseline, lanolin, and olive oil, is often of value in keeping the skin soft and relieving the itching when present. For markedly thickened areas, ointments containing salicylic acid in strength of 20 to 60 grains (1.33–4.) or more to the ounce (30.) may be used; or some of the preparations of chrysarobin, resorcin, oil of cade, or ichthyol recommended for the treatment of psoriasis. Fatty crusts, when these are abundant, are to be removed by shampooings, as in seborrheal affections of the scalp.

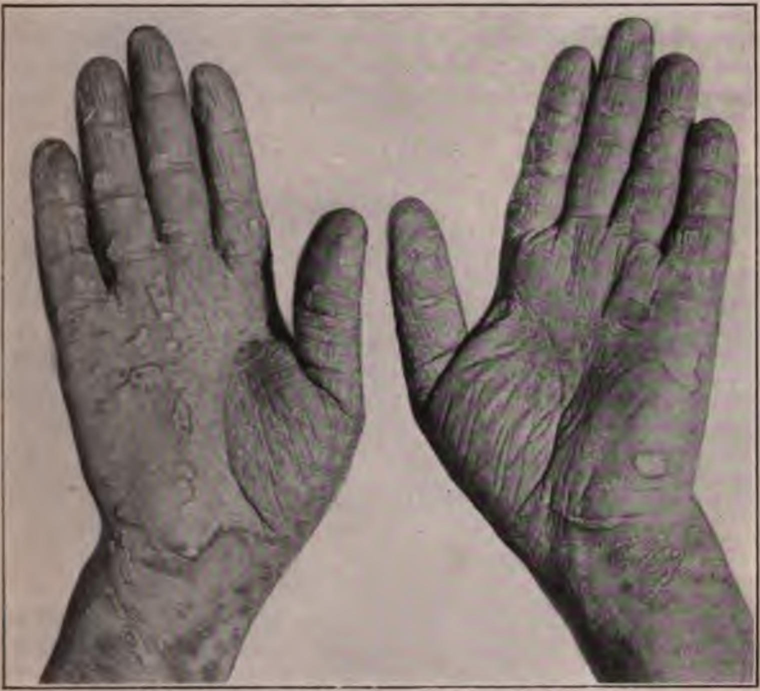
Prognosis.—The prognosis is unfavorable with respect to the cutaneous manifestations, as in those cases in which the disorder disappears temporarily it almost invariably recurs. The tendency of the disease is to persist indefinitely. The general health may be unimpaired, but is affected sooner or later in many instances. The issue in exceptional cases may be fatal.

LICHEN PLANUS.[1]

Synonyms.—Lichen ruber planus, Lichen psoriasis.

Definition.—Lichen planus is an inflammatory dermatosis, in which are displayed multiple, small, flat-topped, angular or polygonal papules, often exhibiting a color containing various shades of crimson or purple, the plane apex of each being usually flat or depressed and covered with a horny film. This disease was first described by Erasmus Wilson, in 1869. The disorder is of frequent occurrence, though it is not one of the common diseases of the skin. It is usually chronic, but may be acute, and, although in most instances limited in distribution, it may be extensive and even generalized.

Fig. 103



Lichen planus of the palms. (Fordyce and MacKee.)

Symptoms.—In a typical case of lichen planus, the primary lesions are pinpoint- to pinhead-sized, angular or polygonal, flat papules.

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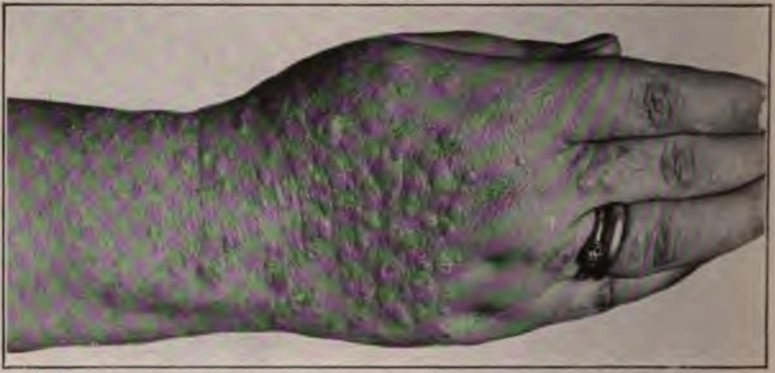
[1] Little, Jour. Cut. Dis., 1919, xxxvii, p. 639; Lichan Planus. A complete description of the disease based on the study of 270 personal cases. Opening address on a discussion of the disease before the American Dermatological Association at its forty-second annual meeting. See also White, ibid., p. 671. Lichen Planus. A critical analysis of 64 cases. Paper read in continuing the discussion above mentioned. Fordyce and MacKee, ibid., p. 320. Clinical types of Licher Planus, well illustrated. Culver, Archiv. of Derm. and Syph., 1920, n.s. vol. i, p. 43. A clinical study of Lichen Planus. Based on a review of 148 personal cases.

These are sharply defined and covered, not with a scale, but with a thin, transparent, horny film, which gives to the lesions a waxy or varnished appearance. As the papules increase in size, they retain their angular or polygonal outline and remain flat, or may become slightly umbilicated. The bases are angular or rounded and the sides precipitous. The greatest diameter attained by any individual papule is about one-half that of a small split-pea, but by coalescence the original lesions may form larger areas, which are also angular, linear, or polygonal in outline, and are sharply defined from the surrounding skin. On the patches the thin, horny covering may be partially broken up into fine, closely adherent scales. The color of recent lesions is a bright crimson, that of the older a purplish or reddish purple. On the surface of the papules may be seen on close inspection minute whitish points and lines, to which Wickham has called attention ("Wickham's striæ"). When the eruption is plentiful, the violaceous color is characteristic, and the peculiar shining or glistening top observed when viewing the lesion in a position where the light falls aslant upon the surface is also characteristic. As the lesions grow older, the shade deepens to a dull purplish or darker color. Involution of the papules often leaves a pigmentation of a smoky, sepia, or even blackish, hue, which is naturally most conspicuous and most persistent on the lower extremities. Occasionally, white, atrophic-looking spots are left, which ultimately disappear.

FIG. 104

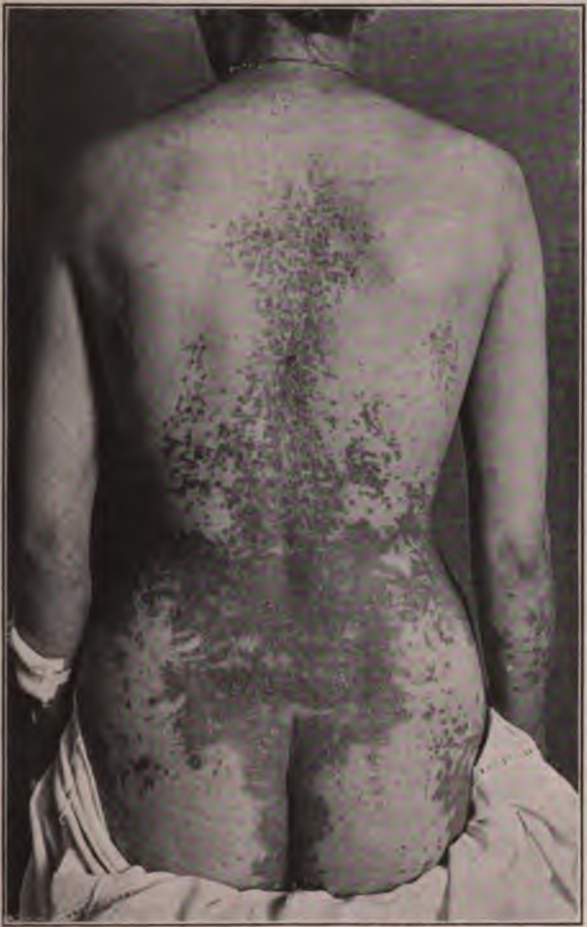


Lichen planus showing umbilication. (Fordyce and MacKee.)

The lesions may be discrete and isolated, or irregularly grouped, but when numerous they tend to multiplication and aggregation and form irregular, linear, angular, or polygonal patches, with sharp outlines. Annular or circinate patches may occur. Rarely combinations of lines and circinate groups form exceedingly odd-looking figures—parallel lines, cockades, scaling crests, rings, or rosettes. The shape of the patch may be determined by an external irritation, such as a scratch-mark.

When the papules coalesce and lose their identity, a crimson-hued eet or mask of the skin is seen, generally characterized not merely by e color of the lichen planus papules, but also by a silvery sheen, due thin, shining scales, which do not completely cover, but which sup- ement, as it were, the empurpled patches beside and over which they rm. The scales are not freely shed from the surface, but are attached

Fig. 105



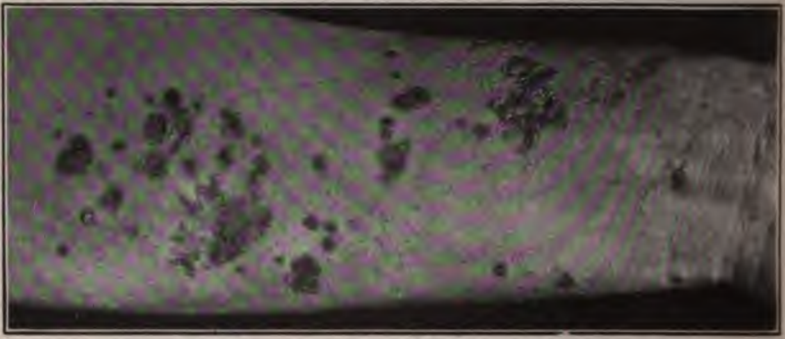
Generalized lichen planus. (Fordyce and MacKee.)

rmly. When there are decided sheets of infiltration, they are most nspicuous over the flanks and anterior part of the trunk; but they ay also be seen elsewhere, as, for example, over the extremities. When e patch is undergoing involution, the scaling ceases, the infiltration bsides, and a pigmentation somewhat similar to that described in nnection with the papules follows.

The disease, though usually limited to a few regions, particularly the flexor surfaces of the wrists and forearms, and the legs immediately above the ankles, is symmetrical as a rule, but may appear on one side only of the body and may cover large areas, and, in rare instances, the entire surface. The disease is seldom seen on the face or scalp, and is unusual on the palms or soles.[1] The papules on the palms and soles, according to Fordyce and MacKee (loc. cit.), are usually rather large, deep seated, perhaps semi-translucent, likely to be globular and suggest deep seated vesicles. After some time exfoliation occurs. The nails may be involved and present lesions similar to those seen in psoriasis and eczema.

The greatest variation is experienced in the way of subjective sensations. Itching may be moderate or severe. In acute generalized cases the suffering of the patient is extreme. The eruption of lichen planus, however, is scratched much less often than that of other cutaneous diseases characterized by itching.

Fig. 106



Lichen planus annularis. (Fordyce and MacKee.)

The course of the disease is chronic, and when untreated it may last for months or years, either through persistence of the original papules and areas, or, what is more frequent, by the successive appearance of new lesions. White[2] reported one case of sixteen years' duration. Occasionally, the disease disappears spontaneously, but its tendency is to persist. The disorder may recur, but recurrence is an exception to the rule.

Occasionally, lichen planus may begin as an acute exanthem and become generalized in a few days, or even within twenty-four hours. In such cases the lesions are usually minute, of bright color, and exhibit no tendency to definite grouping. There may be coincident febrile symptoms and mild systemic disturbance, or severe concomitant disorders, such as pemphigus, diabetes, syphilis, and grave ulceration.[3]

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[1] Dubreuilh and LeStrat, Annales, 1902, s. iii, iii, p. 209.

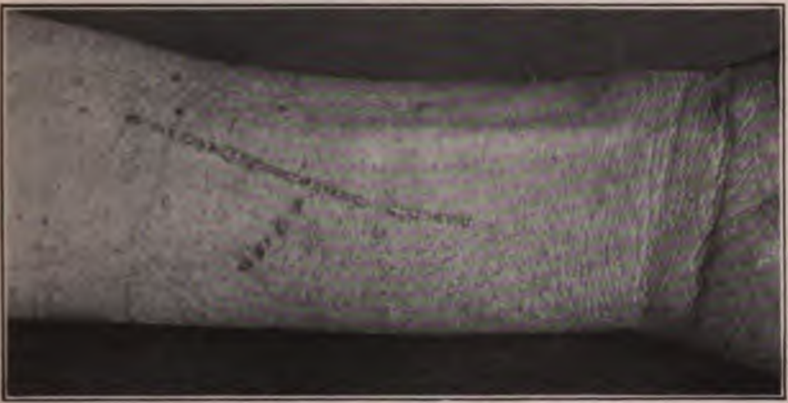
[2] Loc. cit.

[3] Johnston, Jour. Cut. Dis., 1907, xxv, p. 86; Galloway, Brit. Jour. Derm., 1906, xviii, p. 66.

These acute symptoms may develop in an individual previously free from all evidences of lichen planus, but more commonly in those who have exhibited for months or years one or more areas of the disease, which then may run an acute course of a few weeks, yielding readily to treatment, or may persist as a generalized or localized chronic form. A number of variations from the usual clinical types occur.[1]

Vesicles at the summit of some of the papules and bullæ occur in a number of cases of lichen planus, most frequently in patients who have been taking arsenic, but also in others who have taken no arsenic prior to the appearance of the lesions. Trautmann[2] has described a case in which pemphigus appeared to follow an attack of lichen planus. Whitfield,[3] in presenting a patient, analyzed 17 previously reported cases, in 9 of which the patient had taken no arsenic prior to the appearance of the bullæ. He states that the presence of bullæ apparently has no bearing on the severity or prognosis of the disease. Hartzell,[4] Allen,[5] and others report similar cases.

Fig. 107



Lichen planus papules in scratch marks. (Fordyce and MacKee.)

Lichen planus is of rare occurrence in children. Crocker, Liveing, and Colcott Fox[6] all report a spurious form, which the author first named believes to be a subsiding stage of papular or vesicular miliaria rubra. Adamson[7] recently recorded three cases of typical lichen planus in children. The lesions in children differ in no essential from those found in adults.

Occasionally, the lesions of *lichen pilaris seu spinulosus* (Crocker) are found in association with those of lichen planus in certain cases.

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[1] Crocker, Brit. Jour. Derm., 1900, xii, p. 421 (with discussion before the London Dermatological Society).

[2] Zeitschrift., 1906, p. 317.

[3] Brit. Jour. Derm., 1902, xiv, p. 161.

[4] Jour. Amer. Med. Assoc., July 20, 1907, p. 225.

[5] Jour. Cut. Dis., 1902, xx, p. 260.

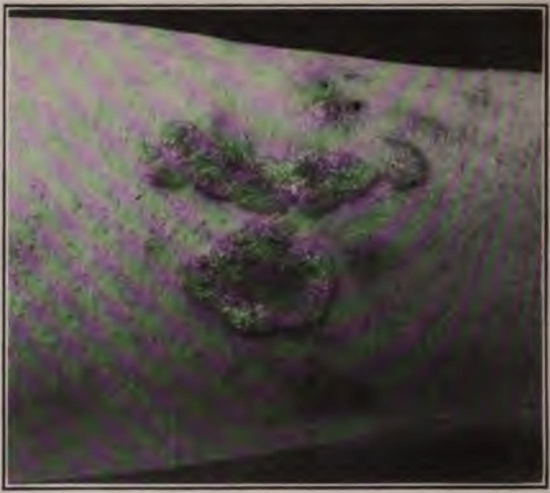
[6] Brit. Jour. Derm., 1891, iii, p. 201.

[7] Brit. Jour. Derm. and Syph., 1920, xxxii, p. 1.

As a rule, the general health is not involved, save when the itching is so severe as to interfere with the patient's sleep or rest. Crocker refers to generalized cases in which the health was affected profoundly, a few of which terminated fatally. In this country one such case has been reported by Fordyce.[1] It may be that the severe systemic disorders present in some of these cases were independent of the lichen planus.

Lesions of a different type from those depicted above, occurring with the ordinary types as well as independently, and which are now admitted to be forms of lichen planus, have been described under various titles.

Fig. 108



Lichen planus hypertrophicus. (Fordyce and MacKee.)

Lichen Planus Hypertrophicus[2] is a special form occurring usually on the lower extremities, occasionally on the upper, in which the lesions, after long persistence, have lost their ordinary characteristics and formed thickened, elevated patches. The patches, which are of varying size, may be rounded, elongated (band-like), or irregular, and present a reddish-brown or purplish color. They are covered with fine, adherent scales and horny projections, giving the lesions a warty appearance (*lichen planus verrucosus*). At times acuminate or conical horny papules occur, which by coalescence produce large patches. Commingled with these are sepia-brown pigmented spots, and at times atrophic areas. Itching is severe and persistent, and the lesions are aggravated by the trauma inflicted by scratching.

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[1] Jour. Cut. Dis., 1899, xvii, p. 56.

[2] Fordyce, Jour. Cut. Dis., 1897, xv, p. 49; Corlett (quoted by Fordyce, loc. cit.); Lieberthal, Jour. Amer. Med. Assoc., January 11, 1902, p. 93 (histology); Ravogli, Jour. Cut. Dis., 1904, xxii, p. 573 (histology). Sutton, Jour. Amer. Med. Assoc., 1915, lxix, p. 1063. Two unusual cases of this type.

Lichen Planus Obtusus is a term applied to a form in which rounded or oval, flat or slightly convex papules of large size (pea to bean or larger) occur, situated chiefly on the arm or forearm. These may occur independently or in association with the ordinary types.

Lichen Planus Linearis.[1]—The tendency of lichen planus papules to form linear groups or bands may be exaggerated to produce this type. In such cases a narrow fillet of typical lesions may extend from the heel to the trunk along the line of the sciatic or other nerve, or, more frequently, from the buttock to a few inches below the knee. Such a case has been under our observation. A similar arrangement of the lesions may occur along the course of the nerves of the upper extremity or on the trunk. Again, the bands may be absolutely straight and apparently independent of the course of any nerve. Galloway[2] has reported a striking example of this type, and we have had a similar case, but less extensive, on the outer surface of the thigh and leg.

Lichen Ruber Moniliformis is a title given a rare form of the disease, described in 1886 by Kaposi.[3] Rowe, Dubreuilh, Gunsett,[4] and others have reported similar cases, in which numerous node-like masses are arranged in lines and bands resembling a necklace of beads, with flattish, punctiform papules, and macules of a sepia-brown hue between the nodes. (A case of lichen ruber moniliformis was shown by Dr. Hyde before the Chicago Dermatological Society in the year 1903.)

Lichen Planus Annularis is that form of the disease in which the papules, while extending peripherally, leave a clear or clearing center, and form thus circular patches in thin rings or bands, at times coalescing in polycyclic outlines. The patches may be few or numerous; the rings faintly or very distinctly outlined; the component parts of the ring, the characteristic papules of lichen planus, either readily distinguishable or so fused as to render their identification difficult. Most English dermatologists[5] believe the annular lesion is formed by the fusing of individual papules rising in a ringed arrangement. Cavafy[6] asserted that in addition to this form of development certain annular lesions were formed by the peripheral extension, with central involution, of a single papule. Engman[7] confirmed Cavafy's view by proving, both clinically and histologically, that annular lesions are developed by both of the methods above outlined.[8]

Lichen Planus Erythematosus.—Under this title Crocker describes two cases in which the papules were of a deep crimson tint, soft to the touch, and obliterated temporarily by pressure. There was in both a marked telangiectasis of the face. Crocker mentions a similar case reported by Stirling.

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[1] *Cf.* Heller, loc. cit., with reference to previously published cases of this type; and Whitfield, Brit. Jour. Derm., 1906, xviii, p. 221; Little, Brit. Jour. Derm., 1917, xxix, p. 220, case report. Auner, Jour. Cut. Dis., 1917, xxxv, p. 166, case report with histology. Ketrou, Archiv. of Derm. and Syph., 1920, ii, 358. A case of lichen planus linearis, its relation to a cutaneous nerve experimentally shown.

[2] Brit. Jour. Derm., 1900, xii, p. 206.

[3] Vierteljahr, 1886, p. 571.

[4] Archiv, 1902, lx, p. 179 (with histological report and bibliography).

[5] Brit. Jour. Derm., 1900, xii, p. 421.

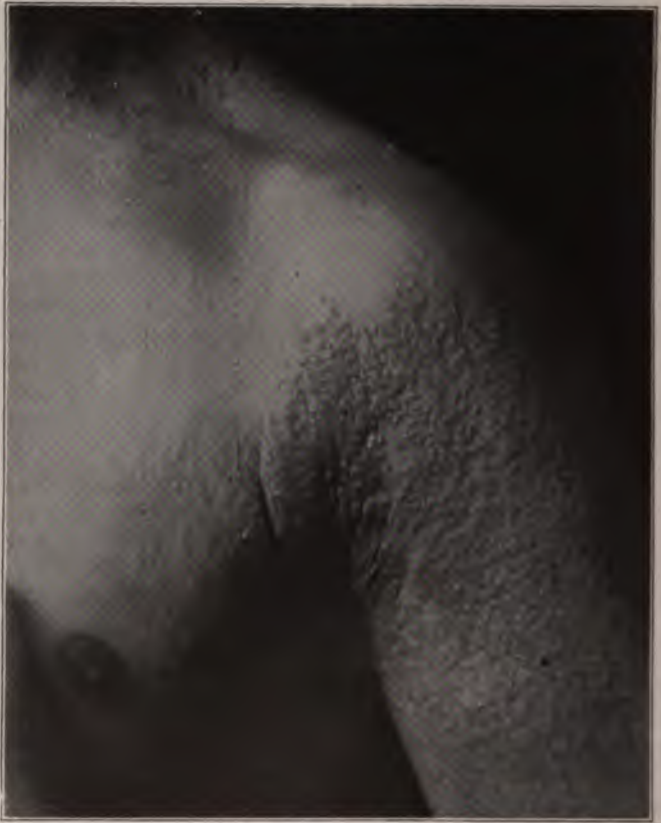
[6] Loc. cit.

[7] Jour. Cut. Dis., 1901, xix, pp. 209–222.

[8] *Cf.* Sutton, Jour. Amer. Med. Assoc., 1914, lxii, p. 175.

Lichen Planopilaris (Pringle) indicates the presence of spiny follicular papules with the ordinary plane papules of the disease. They are unaccompanied by itching and inflammatory phenomena and are not grouped. Little reported six cases of this type all occurring in women (loc. cit. references of other recorded cases).

Fig. 109



Lichen ruber moniliformis.

Lichen Planus of the Mucous Surfaces[1] (tongue, inner surfaces of the cheeks, lips, epiglottis, glans penis, progenital region of both sexes, anus, and perianal region) may occur with or without cutaneous symptoms. In some cases of well-marked cutaneous disease the mucous membranes are so slightly affected and attract so little attention that they are overlooked. Dubreuilh[2] believes that more cases of involve-ent of mucous membranes occur without cutaneous lesions than of he last-named without mucous symptoms. Confusion has been bred

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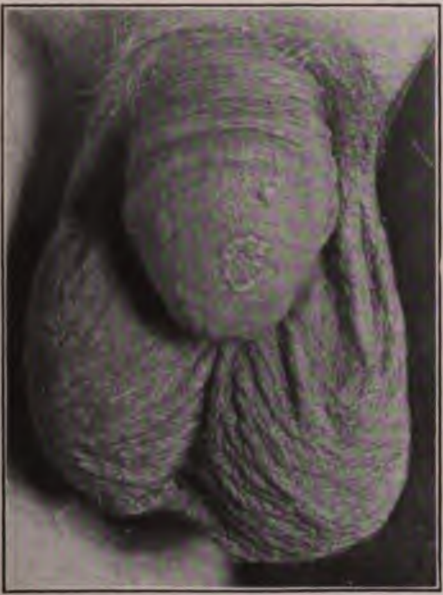
[1] Lieberthal, Jour. Amer. Med. Assoc., 1907, xlviii, p. 559; Lichen Planus of the Mouth.

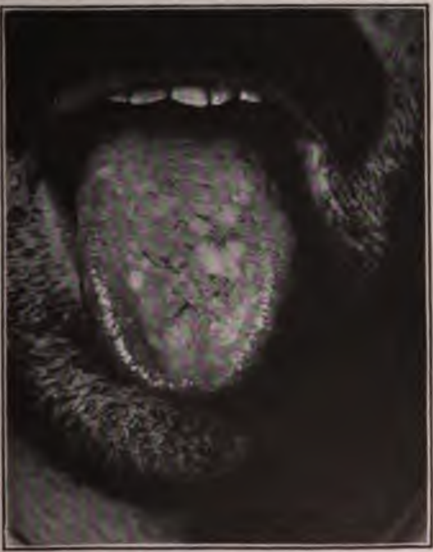
[2] Histologie, Lichen plan des Muqueuses, Annales, 1906, s. iv, vii, pp. 123–129.

FIG. 110



Annular lesions of lichen planus on the penis. (Fordyce and MacKee.)

FIG. 111



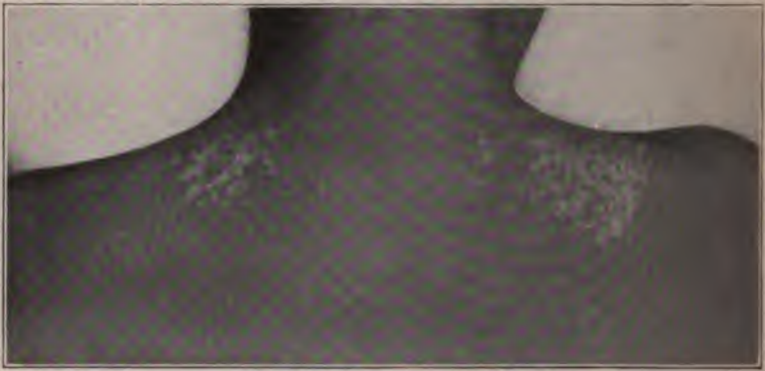
Lichen planus papules on the tongue.

in these cases by the hastily formed conclusion that the lesions here discussed are mucous patches or symptoms of leukokeratosis buccalis.

Pinhead- to hemp-seed-sized, grouped or isolated, slightly projecting, velvety, smooth, grayish, whitish, rounded lesions may be recognized as lesions of lichen planus of the mucous surfaces, the color and size varying somewhat with the individual, the age of the disorder, and the locality involved. Sometimes a slight halo surrounds the base of each; at times they are firm, at others soft to the touch. Again, they may send short ramifying striæ to the neighboring mucous surface. Vörner,[1] and others describe umbilication of the lesions. As distinguished from purely cutaneous lesions, they may be smeared with a whitish mucus.

Lichen Planus Sclerosus et Atrophicus (**Hallopeau**) and **Lichen Planus Morphœicus** (**Crocker**), a rare form of the disease, has been described in addition by Morrant Baker and Stowers in England, and by the author,[2] Schamberg and Hirschler,[3] and Sutton in this country. The characteristic lesion found in this variety is an irregular, often polygonal, flat-topped, white papule. The white color of the lesions is striking, and has been compared with that of ivory and mother-of-pearl. The papules, as a rule, are firm to the touch, neither elevated nor depressed, but slight elevation may be present. Generally, no areola is present, but at times a rosy or moderately pigmented zone may surround the papules. These may be discrete or grouped, and in most cases present both types. When grouped to form plaques, the outline of the individual papules forming the plaques can be determined. Each papule has on its shining, smooth surface from one to several black or dark, horny, comedo-like plugs, or minute bead-like depressions, which show the former sites of horny plugs. These ele-

Fig. 112



Lichen planus atrophicus.

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[1] Zeitschrift, 1906, xiii, p. 107; abst. Annales, 1907, s. iv, viii, p. 145.

[2] Jour. Amer. Med. Assoc., September 10, 1910, p. 901 (report of 6 cases, with a review of the literature).

[3] Ibid., 1909, p. 369.

ments are situated at the pilosebaceous or sweat-pore orifices, and are important from the view-point of diagnosis.

Etiology.—The causes of lichen planus are obscure. It is often difficult to recognize the sources of the disease, but in many cases a history of nervous exhaustion can be obtained. Grief, long-continued anxiety, and overwork, especially when accompanied by great mental strain, frequently precede this disorder. Acute and aggravated cases have presented themselves before us in several instances following a great shock. On the contrary, many patients are well-nourished and not lacking in flesh. In fact, the combination of a fair degree of nutrition of the body with nervous exhaustion is to be frequently recognized in patients affected with lichen planus. Little's statistics show a decline in the incidence of lichen planus during the world war and suggests that this fact is unfavorable to the nervous hypothesis as nervous strain was then at a maximum.

D. W. Montgomery and Alderson[1] suggest that the disorder is due to a toxemia. Fordyce[2] comes to the same conclusion in regard to his fatal case. Engman and Mook[3] suggest that lichen planus may be due to some constitutional disturbances caused by an infectious microbic agent. Chipman[4] suggests that the disease may result from systemic toxemia or bacteremia the origin of which is often in the teeth. Numerous observers have reported the occurrence of two or more members of a family affected with the disease, several examples having come under our own observation.[5]

Other causes cited are: traumatism (dog bite, Walters), digestive disturbances, malaria, malnutrition, and diseases of the generative organs. Within a year we have seen a patient whose attack of lichen planus followed the bite of a black ant. The lesions began shortly after the bite, which occurred upon the thigh, and spread in a band-like arrangement for some weeks, after which they developed rather generally over the body.

Lichen planus is more common after the second decade of life, and is rare in children.[6] Different opinions are entertained respecting the frequency with which men and women are attacked. General experience points to the conclusions formulated by Crocker, who reports more cases among women (English) than among men, while the statistics of the Vienna school reverse the figures. In the statistical studies of Little (loc. cit.) and White (loc. cit.) women were more frequently attacked than men. On the other hand Culver (loc. cit.) found the disease slightly more frequently in men. The disease is encountered more frequently in private practice, among the nervously taxed of the well-to-do classes, than among out-patients of public charities, who suffer to a greater extent than others from cachexia and malnutrition. Hoffmann[1] reports the coexistence of lichen planus with diabetes. A similar observation has been made by the author.

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[1] Jour. Amer. Med. Assoc., October 30, 1909, p. 1457.

[2] Loc. cit.

[3] Interstate Med. Jour., June, 1909.

[4] Trans. Sec. on Derm., Amer. Med. Assoc., 1918, p. 128; the Etiology of Lichen Planus.

[5] Montgomery and Culver, Jour. Cut. Dis., 1919, xxxvii, p. 242; two brothers affected.

[6] Adamson, Brit. Jour. Derm., 1920, xxxii, p. 1, 3 cases of lichen planus in children.

The fact that lesions develop along scratch-lines in predisposed individuals leads Jacquet to state that lichen planus is always traumatic, and found in individuals with a diminished vasomotor tonus, resulting from some disturbance of the nervous centers. Hallopeau and Jomier,[2] on the other hand, bring forward as evidence of the parasitic origin of the disease a case in which lichen planus developed along scratch-marks in an individual who never had had the disease. A similar case was reported by West,[3] in which the scratch-marks were produced by a cat.

Pathology.—The genesis of the disease is not fully explained. Many observers believe that lichen planus is a constitutional disease with cutaneous manifestations, produced by an unknown toxic agent acting on the nerve centers. Robinson[4] first clearly showed the pathological distinction between lichen ruber and lichen planus. His observations have been confirmed by those of Boeck, Kaposi, Touton, Weyl, and others. Among reporters on the histopathology of the disease may be mentioned Crocker, Török,[5] Joseph,[6] Pinkus[7] Fordyce,[8] Engman,[9] Sabouraud,[10] and others.

The histopathology of lichen planus is characteristic and shows unusual uniformity in all types, notwithstanding the wide variation in clinical appearances. In all cases there is found a well-defined cellular infiltration in the upper part of the corium, consisting chiefly of connective-tissue cells and lymphocytes, associated with edema. In the epidermis hypertrophy is noted, as evidenced by acanthosis, hyperkeratosis, with associated edema, and occasionally a few migrated leukocytes. In detail, there is dilatation of the vessels and lymph spaces of the papillary and subpapillary region, with edema, and a sharply outlined cellular infiltration, consisting chiefly of connective-tissue cells and lymphocytes. Occasionally, plasma-, mast-, and multinuclear-cells and polymorphonuclear leukocytes are sparingly found. Giant-cells are rarely described. The dense cellular mass may at times be so closely associated with the epidermis as to interfere with the continuity of the basal layer. The epidermis shows edema, and marked hypertrophy of the rete (acanthosis), of the granular layer, and of the stratum corneum (hyperkeratosis). Colloid degeneration occurs in certain of the epithelial cells. A plausible explanation of the process of umbilication is made by Crocker and Fordyce, to the effect that the thickened, horny layer sinks into the rete in a funnel-shaped manner, and when eliminated leaves the depression or umbilication.

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[1] Annales, 1906, s. iv, vii, p. 420.

[2] Ibid., 1903, s. iv, iv, p. 352.

[3] Brit. Jour. Derm., 1897, ix, p. 162.

[4] Jour. Cut. Dis., 1889, vii, pp. 41, 81.

[5] Jour. Mal. Cut., 1889, s. vi, i, p. 162 (with bibliography).

[6] Archiv, 1897, xxxviii, p. 3.

[7] Ibid., 1902, lx, p. 163 (3 plates and references to literature).

[8] Jour. Cut. Dis., 1910, xxviii, p. 57.

[9] Loc. cit.

[10] Annales, October, 1910, p. 491; abstr. Brit. Jour. Derm., 1911, xxiii, p. 164.

In the hypertrophic form the above described process is exaggerated, the epidermal changes being more marked, and the cellular infiltration extending more deeply into the corium, where, in addition, increase in the collagenous tissue and newly-formed vessels are found.

The histology of the lesions of the atrophic form, as studied by the author,[1] agrees with the original of Darier and others since, and consists in the main of a sclerosis of the papillary and subpapillary layers of the corium, and a deeply situated cellular infiltration, with, in addition, the horny plugs extending into the sweat-ducts. In the annular variety Engman describes the usual lichen planus picture in the active periphery of the ring, occurring, however, in a mild degree. Nearer the center regressive changes are noted, while in the center a regenerative process is in progress.

The histology of the mucous-membrane lesions corresponds closely to that of the cutaneous lesions.

Joseph,[2] Whitfield, and others have reported the formation of small vesicle-like cavities in the basal layer. Joseph explains the umbilication of the papule by absorption of these pseudo-vesicles.

Diagnosis.—The diagnosis rests upon the characteristic features heretofore described. Thus, in its size, apex, color, and course the papule of papular eczema is quite different from that described above, being brighter, redder, more acuminate at the apex, and much more often followed or accompanied by catarrhal symptoms in the skin. In psoriasis punctata the scales are abundant and readily removed; and the individual lesions are increased rapidly by peripheral extension, far beyond the fullest development of the papule of lichen planus. The papular syphiloderm is not, as a rule, pruritic, not flattened when minute, not polygonal in shape, and not covered with a closely adherent, horny scale; and it always occurs in patients in whom careful investigation discloses other symptoms of the disease (mucous patches, adenopathy, etc.). The history and course of the disease will determine the diagnosis.

Chronic lesions of lichen planus on the legs (obtuse, verrucous, hypertrophic) have been confused with the condition of the same parts developed in Kaposi's *multiple idiopathic pigmented sarcoma*. In the disorder last named, the elephantiasic aspect of the limb, the infiltration of the integument, especially at the root of the toes, and the characteristic roundish nodules springing from the general surface, suffice to render the diagnosis facile.

The distinctions noted above in connection with lichenification of patches of chronic inflammation of the skin are not to be disregarded.

Treatment.—Systemic treatment depends upon the condition of the patient. As many of the subjects of lichen planus are neurotic, neurasthenic, or suffering from other depressing or debilitating conditions, it follows that in many instances it is necessary carefully to regulate the diet, habits of rest, sleep, and exercise, and to administer tonics, cod-liver oil, and other remedies which will build up the general health. In some instances a change of climate, scene, and occupation is of the greatest value.

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[1] Loc. cit.

[2] Loc. cit.

Arsenic, though sometimes causing an aggravation of the symptoms in acute cases, is a valuable remedy in many subacute or chronic and extensive cases of the disease. It may be given as directed for the treatment of psoriasis. Mercury, in the form of biniodid, bichlorid, or the protiodid, is increasingly recognized as of unquestioned value in many cases. The protiodid, grain $\frac{1}{6}$ (0.01), with or without arsenic trioxid, grain $\frac{1}{20}$ (0.0033), may be given three times a day. Excellent results have been obtained by the author with the hypodermatic use of the bichlorid of mercury. Deep injections into the muscle are given in the same manner as in syphilis. The dosage varies from $\frac{1}{8}$ to $\frac{1}{4}$ of a grain (0.008 to 0.016) every second day. Usually a dozen injections are sufficient to relieve an ordinary case. Little advises énésol, a combination of mercury and arsenic as an intramuscular injection twice weekly in the dosage of 2 c.c. Crocker recommends the use of salicin in 15-grain (1.0) doses three times a day, and large doses of quinin in an effervescent mixture. Tilbury Fox and Robinson found the alkaline diuretics, taken well diluted after meals, of value, especially in the generalized hyperemic cases. For very acute cases, we have found the remedy of value at times in relieving excessive itching. Aspirin in 5-grain (0.33) doses may be used for the same purpose. Hartzell[1] advocates the employment of the salicylates. Pernet[2] reported a case of acute lichen planus rapidly cured by removal of 7½ c.c. of spinal fluid by a spinal puncture. He further reports Ravaut and Thibierge,[3] of Paris, as having worked along the same lines in treatment.

Local treatment should be directed toward the protection of the skin and the relief of itching. For many cases the use of a paste and dusting-powder, as described in the treatment of eczema and psoriasis, gives satisfactory results. A paste containing equal parts of lanolin, vaseline, zinc-oxid, and talcum, with from 1 to 3 per cent. of salicylic acid, is usually effective. In very acute and extensive cases, more relief sometimes is obtained by the use of the soothing lotions and dusting-powders recommended for the treatment of the acute stages of eczema. The same care should be taken as in eczema to have the clothing next the skin of soft cotton or linen. In many instances bathing once a day in tepid oatmeal- or bran-water, with or without the addition of an alkali, may precede the application of the paste or other remedy. Some patients, especially those with much scaling and infiltration of the skin, are made more comfortable with the use of ointments than with pastes. In subacute and chronic cases tar, in the form of lotion, ointment, or paste, is often of value. Directions for its use are given in the section on Eczema. For stubborn patches the treatment differs little from that recommended for inveterate psoriasis. For hypertrophic areas, salicylic acid is most effective. It may be applied in a paste or ointment containing from 30 to 60 grains (2.–4.) to the ounce (30.); or, better, it may be dissolved in equal parts of alcohol and ether, and the solution painted on the patch. The alcohol and ether evaporate and leave the acid in contact with the lesion. After a sufficient amount has been applied, the whole may be covered with adhesive plaster. The dressing should be changed every day or two, and when the part becomes greatly inflamed a soothing dressing should be substituted. Brocq and Jacquet recommend the daily use of a tepid douche for from two to ten minutes at a time, alternated with the application for a few seconds of a cold spray.

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[1] Jour. Amer. Med. Assoc., July 20, 1907, p. 225.

[2] Brit. Jour. Derm., 1913, xxv, p. 261.

[3] Annals, 1913, p. 461.

For chronic cases with much infiltration, the *x*-rays are indicated. We have used the method, in conjunction with other treatment, in a large number of cases with decided improvement, including relief of itching in all, and unusually rapid recovery. The number of exposures in each case varied from two to nine, and the technique was that commonly employed for psoriasis.

Prognosis.—The prognosis is in general favorable, since even cases of long standing are relieved when the subjects of the disease are placed under conditions favorable for recovery. When the patient is neurasthenic, the eruptive symptoms may persist for years, accompanied by intense itching and a consequent teasing of the nervous centers. In this class of subjects it is generally well to make a guarded prognosis, and to pronounce upon the future with reserve.

LICHENIFICATION.[1]

Synonyms.—Lichen simplex chronica (Vidal). Fr., *Névrodermite.*

Definition.—This term was applied by Brocq to denote a condition in the skin where the normal lines are increased and the skin becomes thickened, producing patches resembling lichen planus.

Symptoms.—The disorder may be local, or more or less diffuse. Early there is intermittent itching in the area, which induces rubbing and scratching, this being followed by various changes in the skin. It becomes darker in color and thickened, the normal lines being increased, producing flat, irregular papules, some of which have a faceted appearance. The surface of a fully formed patch is divided into more or less elevated, triangular, square, or irregular areas, presenting a mosaic-like appearance. Moderate scaling occurs. The patches vary in shape, being oval, irregular, or angular in outline. They occur most frequently over the nape of the neck, the upper and internal surfaces of the thighs, the loins, the anterior and external part of the leg, the scrotum, the vulva, the popliteal and axillary folds, and on the palms and soles.

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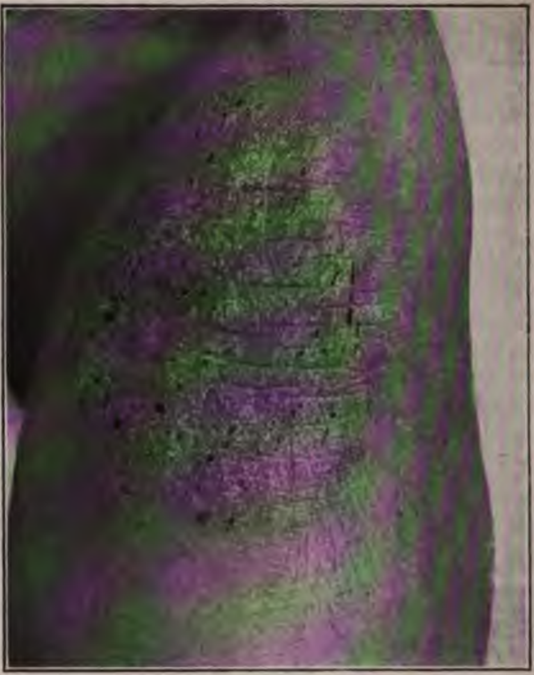
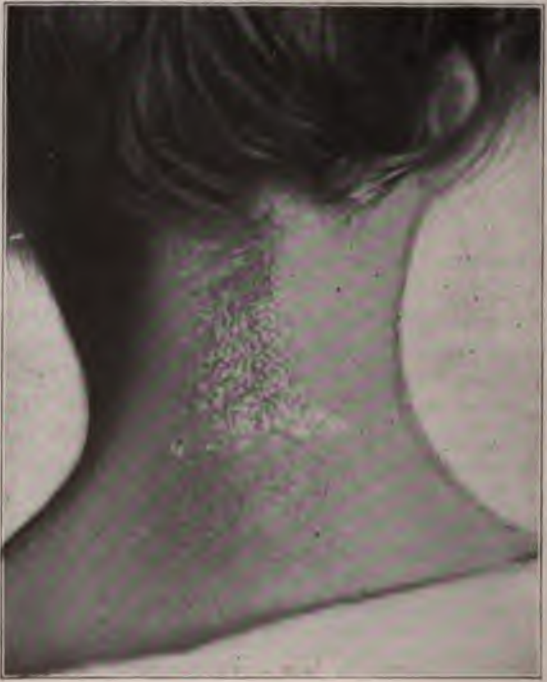
[1] For full discussion of the subject, see Brocq's chapter on "Les Lichens," La Pratique Derm., Tome iii, p. 119; also see Wise, Jour. Cut. Dis., 1919, xxxvii, pp. 590 and 798. The Neurodermatoses and Pseudolichens: A consideration of their nosological and clinical features.

Fig. 113



Neurodermatitis. (Wise.)

Fig. 114



Lichenification.

They may be single or multiple, and they present a tendency to symmetrical distribution. The itching is intense, the paroxysms being worse at night.

In the generalized cases the skin becomes discolored and pigmented, and interspersed here and there are patches similar to those described above, with papules irregularly distributed over the surface. A group of cases described by Fox,[1] Fordyce,[2] Haase,[3] and Withers,[4] presenting a lichenoid eruption in the axillary and pubic regions, closely resembles the group of cases above described. In these cases the eruption is described as being composed of numerous small, firm, smooth, and rounded papules, which are aggregated, and form deeply infiltrated patches, slightly reddened or of the normal hue of the skin. Intense itching leads to scratching, which produces secondary changes.[5]

The disorder occurs in neurotic individuals, and in those who have undergone nervous strain. Many of the etiological factors mentioned in connection with lichen planus apply to this disorder. The disease is persistent, and when relieved tends to recur, the axillary cases having been particularly resistant to treatment. A variety of lichenification may follow certain inflammatory conditions of the skin, such as eczema.

Histopathology.—The principal changes consist in hyperkeratosis, acanthosis, cellular infiltration, and edema in the papillary layer of the corium, without much change in the bloodvessels. In the axillary cases described by Fordyce and Haase, similar changes were noted, with the additional involvement of the sweat-coils. These were dilated; some of the tubules contained partial or complete casts, and their cells had undergone parenchymatous degeneration. An infiltration of lymphocytes and plasma-cells occurred about the bloodvessels and in the region of the coils. In other places the collagenous tissue had undergone mucoid degeneration.

Diagnosis.—The disorder is to be differentiated from lichen planus and papular eczema. In the former condition the patches have a better definition; their color is more marked, being of a violaceous or reddish tint; the flat-topped, shining papules are more definite; white striæ over the papules are likely to be present; and the sites of predilection, over the flexors of the wrists and about the ankles, are different.

Treatment.—The treatment is similar to that employed in chronic eczema, and consists in stimulating applications containing tar, salicylic acid or resorcin. For localized patches, we have found radiotherapy the most successful method of treatment. The general management is that indicated by the individual case.

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[1] Jour. Cut. Dis., 1902, xx, p. 1: Two cases of a rare papular disease affecting the axillary regions (histopathologic report by Fordyce).

[2] Trans. Amer. Derm. Assoc., 1908, p. 118: A chronic, itching, papular eruption of the axillæ and pubes: its relation to neurodermatitis.

[3] Jour. Amer. Med. Assoc., January 21, 1911: A chronic, itching, papular eruption of the axillæ, pubes, and breast.

[4] Archiv. of Derm. and Syph., 1920, i, p. 8.

[5] Withers, Archiv of Derm., 1920, n.s. vol. i, p. 8; Chronic Itching Papular Eruption of the Axillæ and Pubes (Fordyce). Four cases reported; one histological study.

LICHEN NITIDUS.

This disorder was first described in 1907 by Pinkus.[1] A clinical and histological study of the disease has since been made by Kyrle and McDonagh,[2] Arndt,[3] Sutton,[4] and Reines.[5]

Symptoms.—The disorder resembles lichen planus to a moderate degree. The lesions are small, glistening papules or nodules, sharply defined, roughly circular or polygonal in outline, slightly raised above the level of the surrounding skin, and pinkish in color or of the same hue as the surrounding skin. They occur in groups, without coalescence of the individual papules. In the center of a papule or nodule a fine aperture has been described. No particular arrangement of the lesions occurs. The favorite sites have been the genital region, the abdomen, breast, flexors of the elbows, and palms of the hands. As a rule, the lesions remain localized, but a more general distribution has been noted. Subjective sensations are absent, and it is suggested that the disease has so long remained undescribed for this reason. The disorder is chronic.

Etiology.—The disorder has occurred, in most cases, in men, only one case having been reported in a woman. Tuberculosis is suggested as the etiological factor, from the histological architecture. However, animal experiments have thus far been negative. In the case described by Kyrle and McDonagh, a positive reaction was obtained after the injection of tuberculin.

Pathology.—The pathogenesis of the disease is unknown. The histopathology appears to be characteristic, the disease belonging to the granulomata. The cellular infiltration is situated chiefly in the papillary and subpapillary layers of the corium. The infiltration is dense and well defined, and consists of giant-cells, epithelioid, and mononuclear round cells. The collagen in the affected area shows degenerative changes, and in certain cases has almost entirely disappeared in the infiltrated areas. The epithelial changes are secondary. Directly over the nodule, the epidermis is thinned, but on either side prolongation of the epithelial pegs is noted. No tubercle bacilli or caseous degeneration has been found, and animal experiments have been negative.

Diagnosis.—The disease is to be distinguished chiefly from flat warts and lichen planus. From the former it may be distinguished by the small size of the lesions, their greater number, and their distribution; from the latter by the absence of itching, absence of color, the distribution of the lesions, and their arrangement.

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[1] Ueber eine neue knötchenförmige Hauteruption: Lichen nitidus. Archiv, lxxxv, p. 11.

[2] Brit. Jour. Derm., 1909, xxi, p. 339.

[3] Zeitschrift, xvi, p. 551; abstr. Brit. Jour. Derm., 1910, xxii, p. 30.

[4] Jour. Cut. Dis., 1910, xxviii, p. 597, and Archiv. of Derm. and Syph., 1920, ii, p. 1.

[5] Med. Klin., 1910, xxx; abstr. Brit. Jour. Derm., 1911, xxiii, p. 299.

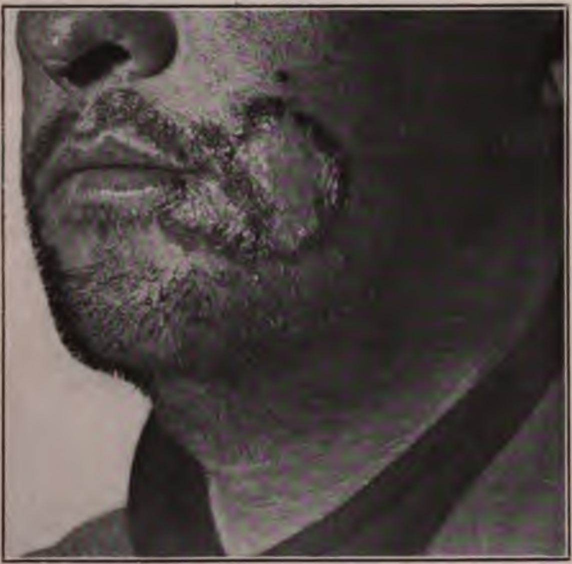
Treatment.—Sutton reports the disappearance of lesions in his first case with the use of a salicylic acid and resorcin ointment, used alternately with a benzoinated zinc-oxid ointment.

IMPETIGO.

Synonyms.—Ger., *Eiterflechte.* Fr., *Impétigo, Darte humide.*

The various forms of impetigo described by older writers are now otherwise classified, leaving impetigo contagiosa, impetigo of Bockhart, and impetigo herpetiformis as the only diseases designated by this title. There is little connection between these three, except that they are pustular dermatoses. The impetigo of Bockhart is a superficial pustular folliculitis, and impetigo herpetiformis is a rare and grave disorder.

FIG. 115



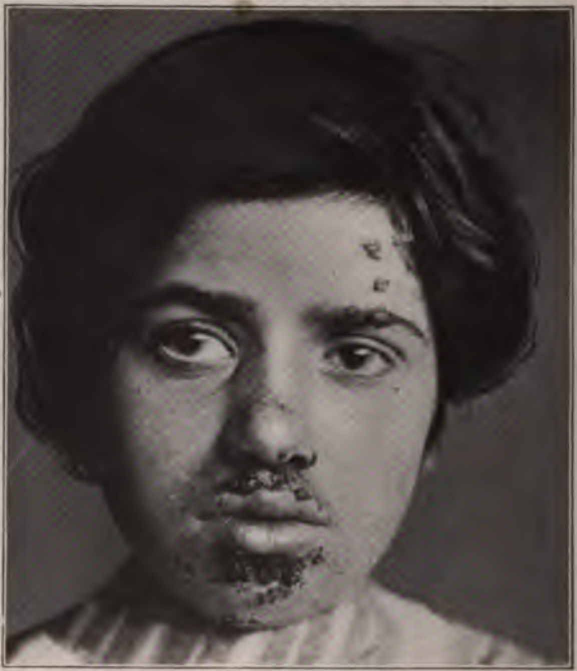
Annular lesions of impetigo.

Impetigo Contagiosa (Tilbury Fox).—Impetigo contagiosa is a contagious disease of the skin, characterized by vesicles, pustules, and superficial crusts, usually occurring on exposed portions (the face and hands), devoid of subjective sensations, and terminating without sequels.

Symptoms.—The early lesion is a flat and erythematous spot or a vesicle, the latter rapidly changing into a pustule, which subsequently dries, forming a superficial crust. In most cases the lesions are located on the face, ears, neck, and hands, but any part of the cutaneous surface may be attacked, including the mucous membrane.

The crusts are gummy-like, yellowish, or occasionally darker colored when admixed with blood. They are very superficial and appear to be stuck on the skin. They extend somewhat beyond the borders of the original lesion, and their edges are sometimes slightly curled upward. Underneath the crusts there is a superficial erosion, having a distinct outline. When the crust is removed, there is presented simply a reddened, weeping surface. The lesions vary from pinhead- to dime-sized or larger; rarely there is a sprinkling of red papules associated. In a given case all the lesions above described are usually present. In children, pustular lesions of the fingers are not uncommonly associated with impetigo of the face. Stomatitis is observed from time to time. Montgomery[1] reports a group of cases of involvement of the lips, mouth, throat, nostrils, eyelids, and conjunctiva.

Fig. 116



Impetigo contagiosa.

Impetigo Contagiosa Gyrata.—This is a clinical variety of the disease in which the lesions spread peripherally, clearing in the center, forming circles, and by coalescence with other lesions produce a gyrate appearance. Schamberg[2] reported a remarkable instance of this type. Crocker,[3] in 1895, described this variety as having been seen in England only during the past two years. While this form is uncommon, it is met with sufficiently often to be easily recognized.

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[1] Jour. Cut. Dis., 1910, xxviii, p. 445.

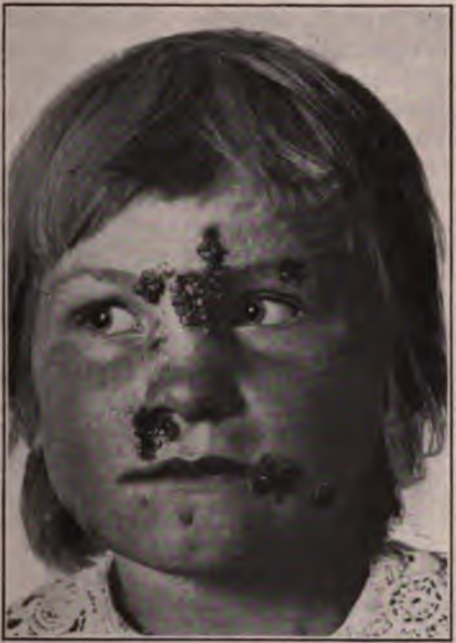
[2] Ibid., 1896, xiv, p. 169.

[3] Brit. Med. Jour., November 2, 1895.

Impetigo Contagiosa Bullosa.—This is a descriptive term for that variety of the disease in which bullous formation predominates. It is uncommon in adults except in warm countries. Sir Patrick Manson[1] has described this form under the title: "Pemphigus Contagiosus Occurring in Children as well as Adults." Corlett[2] reported a very interesting epidemic of this type occurring in soldiers recently returned from Florida, after the close of the Spanish-American war. In several carefully observed cases Corlett described the evolution of the lesions in the main as follows:

The lesions begin with one or more small, reddish spots, from pinhead to split-pea in size. After about twelve hours, the epidermis becomes slightly raised with a clear, serous fluid. The vesicle thus formed extends peripherally, commonly attaining a size varying from 1 to 5 c.c. in diameter. The epidermic covering of the bulla is thin, and rubbing of the clothing is sufficient to cause its rupture, leaving a shrivelled tent, containing a serous fluid in its folds. About the third day the contents of unruptured vesicles become opaline or tinged with streaks of yellow, the latter process beginning below and extending upward. The entire contents rarely become purulent. The bleb rises from the sound skin, in most instances, without an areola or other

FIG. 117



Impetigo contagiosa showing the "stuck on" crusts.

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[1] Manson on Tropical Diseases, 1898.

[2] Cleveland Jour. of Med., 1898, iii, p. 513.

sign of inflammation. At first fully distended, after maturity it becomes flaccid; at other times the blebs are round, oval, or horseshoe-shaped. In all instances there is marked tendency to extend peripherally, and after rupture the distal margin is the only part distended with fluid. At times the center heals, while the serous undermining of the epidermis continues at the periphery, giving rise to the regular ring or gyrate lesions. When the epidermis is removed, there appears a moist area, at first reddish and glazed, which later becomes covered with a light coating of friable crusts or tissue-paper-like scales. After healing, slight pigmentation remains for a month or so.

The location of lesions of this type is the face, the hands, the axillæ, and, finally, a generalized distribution.

In older children and adults, a bullous impetigo may follow vaccination,[1] or may occur independently, this form having been seen by the author, particularly in public practice, the bullæ very closely simulating those seen ordinarily in pemphigus. Engman[2] states that bullous impetigo is not uncommon in St. Louis, particularly in the summer. In infants the disease occurs sporadically and epidemically. It is comparatively common,[3] and is known as *Pemphigus neonatorum.* It frequently occurs in epidemics in obstetric wards of public institutions, and in foundling homes, and many cases have been traced to the practice of certain midwives.

The lesions develop at variable periods after birth, beginning on any part of the body and spreading to other portions. They consist of vesicles and bullæ, usually situated on an erythematous base and occurring in various sizes, with serous or purulent contents. The lesions often burst before reaching maturity, the area meantime spreading over a space with a diameter of several centimeters. After bursting, the areas of involvement spread, with centrifugal denudation of the epidermis. The fluid furnished by the lesions is scanty or abundant, of a yellow or (especially in cases that have proved fatal) grayish tinge. In mild cases the lesions are few and constitutional symptoms absent. In others there is a rapid development of the lesions over large surfaces, with an early fatal termination. Rarely there are constitutional symptoms, including inappetence, elevation of temperature, diarrhea, and exhaustion. Cole and Ruh[4] believe pemphigus neonatorum, so called, should not be classed with impetigo because of bacteriological findings.

The several clinical pictures of impetigo differ on account of the greater or lesser diffusion of the contagious elements in each case; also, the vulnerability of the skin, largely expressed by the age of the patient, and also by climatic conditions, the serious extensive and bullous form occurring in warm or hot climates. In the ordinary case there may only be a few isolated, pea-sized or larger vesico-pustules on one hand; or many may be scattered about the mouth and lips; or dense, greenish crusts may succeed such lesions over the occiput or scalp; or there may be much larger pustulo-bullous lesions over the legs, which are torn, scratched, and thickly covered with pustular or hemorrhagic incrustations.

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[1] Elliot, Jour. Cut. Dis., 1894, xii, p. 194.

[2] Ibid., 1901, xix, p. 180 (with extensive bacteriological bibliography).

[3] Foerster, Jour. Amer Med. Assoc., 1909, liii, p. 358 (with discussion of the relationship between impetigo contagiosa and pemphigus neonatorum, and literature); Biddle, Jour. Cut. Dis., 1914, xxxii, p. 268 (a report of two epidemics of so-called pemphigus neonatorum).

[4] Trans. Sec. on Derm., Amer. Med. Assoc., 1914, p. 299. Pemphigoid of the Newborn, Pemphigus Neonatorum. An epidemic of nine cases. Discussion of other reported epidemics - Bibliography.

Etiology.—The disease is a pus infection, the result of the transmission to the skin, through the medium of the finger-nail, filthy or otherwise, of an infection of streptococci, staphylococci, or both. In children the disease is often associated with pyogenic nasal infection. It may be conveyed from one child to another, and hence is frequently contracted in schools. Women contract the disease from children, while in men the most frequent source of infection is the barber-shop. The eruption often occurs during convalescence from a more or less actively contagious disease. The antecedence of some fever in many cases is admitted by all observers. Duhring and Fox have seen it follow vaccinia. It may occur typically in a series of children, each of whom is convalescent from varicella.

Montgomery and Morrow[1] reported a large increase in impetigo following the great San Francisco fire, and suggested that the spread was due to flies, opportunity for infection being presented by the disorganized state of society at that time. In the infantile and epidemic cases infection is simply transmitted either directly or by mediate objects. Even with strict precautions the disease will spread in an obstetrical ward when once started, and the epidemics following in the wake of midwives indicate the factor there.

A large number of observers have examined the lesions bacteriologically, and in the major number of cases staphylococci have been found. Griffon and Balzer,[2] Sabouraud,[3] Leroux,[4] Gilchrist,[5] and many others have found streptococci in unruptured vesicles. Engman[6] obtained *Staphylococcus pyogenes aureus* in pure culture in 7 out of 8 cases; and in the infant variety Foerster[7] also obtained chiefly *Staphylococcus pyogenes aureus*. Cole and Ruh[8] isolated *Staphylococcus aureus* from unbroken vesicles in their group of cases. Clegg and Wherry[9] isolated a diplococcus in 5 cases studied in the Civil Hospital at Manila. This organism was identical with *Micrococcus pemphigi neonatorum*.[10] Dohi and Dohi[11] describe two forms found in Japan—one produced by a white staphylococcus, the other by a streptococcus. Both forms may be infected secondarily by a yellow staphylococcus.

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[1] Jour. Cut. Dis., 1909, xxvii, p. 435.

[2] La Presse méd., 1897, lix, p. 130.

[3] La Pratique Derm., 1901, ii, p. 878.

[4] Annales, 1893, iv, p. 290.

[5] Trans. Amer. Derm. Assoc., 1899, p. 87.

[6] Loc. cit.

[7] Loc. cit.

[8] Loc. cit.

[9] Jour. Infect. Dis., 1906, p. 165.

[10] Almquist, Ztschr. f. Hyg., 1891, p. 253.

[11] Archiv, 1912, cxi, p. 629; abstr. Jour. Cut. Dis., 1913, xxxi, p. 123.

From a survey of all the findings, it is apparent that both staphylococci and streptococci are concerned etiologically in the production of the disease.

Pathology.—The cause of the disease being an infectious one, a pathological study has been made (largely of the vesicles, bullæ and crusts) to determine chiefly whether a particular organism could be implicated. The histology of an early lesion is well described by Gilchrist.[1] The contents of the vesicle consisted of a large number of polynuclear leukocytes, a considerable number of round mononuclear cells, a few detached epithelial cells, a small quantity of fibrin, and a large quantity of coagulated albumin (serum). There was a collection of polynuclear cells in the vesicle; and in the collection were found, on special staining, a large number of cocci, which, on culture from other vesicles, proved to be *Staphylococcus pyogenes aureus.*

The vesicle was situated between the horny and mucous layers. The stratum mucosum was swollen, and numerous polynuclear leukocytes were found traversing this layer. The corium, chiefly in the upper part, showed an acute inflammatory condition, indicated by dilated vessels, serous infiltration, increase in the number of round and mononuclear cells, and numbers of migrated polynuclear leukocytes. In cultures taken at this time Gilchrist only succeeded in obtaining *Staphylococcus aureus* and *albus.* In his later work, as above mentioned, he was able to obtain a streptococcus.

Unna[2] states that the cocci are never found in leukocytes, and that they are always extracellular. Engman,[3] on the contrary, found both large masses and small numbers of cocci within the bodies of leukocytes; from which fact the latter draws the conclusion that the virulence of the organism is mild.

Dewèvre[4] reports a number of successful inoculations and autoinoculations practised with the contents of the vesico-pustule, with finely powdered impetiginous crusts, and with the products of scraping the subjacent erosion. In 1884, Dr. Hyde reproduced an almost typical vesico-pustule upon the left forearm by inoculation with the moistened débris of crusts. This inoculation was done in the clinic, the crusts being taken from typical lesions upon the face of a young girl inoculated, while under observation, from the lesions of exactly similar character on the face of her twin sister. The lesion on the forearm produced a characteristic crust, which in seven days was also used for the inoculation of two students then present at the clinic, in one of whom there was no result and in the other an abortive lesion.

Diagnosis.—The discrete vesicular, pustular and crusted lesions of impetigo are readily distinguished from pustular eczema by the presence in the latter of infiltration, weeping, itching and the occurrence of larger patches.

In ecthyma the pustules are in appearance much more formidable than those of impetigo, in consequence of their size, depth, inflammatory base, areola, flat, hard and bulky crust, and erosive action upon the skin.

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[1] Duhring's Cutaneous Medicine, vol. ii, p. 431.

[2] Histopathology, p. 189.

[3] Loc. cit.

[4] Arch. de Méd. et de Pharm. mil., 1885, vi, p. 210.

In varicella the lesions are small, much more widely distributed over the body, and are vesicular only, rarely bullous. In pemphigus and herpes iris the seat, character, and period of evolution of the lesions suffice to establish the diagnosis.

From hyphogenous sycosis it is so readily distinguished that the differential points need not be described. The latter is mentioned only for the reason that it represents the disease popularly called "barber's itch;" whereas today large numbers of barber-shop cases of contagious impetigo are erroneously being called, both by physicians and laymen, "barber's itch."

Treatment.—Pustules, when present, are to be opened with an aseptic needle, the purulent contents gently removed by irrigation with a mild antiseptic solution, such as boric acid, and the lesion dressed with a mild antiseptic ointment, such as a 1 to 4 per cent. ointment of ammoniated mercury. In the ordinary cases in children, this procedure will readily clear up the trouble. In adults, in the common types about the face and hands, the procedure may be as follows: Bathe the areas thoroughly in the evening with a 1 to 2000 bichlorid solution. After the bath apply an ammoniated mercury ointment in the strength of 3 to 5 per cent. In the morning repeat the bichlorid bath, after which apply a dusting-powder composed of the mild chlorid of mercury 1 drachm (4.), boric acid 1 drachm (4.), and talcum powder 6 drachms (24.).

In the epidemic infant variety, Foerster obtained good results by opening the vesicles and bullæ and applying a 2 per cent. ammoniated mercury ointment. The individual lesions were dressed to prevent their spread. In addition, a general bath was given in a warm permanganate of potassium solution. This appeared to be of value also in preventing autoinoculation. In extensive cases external heat, with strychnin and brandy, was also recommended as a supporting measure. Cole and Ruh highly recommended autogenous vaccine.

Ecthyma (Ger., *Ekthyma, Eiterblase*).—Ecthyma is a pustular infection of the skin of the same nature as impetigo, but of deeper situation. It is inoculable and autoinoculable, and characterized chiefly by the formation of large, flat, variously colored crusts.

Symptoms.—The disease occurs most frequently on the lower extremities, chiefly below the knees; in children about the buttocks. The inflammatory process is comparatively deep-seated.

The primary lesion is a vesicle or a vesico-pustule, situated on an inflamed base, the inflammatory process soon extending deeply. The lesion spreads peripherally, and in the course of several more days crust-formation occurs. As the crust dries, it becomes dark-brownish in color and firmly attached at the margins. Beneath the crust a superficial ulcerative process proceeds, the margin of this area being elevated, producing a crater-like appearance when the crust is removed. The elevation of the margin causes the shallow ulcer to appear deep.

In the course of from two to four weeks, healing occurs beneath the crust, the latter being exfoliated, leaving usually a slight scar, with more or less pigmentation.

In certain debilitated states, a gangrenous ulcer appears in place of the one described, which is more deeply situated, causes greater destruction of tissue, and is followed by more intense sequels.

The subjective phenomena are sensations of heat, burning, pain, and soreness. In certain cases there may be accompanying lymphangitis or adenopathy. In public practice a mixture of symptoms may be present, due to trauma and to autoinfection from scratching.

The disorder was common and presented severe symptoms among soldiers during the late World War. According to MacCormac[1] it was characterized by deep ulcers with undermined edges covered with heavy black crusts. These usually were situated on the legs and thighs but occasionally were generally distributed. In certain instances the lesions subsequently became papillomatous or verrucous.

Etiology.—Among predisposing factors should be mentioned a loss of resistance through ill health. The infection, therefore, occasionally follows anemia, asthenia, and the convalescence from many diseases. It is a disease usually seen in the cachectic and poorly nourished of the public-patient class and rarely in private practice. Improper and insufficient food, bad hygiene, over-work, and uncleanliness are all predisposing factors. It occurred as a frequent and serious complication in many cases of scabies and pediculosis during the war.[2] The exciting cause is the same as that of impetigo, and investigators disagree as to whether it is the streptococcus or the staphylococcus. A larger number of microörganisms have been isolated than in impetigo. The identity of cause between these two disorders is frequently seen by their presence in an individual at the same time, typical lesions of impetigo being present on the face, with those of ecthyma on the limbs below the knees. In Corlett's[3] cases occurring in soldiers this fact was well illustrated.

Balzer and Griffon,[4] in a study of a number of cases of ecthyma, found in all cases streptococci in the form of diplococci-like fine grains. When cultivated, this organism presented the features of *Streptococcus pyogenes*, and experimentally it was pathogenic.

A streptococcus of the fecalis type was demonstrated in several of MacCormac's cases by Capt. Henery.

Pathology.—The pustule of the disease differs from the pustule of eczema or the pustule of impetigo in the severer exudative process by which it is produced, and in its limitation to the exact site of external irritation. Early there is found in the corium the usual evidences of inflammation—dilated bloodvessels, with perivascular cell-infiltration, and migration of leukocytes up into the epidermis. In severe cases, necrosis occurs, destroying the papillary layer, the result being a cicatrix, which contracts as it grows older, and which in mild cases is finally barely visible as a minute, cicatriform punctum. According to Unna, the ecthyma pustule, as distinguished from that of impetigo, is less an epidermal abscess than the result of epidermal inflammation, fibrinous at the center and exceedingly edematous at the periphery. The crust contains fibrin and epidermal layers.

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[1] Brit. Jour. Derm., 1917, xxix, p. 141.

[2] *Cf.* Semon and Barber (Brit. Jour. Derm., 1917, xxix, p. 173). They reported pyodermic infections which included impetigo, ecthyma and furunculosis in 631 patients out of a total of 669 in a military hospital for skin diseases during a period of six weeks.

[3] Loc. cit.

[4] Loc. cit.

Sabouraud points out that the original streptococcic infection is often succeeded by a secondary microbian involvement, whereby the staphylococci present are enabled to produce the peripheral lesions of impetigo and furunculosis.

Diagnosis.—Ecthyma is likely to be confounded with the other pustule-producing exudative affections, but as the distinction between them is largely artificial and based upon the severity of the inflammatory process, there is small danger in consequence. Kaposi expresses the truth in his suggestion that there can be but little objection to the employment of the term "ecthyma" when it is desired to characterize precisely the pustular grade of any cutaneous inflammation at a given time. The pustules of variola are "ecthymaform," and many of those seen in syphilis exhibit similar characters; but the history of the general affection should throw light upon the identity of the cutaneous disease. In syphilis, moreover, the ulceration at the base of the lesion exhibits the pronounced features of the syphilitic ulcer in its secretion, floor, edges, base, crust, and career, The crust, in particular, of the flat, pustular syphiloderm has the rupioid, conical appearance which suggests the shell of the oyster, and the underlying ulcer is larger and deeper than in ecthyma. In the furuncle there is usually a central core. In impetigo the pustules are not deep-seated, and there is no ulceration at the base; the crust is superficial, yellowish, firmly adherent, and the lesions are more numerous.

Treatment.—The general treatment of patients affected with ecthyma is a matter of importance. A proper regulation of the food and hygienic surroundings is not to be neglected. Tonics are frequently indispensable, including iron, quinin, and strychnin. The destruction of any pediculi and the cleansing of the skin with soap and water will often be sufficient to effect a great change. This fact is well illustrated in hospital practice, where young patients rapidly improve after a bath, followed by inunction with vaseline, and a few substantial meals of a nutritious character. Crusts are to be removed after soaking with oil or fat; and the floors of the former pustules, after washing with a 5 per cent. solution of phenol in water, should be dressed with an ointment containing from 10 to 15 grains (0.66 to 1.) of ammoniated mercury to the ounce (30.) of lard. Moist dressings of boric acid or aluminium acetate are often serviceable. If the minute basal ulcers are sluggish, they may, after careful cleansing, be touched with a small swab that has been dipped in a 5 per cent. formalin solution or in a solution of mercuric chlorid in tincture of benzoin, 1 grain (0.066) to the ounce (30.). Phenol or boric acid or iodoform may be employed for the same purpose. For the salve mentioned above may be substituted one containing 10 grains (0.66) of calomel, or ½ drachm (2.) of bismuth subnitrate to the ounce (30.) of salve-base.

MacCormack found a 3 per cent. solution of silver nitrate in sweet spirits of nitre painted on the lesions a valuable method of treatment.

Veldt Sore (Natal Sore) (*Barkoo; Barcoo rot of Queensland*).—Under this title has been described a disorder which Crocker[1] reported as somewhat common among the medical officers and soldiers of the English army during the late war in South Africa. It most often attacked cavalrymen. As distinguished from the Natal sore, which was chiefly found in the lower part of that country, the veldt sore was most abundant in the high, barren table-lands. Multiple lesions appeared on the hands, forearms (chiefly on the backs), feet, and legs, but were rare on the face and other portions of the body. A pinhead-sized, itching papule, vesicle, or pustule first appeared, subsequently enlarging and filling with a yellowish serum, which later became turbid, ruptured, and left a small- to large-coin-sized, painful, crusted ulcer, exuding sero-pus, and often accompanied by inflammation of the lymphatics and glands. In some cases the back of the hand was entirely covered.

Martin[2] observed a number of cases among soldiers operating in the desert east of the Suez Canal. He concluded that the disease is primarily a folliculitis.

The disease is produced by pyogenic microörganisms. Castellani and Chalmers[3] found streptococci in the lesions. *Staphylococcus aureus* has been isolated by Harland. Crocker was inclined to believe that the disorder is a semitropical varient of impetigo contagiosa. Martin found the hair follicles infected with *Staphylococcus albus*, chiefly with a few *S. citreus* and *S. aureus*.

Treatment.—The usual treatment of such infected lesions (boric-acid fomentations and ointments) was speedily effectual. Staumes[4] recommended a 4 per cent. salicylic acid ointment. Epilation around the lesions promptly stopped the spread of the infection in Martin's cases.

DERMATITIS VEGETANS.

Under the title of *Pyodermite végétante*, Hallopeau[5] describes five cases of a disease affecting chiefly the scalp, axillæ, genitals, groins, lips, and the mucous membrane of the mouth, in which there appear miliary pustules, which soon are surrounded by a hyperemic base. The pustules appear in successive groups, coalesce, and the area thus formed becomes covered with crusts, beneath which form more or less elevated vegetating surfaces. These patches may increase by peripheral extension, but more commonly by the formation of new pustules at the border. On the mucous membranes, rupture of the pustules is followed frequently by superficial ulcers. The disease yields readily to antiseptic treatment, leaving only a pigmentation, which gradually disappears. Hallopeau considered the disorder a type of local infection spreading by autoinoculation. Similar cases have been reported under the title of *Dermatitis vegetans* by Hartzell,[1] Jamieson,[2] Crocker,[3] King-Smith,[4] Pernet,[5] Pusey,[6] Fordyce and Gottheil,[7] and others. Wende and DeGroat[8] report 6 cases in infants, with 4 collated from the literature. Six of the 10 developed during the course of eczema, 4 independently of any other dermatosis. In these the lesions began as papulo-pustules, followed by crusts and vegetations. The face and scalp were chiefly involved, but in some the wrists, arms, and legs were involved. In all there was the same type of papulo-pustules appearing in groups; the resolution of old lesions with the appearance of new; the production of vegetations; and the disappearance of the disease under antiseptic treatment. The disorder is probably the result of an infection, and not directly related to the eczema which preceded the disease in some of the cases.

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[1] Dis. of the Skin, 3d ed., 1903, p. 1075.

[2] Brit. Med. Jour., June 9, 1917, Barcoo Rot.

[3] Manual of Tropical Med., p. 1468.

[4] Ibid., February 23, 1918, Barcoo Rot (Veld Sore).

[5] Archiv, 1898, xliii, p. 289; and xlv, p. 323.

The disease is distinguished easily from pemphigus vegetans, which it resembles clinically, by the readiness with which it yields to antiseptic treatment, and by its failure to affect the general health of the patient.

FURUNCULUS.

Synonyms.—Furuncle, Boil. Fr., *Furoncle, Clou;* Ger., *Furunkel, Blutgeschwür, Eiterbeule, Eitergeschwür.*

Definition.—A furuncle is a staphylococcic infection of a hair-follicle, producing a painful cellulitis, which terminates in the death of tissue and the expulsion of a necrotic plug. Furunculosis is the succession of furuncles.

Symptoms.—Furuncles commonly begin as tender and painful indurations in the skin or its subjacent tissues, the summit of each nodule soon becoming visible in the epidermis as a reddish punctum. A furuncle is the result of an active inflammatory process, limited to a definite area, and of greatest intensity at the center of the involved mass. This center is often represented by a hair-follicle, the pustule that forms subsequently being perforated by a hair.

More or less rapidly thereafter these symptoms are succeeded by increased redness, heat, and tumefaction, the latter producing a nut- or egg-sized tuberosity, well projected from the surface or fairly imbedded within or beneath the derma. A yellowish point in the center of the erythematous swelling soon announces the occurrence of suppuration. When accidentally or artificially opened at this summit, exit is given to thick, yellowish pus, with which blood may be commingled from the traumatism of neighboring capillaries. The small abscess may then, after discharging its purulent contents for a few days, gradually close by granulation; or may also expel from its cavity a tenacious, pus-covered, yellowish-green slough, known as the "core." This evacuation is usually followed by relief of the tense and throbbing pain which is the well-known subjective characteristic of the furuncle.

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[1] Jour. Cut. Dis., 1901, xix, p. 465 (with histology).

[2] Brit. Jour. Derm., 1902, xiv, p. 407.

[3] Ibid., 1909, xxi, p. 87.

[4] Jour. Cut. Dis., 1910, xxviii, p. 605.

[5] Ibid., 1912, xxx, p. 517.

[6] Ibid., 1906, xxiv, p. 555.

[7] Ibid., 1906, xxiv, p. 543.

[8] Ibid., 1911, xxix, p. 473.

The length of time requisite for the completion of this process varies, with the extent of tissue involved, from a few days to several weeks. Boils may occur in any part of the body, but are most common about the face, the auricular region, the neck, the armpits, the anogenital surfaces, the hips, the buttocks, the breast, and the extremities. They may occur as single or as multiple lesions, or they may succeed each other in crops, especially about the buttocks, trunk, and thighs, for a period of several months. The disease of the skin may produce a constitutional effect, manifested in pyrexia, which is usually encountered only in individuals of irritable constitution, when the furuncles are few and short-lived. There is also a decided chloroanemia, due to pain, fever, purulent drain, irritability of nervous centers, inappetence, and consequent perversion of nutrition.

The sequels of boils are maculations of a violaceous tint, often perceptible in the skin for weeks and even months after disappearance of the lesions; and pinhead- to penny-sized cicatrices, which are permanent.

Etiology.—The exciting cause of furunculus is local infection with one of the pyogenic microörganisms, usually, if not always, *Staphylococcus pyogenes aureus*,[1] though other pus-producing cocci also are found in the lesions. The remote cause is often exceedingly obscure. It is true that boils are encountered in typical subjects of diabetes, of the exanthemata, and of "hospitalism," in whom anemia, asthenia, marasmus, malnutrition, and exhaustion resulting from excesses, from grave general disease, from low fevers, and from nervous strain, play a prominent part. But the reverse is also true.

Scratching, eczema, scabies, other cutaneous diseases, lice, and external irritants of various sorts are responsible for many boils, especially those that are few and not followed by similar lesions. When, however, such sequence occurs, it should not be forgotten that the pus is autoinoculable, and that furuncles, if sufficiently numerous and large, are capable of disturbing the general economy. A collar-button at the back of the neck; the edges of an unyielding corset in one unaccustomed to it; a hard bench; a saddle-tree; a velvet coat-collar sheltering the germs responsible for a previous attack; and many similar articles may be the exciting cause of furuncles.

Account should always be had, in cases of persistent furunculosis, of externally operating poisons. In this category must be included arsenical wall-papers, and the poisons handled in the trades, *e. g.*, by dyers, lead-manufacturers, and others.

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[1] Gilchrist, Johns Hopkins Hosp. Reports, 1903, xiv.

Lastly, it is exceedingly common for patients thus affected to apply to practitioners for remedies intended to "purify the blood;" and, inasmuch as potassium iodid is often prescribed in response to this demand, the original trouble is thus enhanced to a manifold extent. Many cases of furunculosis are instances of boils, resulting originally from external irritation, that have greatly multiplied and finally profoundly affected the system under the impulse of the so-called "blood-purifying" process.

Pathology.—According to Unna, most furuncles begin with an impetiginous lesion due to the inoculation of the pilo-sebaceous follicle with pus-cocci, the organism being, in the majority if not in all instances, *Staphylococcus pyogenes aureus.* The cocci penetrate deeply into the follicle, into ramifications of the sebaceous gland, and into the surrounding tissue. An abscess surrounding the follicle is thus produced, which undergoes a necrosis *en masse*, producing the characteristic central core or slough. It is probable that in some instances the cocci are carried along the lymphvessels to form abscesses about the neighboring follicles and glands. The lanugo hair-follicles are affected much more frequently than those of the stronger hairs.

Diagnosis.—Boils are to be distinguished from carbuncles by the aggravated symptoms of the latter. Circumscribed furuncular abscesses of the groins and the axillæ are not to be confounded with suppurating, sympathetic, or virulent buboes of these regions, associated with genital or extragenital contagious venereal sores. Errors of this sort have been made. Furuncles of the anal and genital regions in point of diagnosis may be significant of surgical affections of the neighboring parts (perineal, periprostatic, periurethral, and scrotal abscesses in men; suppuration of the vulvo-vaginal gland in women).

Treatment.—The debilitated constitution of many patients affected with boils indicates clearly the need of a tonic regimen, including the administration of iron, quinin and strychnin, the mineral acids, and a generous diet of milk, cream, eggs, and fresh meats. To these articles of diet, wines and malt liquors may at times be added with advantage. Change of climate, of diet, of cooks, and of habits of life is most serviceable in cases of prolonged furunculosis. The mineral waters at some health-resorts prove especially valuable for the debility which often results from these disorders. The urine should always be examined for sugar, albumin, and an excess of urates. The internal remedies which possess reputation in this complaint are arsenic, sulphur, the alkalies, tar, fresh yeast in tablespoonful doses, phosphorus, and the syrup of the hypophosphites of calcium, iron, sodium, and potassium.

Calcium sulphid, which was once more highly esteemed than any other of the internal remedies named, is given in doses of $\frac{1}{10}$ to $\frac{1}{5}$ grain (0.0066–0.0133) every three or four hours. It is doubtful whether the drug exerts any influence whatever upon furuncles. In lithemia potassium acetate or citrate is given in large dilution, or the liquor potassæ; in gout colchicum, salol, and the alkalies, including sodium salicylate. No one of these articles, however, may be described as an efficient and certain remedy for the complaint; many cases will progress without hindrance from any or all of them. Fresh brewer's yeast, recommended by Löwenberg, Crocker, Brocq,[1] Desfosses,[2] and others, is sometimes of service. A tablespoonful or less may be given three times a day.

Attempts in the direction of aborting a furuncle by the topical application of the stronger alkalies (aqua ammoniæ) or acids, caustics, cautery, ice, iodin, or phenol, or premature complete excision with the scalpel, occasionally succeed, but often they fail. Boils may be aborted at times by the injection beneath the lesions of from 3 to 6 drops of a 3 per cent. solution of phenol.

The objects of local treatment are to reduce the inflammatory process, allow the free escape of pus, and prevent infection of other follicles in the neighborhood. The surface of the boil and the skin in the neighborhood should be kept thoroughly clean by frequent use of hot water and green soap, and the application at least twice daily of some simple antiseptic solution, such as 50 per cent. alcohol, 1 per cent. phenol lotion, or weak bichlorid solution. Stelwagon[3] recommends for the purpose:

℞—Resorcin.,	gr. xv-xxx;	1–2	
Acidi borici,	ʒjss;	6	
Alcoholis,	f℥j;	30	
Aquæ dest.,	f℥v;	150	M.

Before rupture of the furuncle it may be protected by means of an ointment or paste containing ichthyol, 1 to 2 drachms (4.–8.) to the ounce (30.); or, by protecting the surrounding skin with such an ointment or paste, hot antiseptic applications may be applied to the lesion itself. A convenient and effective dressing at this stage is found in the official cataplasma kaolini, containing sterilized clay, glycerin, and a mild antiseptic. Such a dressing may be continued even after the opening of the furuncle, if care be taken to permit free discharge of the pus.

Jackson[4] recommends boring into the boil, as soon as it has pointed, with a stick sharpened to a fine point, covered with cotton, and saturated with 95 per cent. phenol. The surface and surrounding parts are then cleansed with hydrogen peroxid, or a 1 to 1000 solution of bichlorid of mercury; after which a 5 to 10 per cent. salicylic-acid ointment is applied.

The furuncle should be opened freely with a clean incision when pus has formed, but not before. Violent squeezing of the furuncle to separate its slough or evacuate the contents should never be practised, though it is permissible in some instances to scrape out the contents with a curette. The cavity should be cleansed thoroughly, at least twice a day, with hydrogen peroxid, or with a solution of phenol or mercuric chlorid, and packed with boric-acid, aristol, or other powder. In place of these powders, phenol in crystal or in strong solution may be employed.

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[1] La Presse med., 1899, lxi, p. 45 (with bibliography).

[2] Ibid., 1892, liv, p. 653.

[3] Diseases of the Skin, 7th Ed., p. 411.

[4] Amer. Jour. Med. Sc., June, 1909.

In the chronic recurring lesions about the back of the neck, a moderate course of radiotherapy is a valuable method of treatment. Vaccine therapy is valuable in a certain proportion of cases, especially the chronic forms. Autogenous vaccines are preferred, though stock vaccines are often of much value (for technique, see section on General Therapeutics).

Prognosis.—Eventually, the worst cases are relieved, when unaccompanied by systemic or visceral disorders, and when the circumstances of the sufferer permit him to pursue the most advantageous course (travel, diet, and rest). The resulting cicatrices depend upon the severity of the process. Often they are small and in the course of years become scarcely distinguishable; in exceptional cases they are large, persistent, and disfiguring. Lympius[1] calls attention to the serious and even fatal complications (purulent arthritis, meningitis, thrombosis of frontal veins, septic infarct in lung) which may complicate furunculosis of the face, particularly of the upper lip, owing to the vascularity of the region.

CARBUNCULUS.

Synonyms.—Anthrax Simplex, Carbuncle. Ger., *Karbunkel*, *Brandschwär*; Fr., *Anthrax*.

Definition.—A carbuncle is an acute, flattish, circumscribed, cutaneous and subcutaneous abscess, usually larger than a furuncle, that is due to the presence of staphylococci, and is characterized by dense induration and sloughing, terminating, in favorable cases, by the production of a persistent cicatrix.

Symptoms.—Carbuncles are often preceded by malaise, chill, and pyrexia of severe grades. There is commonly a burning pain at the site of the lesion. In cases in which the carbuncle is formidable and seated upon or near the head, alarming symptoms of prostration, stupor, somnolence, and even coma, may be noted. With and without these concomitants, a dense, dull-red, indurated, and painful phlegmon soon appears, varying in size from that of a small hen's egg to that of an orange and even much larger, involving not only the skin, but also the tissues beneath. Suppuration finally occurs, but the pus is not confined to a single space; it undermines the integument and often through several apertures leaks out indolently to the free surface. The fenestrated or cribriform appearance of the skin covering the carbuncle constitutes in this stage one of its most striking features. Through these apertures may be distinguished the whitish or yellowish pus-soaked sloughs or portions of a single slough, which can at times be extracted through the orifice. Often the entire mass separates in a single slough, involving the skin and subcutaneous tissues, leaving a crateriform ulcer of formidable size, which in favorable cases proceeds to heal by granulation. The resulting cicatrix is at first of a deep violaceous tint and later becomes blanched.

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[1] Deut. med. Wchnschrft., 1899, xxv, p. 474.

The fever which usually accompanies this process may be mild or be severe, or, more commonly in dangerous cases, be of a typhoid character. It results unquestionably from sepsis due to unliberated pus and necrotic tissue, and is naturally most grave in its consequence in patients weakened by previous asthenic disorders. Under these unfavorable circumstances, the carbuncle may spread at the periphery, enclosing islands of necrotic tissue and pus, separated by bridges of empurpled, infiltrated, and yielding skin.

The characteristic lesions of this disease most often appear on the back of the neck, the back of the trunk, and the lateral aspect of the hips and thighs, usually as a single lesion, though occasionally two or three carbuncles of small or of medium size may coexist. The reason for their appearance in the localities named is clear. It is here that the skin is most thick and resistant, and, as a consequence, purulent foci when formed are covered in by the most voluminous layers of the connective tissue of the corium.

Etiology.—Carbuncles are produced by the causes to which reference has already been made as probably effective in the production of boils. Carbuncles and boils may coexist; or the one lesion may follow the other; and there may occur intermediate forms assignable to either class. The disease is encountered more often in men than in women, and in later than in earlier life, simply because the tissues constituting its sites of preference offer in these individuals and at these ages a greater resistance to the exit of pus. The various pyogenic microörganisms found effective in furuncles are the direct exciting factors here. Carbuncles are at times an epiphenomenon in cachexia, diabetes, albuminuria, syphilis, pemphigus, and exfoliative dermatitis.

Pathology.—The pathological anatomy of carbuncle has been well described by Warren,[1] whose observations conclusively show that the inflammatory process here is that seen in the simplest pustule. The special symptoms of carbuncle are due solely to the formation of the phlegmon beneath the dense and extremely thick masses of fibrous tissue found in the back "for the protection of that comparatively defenceless portion of the body." The cell-elements, multiplying with the intensity of the inflammatory process in the subcutaneous adipose tissue, pass upward along the fat-columns, crowd between these, and push along the horizontal clefts branching from either side, infiltrate the derma, pass along the edges of the hair-follicles, fill the papillæ until the latter "balloon" with pus, ooze to the surface through the cribriform aperture in the undermined epidermis, and macerate the bundles of fibrous tissue, relatively intact, that constitute the undetached mass of sloughing tissue.

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[1] The Pathology of Carbuncle, or Anthrax. Cambridge, 1881, p. 15.

The constitutional symptoms in carbuncle (pyemic, septicemic, or sympathetic) are due solely to pus-absorption. The pus-formation is due to the presence of *Staphylococcus pyogenes aureus* and its toxin. Back of all (in the diabetic and the cachectic) lies the favorable soil for multiplication of the microörganism.

Diagnosis.—It follows from what has preceded that carbuncle and furuncle differ solely in the depth of the starting point of the phlegmon, and the density and resisting power of the overlying tissue. The carbuncle, is, therefore, flatter, denser, less rapidly developed, larger, less tender, and more painful; it opens by many rather than by one or two apertures; and is followed by larger sloughs, ulcers, and cicatrices, and occasionally by fatal results.

Carbuncle is readily distinguished microscopically from anthrax by the demonstration of *Bacillus anthracis* in the latter disorder.

Treatment.—Crucial and other deep incisions in the local treatment of carbuncle are certainly inferior in results to the course advocated by Wood[1] and Taylor,[2] whose method is employed in cases with complete success, as follows: A saturated solution of pure phenol is injected with a hypodermatic syringe through the several apertures in every direction into the sloughing tissues. When the orifices are not sufficiently numerous, the point of the needle is thrust through the thinned integument at the summit of the swelling at several points. The pain is severe but short-lived; the tissues are blanched, indurated, and destroyed; the slough in a few days is readily separated after division of its slender fibrous attachments; and the ulcer rapidly contracts, with the sequel of a smaller scar. It is necessary to use pure phenol in saturated solution to prevent absorption of the injected fluid and the resulting toxic effects.

Relief is afforded in many cases by hot borated lotions and fomentations; by carbolated lotions; extraction of the slough wholly or in portions with the forceps; and the subsequent employment of boric acid or the paste recommended in the treatment of furuncles. An excellent method of withdrawing the purulent and sloughing contents of the carbuncle is to apply over it at the proper period an exhausted receiver, such as a common cupping-glass.

Erasion of the entire abscess with a curette and subsequent antiseptic dressing is an accepted radical measure of relief for employment in proper cases.

The antiseptic treatment of a carbuncle, however, furnishes the best results as regards the comfort of the patient and limitation of the disease. By this treatment there is absolutely no surgical interference with the lesion beyond the incisions made for the evacuation of pus. Violent squeezing and manipulation of the carbuncle are interdicted.

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[1] Toledo Med. and Surg. Jour., December, 1880.

[2] Australian Med. Gaz., December 1, 1881.

It is freely powdered with boric acid, over which is laid soft felt cloth thickly spread with any emollient and antiseptic salve, such as the ordinary zinc-salve. Boric acid in powder or iodol, thickly dusted over the carbuncle and covered with antiseptic wool, will also be found a useful dressing.

Constitutional treatment may be demanded in carbunculosis, including the liberal employment of tonics, a generous diet, a strict observance of the rules of hygiene, and stimulants when indicated. Pyrexic, septicemic, pyemic, and adynamic states require the special management of such complications, including cold sponging of the body-surface in fever, and the use of quinin, the mineral acids, and stimulants, with artificially applied heat in the algid condition. The urine should always be examined for sugar and albumin.

Prognosis.—A serious issue need only be anticipated when the complications described above are grave in character or they occur in asthenic constitutions.

MULTIPLE ABSCESSES OF THE SKIN OF INFANTS.[1]

The disorder described by Fox under the above title is not common, but is of sufficiently frequent occurrence to render its recognition necessary.

Symptoms.—The abscesses characteristic of this disorder may occur on any region of the skin, but are found particularly over the buttocks, thighs, and back, and also about the occiput and on the hands and feet. The abdomen and chest are more rarely attacked. In number the lesions may be few or many; as a rule, before the attack subsides, large numbers have been present. It is stated that the whole body may become studded with them, numbering 150 or more. They are situated at different depths, some being superficial and others entirely subcutaneous. They vary in size from that of a pea to a pigeon's egg or larger, and suppurate freely. After rupture and discharge of the contents, the lesions heal, leaving a moderate scar. Some of the deeper lesions may only be felt. In infants of low vitality, whose nutrition has been much interfered with, ulcers or fistulæ may remain. Associated lymphangitis and a suppurative inflammation of the neighboring lymph-glands may occur. Gastro-intestinal disturbance may complicate the disorder, with associated loss of appetite and diarrhea. Cachexia and prostration may supervene and the process be terminated by septicemia or broncho-pneumonia.

Etiology.—The disorder, as a rule, occurs in infants of the poor, who are illy nourished, badly fed, and in unhygienic surroundings, though it may occur in infants of the better class whose vitality has been impaired by other causes. It occurs in both sexes. The direct cause is *Staphylococcus pyogenes*.

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[1] Colcott Fox, Allbutt and Rolleston's System, vol. ix, p. 199, from which the description in this text is largely taken.

Diagnosis.—The disorder is to be differentiated from abscesses due to tuberculosis and from syphilitic gummata. It is differentiated from the former by the absence of the tubercle bacillus and the presence of *Staphylococcus pyogenes* found in deeply situated lesions. In addition, there are no other evidences of tuberculosis than the presence of cachexia. The gummata of syphilis present lesions showing more induration, and are rare in infancy.

Treatment.—The treatment consists in incision of the abscesses, either with a knife or by igni puncture, after which they are dressed in accordance with the principles of surgery. Particular attention is to be paid to the general care of the patient relative to the diet, and, in the way of prophylaxis, strict cleanliness of the entire body-surface.

Prognosis.—The prognosis varies; in mild cases, and even in some of severer grades, recovery takes place. In neglected cases and those with bad surroundings, the prognosis is grave.

PHLEGMONA DIFFUSA (CELLULITIS).

This is a suppurative inflammation of the subcutaneous tissue caused by a streptococcic infection. Frequently the infection enters through a discernible wound of the skin, but it may occur without previous lesion.

Symptoms.—In severe cases the disease begins with a chill and elevation of temperature. Usually, the first evidence of the disease is a stinging sensation at the point of infection, which develops so suddenly and so closely simulates the feeling produced by an insect-bite that the patient insists that he has been stung. The physician should exercise due caution in accepting such statements.

The lesion which first appears is a red nodule situated deeply in the derma or subcutaneous tissue. Movement of the subjacent muscles causes pain. The redness soon spreads until it involves an area the size of the palm of the hand, and the original nodule assumes a bluish color, so that there is presented a bluish nodule surrounded by an area of redness. The outer red area never presents a distinct outline, but blends gradually with the healthy skin. The reddened area is infiltrated, hard, pasty-like; and pits on pressure. The central nodule becomes capped with a vesicle, ruptures, and discharges pus and necrotic tissue, much to the relief of the patient. Painful streaks of lymphangitis extend to the neighboring lymphatic glands. Where the skin becomes gangrenous (gangrenous phlegmon), the danger of sepsis is so great that immediate operation is the advisable procedure. Metastatic abscesses may develop.

Treatment.—The lesion should first be treated with hot, moist dressings. When the fever continues and the inflammation spreads, surgical interference is needed. The operation is not to be undertaken lightly. It is often necessary to open deeply between muscles. In some cases amputation is necessary to save life.

DISSECTION-WOUNDS AND ANIMAL POISONS.

Aside from verruca necrogenica, or anatomical tubercle, described in the chapter on Tuberculosis Cutis, lesions generally known as "dissection-wounds" occur, with symptoms of acute poisoning, upon the hands of those exposed in post-mortem examinations and dissections. At the inoculation-point, which may be either the site of a former abrasion, a rent, or the mouth of an open follicle, a painful vesicopustule, papule, tubercle, wart, furuncle, or hemorrhagic bulla rapidly rises from an angry and indurated base with hyperemic areola of dull-red shade. Suppuration, crusting or ulceration, limited to the seat of the lesion, may follow; or there may occur lymphangitis in various grades, with consequent pyemic or septicemic involvement of the system. Suppurative and non-suppurative axillary buboes are common. Gangrene and necrosis of the soft parts and the bones, especially the phalanges, may ensue, as may also a fatal result from the systemic disorders named. Rarely an acute and fatal septicemia may result when the lesion at the point of inoculation is so slight as to pass unnoticed. In a few cases chronic marasmus is induced.

Post-mortem pustules originate from infection with cadaveric poisons in the dissecting-room or dead-house. An itching macule, either at the site of an abrasion or elsewhere, soon develops, and is transformed into a vesico-pustule with a reddish halo, which bursts, and is covered with a crust, beneath which pus repeatedly forms. Occasionally, there is coincident adenopathy.

The nature of the infection varies in different cases. It is most commonly due to pyogenic bacteria, but may be caused by the specific microörganisms of tetanus, erysipelas, anthrax, or other infectious disease. The absorption of toxins resulting from the decomposition of animal tissues is undoubtedly an important factor in the infection.

Treatment.—The wound should be cleansed and opened and a moist dressing of alcohol 3 parts and water 1 part applied both to the wound and the accompanying lymphangitis.

Gayle.—Crocker,[1] under this title, describes an affection of ewes in the lambing season, who are liable to a species of puerperal disorder undoubtedly infective. Men who have skinned the animals dead of the disease have suffered by inoculation. The lesion produced at the site of the infection is a flat-chambered vesicle or bleb, slightly depressed at the center, a centimeter or more in diameter, bluish-gray in color, surrounded by a halo, and containing clear or blood-stained serum. There is apt to be axillary adenopathy, and the hand may swell. Klein has demonstrated *Staphylococcus hemorrhagicus*. In some cases there are pain and mild fever.

Treatment.—Treatment has been by sublimate lotions.

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[1] Diseases of the Skin, 3d Ed., 1903, p. 509.

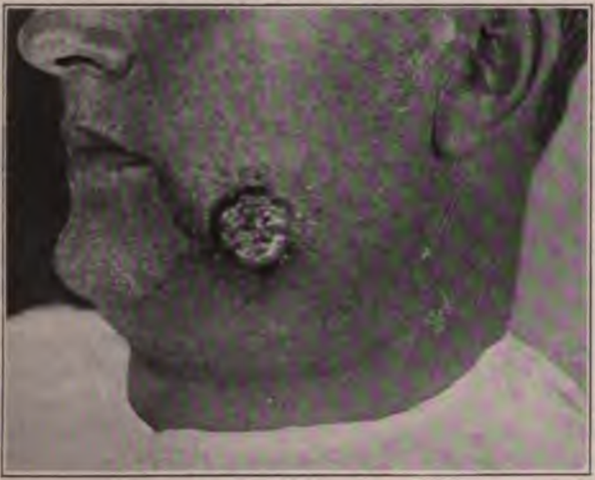
ANTHRAX.

Synonyms.—Malignant pustule, Splenic fever carbuncle. Fr., *Pustule maligne, Charbon;* Ger., *Milzbrand, Milzbrand Karbunkel.*

Definition.—Anthrax is an acute infection produced by *Bacillus anthracis,* characterized by a gangrenous carbuncle-like lesion associated with constitutional symptoms of varying degree.

Symptoms.—The lesion, usually single, most often occurs on the face and head, neck or hands; occasionally on the lower extremities and trunk. In from twelve hours to three days an inflammatory papule resembling a flea-bite develops at the site of inoculation. This is accompanied by slight burning or itching sensations. In a short time a bulla forms and an intense inflammatory infiltration follows. The bulla contains pus or blood and soon ruptures, exposing a dark eschar. This is surrounded by an area of redness in which groups of small vesicles or pustules are found. The swelling and edema may extend for some distance around the lesion, producing a markedly indurated area.

Fig. 118



Anthrax. Induced by use of an infected shaving brush.

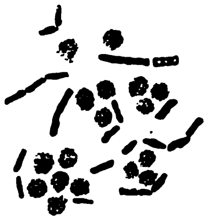
The adjacent lymphatic glands enlarge, often suppurate, and metastatic abscesses form. In some cases severe intoxication is indicated by a headache, elevation of temperature, delirium and prostration. In others constitutional symptoms are mild. In cases that recover, the gangrenous mass sloughs, leaving an ulcer which heals. In grave cases edema and gangrene increase, the constitutional symptoms become more severe and a fatal termination results from septicemia.

In some instances infection of the skin is followed by marked swelling and edema, the surface is livid and soon exhibits bullæ and gangrenous areas. This type is usually followed by a rapid systemic involvement and results fatally within a few days to one or two weeks.

Etiology and Pathology.—The disease occurs in people who come in contact with animals suffering with splenic fever or who handle the hide, hair, wool or carcass of such animals. Herders, ranchmen, tanners,[1] wool sorters, and others, are thus most commonly affected. Splenic fever occurs usually in herbivorous animals and is endemic in several foreign countries, which have been a source of supply of the infection to this country. This supply increased during the late war.[2] Shaving brushes[3] having infected hair have induced many cases recently. It is said that flies and other insects may transmit the disorder. The consumption of undercooked infected meat may cause intestinal anthrax.[4]

Pollender first described the bacillus in 1849. Davaine[5] first transmitted the disease to animals, employing infected blood from other animals. *Bacillus anthracis* is a straight rod, 4.5 to 10 microns in length and 1 to 3 microns in width. It occurs singly or in pairs in film preparations from blood. It stains readily with the ordinary aniline dyes. These bacilli when grown on artificial media form long threads and develop spores which are resistant and retain their vitality for long periods, even under unfavorable conditions.

Fig. 119



Malignant pustule bacilli and pus-corpuscles. (About × 300.)

Pathological Anatomy.—According to Unna the process is a serofibrinous inflammation which rapidly leads to necrosis of the invaded tissue. In the subpapillary vascular net were enormous numbers of bacilli and from here they extended up into the papillæ and epidermis. In the corium and hypoderm there was marked vascular dilatation and interstitial edema. The escaped lymph formed fibrinous nets which followed the course of the collagenous bundles and filled the lymphatics and veins, extending deeply, penetrating the fat cells and surrounding the entire hypoderm. Collagen bundles were swollen and split up into finer bundles and individual fibers. The elastic tissue had partly disappeared and the remaining fibers stained more faintly. Connective-tissue cells were present but showed no signs of development. A few small round mast-cells were noted. There was a marked leukocytic infiltration throughout the whole area, but they showed no signs of phagocytic action on the bacilli. The interstitial edema present in the epidermis produced the vesicles. Unna considers the fibrinous degeneration above described as characteristic of the anthrax pustule.

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[1] Brown and Simpson, Jour. Amer. Med. Assoc., 1917, lxviii, p. 608; "Human anthrax: twenty cases in three tanneries in men who handled hides from a single shipment from abroad, where anthrax was found to be endemic." Also *Cf.* Herrington, Boston Med. and Surg. Jour., 1917, clxxxii, p. 867; "Anthrax as an occupational disease."

[2] Public Health Reports, 1919, xxxiv, p. 994.

[3] Elworthy, Lancet, 1916, i, p. 20; "An outbreak of anthrax conveyed by infected shaving brushes. B. Anthracis found in the brushes." Snell and Masterman, Ibid., 1916, i, p. 242; "Fatal case of anthrax: infection by a shaving brush." Schultz, Jour. Amer. Med. Assoc., 1918, lxxi, p. 1571; "Two cases of human anthrax at Camp Jackson." Public Health Reports, 1918, xxxiii, p. 1151, "Anthrax from shaving brushes." Ibid., 1919, xxxiv, p. 994; "An investigation of the shaving-brush industry, with special reference to anthrax." Leake and Lederer, Amer. Jour. of Public Health, 1919, ix, p. 114; "Isolation of the anthrax bacillus from shaving brushes." Norton and Kolman, Jour. Amer. Med. Assoc., 1919, lxxii, p. 1129; "Anthrax in a soldier: a fatal case probably infected with shaving brush and showing intestinal carbuncles." Dennett, Ibid., 1919, lxxii, p. 270; "Anthrax at Camp Dodge."

[4] Brumbaugh, Jour. Amer. Med. Assoc., 1919, lxxii, p. 482; "Anthrax enteritis: two fatal cases with necropsy."

[5] Comptes rend. de l'Acad. des Sci., 1863, lxvii.

Diagnosis.—The characteristic features of the disorder, which are a gangrenous carbuncle-like lesion surrounded by swelling and edema, with accompanying general symptoms together with a microscopic demonstration of the bacillus in material removed from the vesicles or pustules, determine the diagnosis. The occupation of the individual is important and the history of possible employment of escharotic substances, such as phenol, arsenic or mercuric chlorid must be ascertained. Carbuncle, septic wounds, and chancre are differentiated by their course and absence of gangrene.

Treatment.—Complete excision has been employed where practicable. In many cases the following procedures have been successful. Multiple incision followed by antiseptic dressings, or direct application of phenol, injection into various portions of the affected area of tincture of iodin, or a 5 per cent. solution of phenol.

Serum Therapy.[1]—Serum therapy has been employed largely in recent years. A specific serum elaborated by Sclavo in Italy[2] is used both as a prophylactic and curative measure. In a series of 164 cases so treated, a mortality of 6.09 per cent. was recorded. Anti-anthrax and normal beef serum have both been employed with success. The former is recommended in dosage of 200 c.c. daily as long as necessary. The initial dose may be 100 c.c. and is given as soon as the diagnosis is made. Mix recommends the surgical removal of the local lesion in addition. J. C. Regan recommends local injections of 10 to 15 c.c. into the affected areas, in addition to the larger dosage for the general effect. Hyman and Leary employed normal beef serum in place of immune serum, with good results. Sodium hyposulphite and quinin are of value for systemic involvement and supportive and other measures are recommended to meet individual indications.

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[1] Mix, Med. Clinics of North America, 1918, ii, p. 587; 6 cases, 5 recovered; clinical description and discussion of treatment. Regan, J. C., and Regan, C., Amer. Jour. Med., Sc., 1919, clvii, p. 782; "Human anthrax. Employed anti-anthrax serum by deep intramuscular injection and locally into the lesion." Regan, J. C., Jour. Amer. Med. Assoc., 1919, lxxii, p. 1724; "The local use of anti-anthrax serum in the treatment of anthrax." Hyman and Leary, Boston Med. and Surg. Jour., 1918, clxxvii, p. 318; "The treatment of anthrax with normal (beef) serum. Review of pertinent literature with report of a case." These authors believe the beneficial effects of serum are due to the protein reaction rather than to immune substances in the specific anti-anthrax serum. They quote results of other workers who employed normal serum in 146 cases, with a mortality of less than 1 per cent.

[2] Quoted from Osler's Modern Medicine, iii, p. 51.

Prognosis.—The disorder is grave and frequently terminates fatally. Early recognition and prompt and efficient treatment give favorable results. The mortality in various groups of recorded cases varies from less than 1 to over 60 per cent. of cases. Formerly an average of 33 per cent. was the accepted mortality. Later it was reduced to 20 per cent., while recently the percentage has been still further decreased.

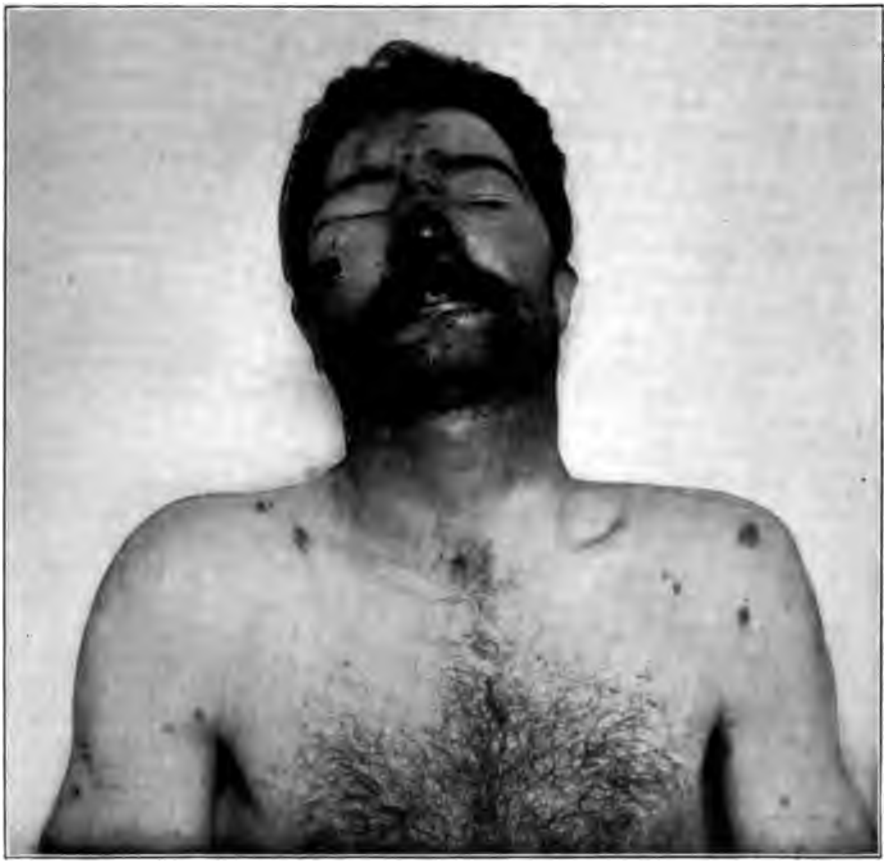
EQUINIA.

Synonyms.—Glanders, farcy, malleus. Fr., *Morve, Farcin;* Ger., *Rotzkrankheit, Maliasmus.*

Definition.—Equinia is an acute or chronic, contagious and inoculable disease transmitted to man from the horse, mule, or ass, characterized by cutaneous and mucous membrane symptoms and produced by *Bacillus mallei.*

Symptoms.—Infection occurs through an abrasion in the skin, usually on exposed surfaces, such as the hands, face or neck, or through the mucous membrane of the upper air passages, chiefly the nose and mouth. The site of inoculation is not always evident. From three days to two or three weeks after inoculation constitutional symptoms develop, indicated by malaise, headache, elevation of temperature and muscular and joint pains. When the skin is the portal of entry there occurs at the site of inoculation a reddened, tense and painful swelling which soon breaks down, forming an ulcer which rapidly enlarges. The ulcer may be the result of enlargement of the wound through which infection occurred, or may begin in the area after healing has taken place. The ulcer is irregular in outline and has a purulent, hemorrhagic and offensive discharge. The lymphatics and glands in the region become infected, are swollen and tender, and frequently break down and form abscesses. After a period of a few days to three or four weeks lesions begin appearing in different areas on the cutaneous surface. They may be few or numerous and are usually grouped. They are at first dark red spots which soon develop into yellowish papules, many of which form pustules or bullæ and terminate in ulceration. The ulcers thus formed enlarge, coalesce with others and form large, irregular areas covered with necrotic tissue. Interspersed with these are black, gangrenous patches. In addition there are deep abscesses which form in the subcutaneous tissue, break down and produce destructive ulceration. Large, brawny, infiltrated areas occur frequently with multiple openings and discharge purulent material. Nodules form along the course of the lymphatics in regions at some distance from the site of inoculation. These either undergo resolution or break down, producing irregular, discharging ulcers with indurated edges and floors. The nasal discharge is characteristic and in the early stages is thick and tenacious; later, through ulceration, it becomes purulent or hemorrhagic and offensive. In this region ulceration may extend deeply and involve the bone, producing perforation. The inflammatory process may extend and involve the mucous membrane of the upper air passages or the eyes. Involvement of the nasal mucous membrane occurs in all acute cases, and the discharge is usually profuse. In chronic cases it may not occur until late in the disease, and is absent in about one-half the cases. In the acute cases, as the infection spreads constitutional symptoms increase. The temperature remains high, chills, severe sweats and diarrhea occur, followed by rapid exhaustion and a fatal termination in three weeks or earlier. In some cases death occurs before the development of the cutaneous lesions.

FIG. 120



Cutaneous lesions in equinia. (Howard Morrow.)

In the chronic form cutaneous symptoms are similar to those occurring in the acute variety, but are less marked. The constitutional symptoms are less severe and the disease is characterized by periods of remission. The mucous membranes are less actively inflamed, although in some instances severe ulceration occurs. As stated above, about one-half of the cases escape mucous membrane involvement. The disease may continue for months or years more or less actively and terminate in recovery, or a fatal termination may result at any time through development of the acute symptoms of the disorder.

Meyer and Crohn[1] fully reported a case of acute glanders from New York City in 1907, and Bevan and Hamburger[2] reported 3 cases occurring in Milwaukee in the same year. A fatal case occurred in the Presbyterian Hospital of Chicago, in 1908, in a physician infected from laboratory cultures with which research work was being performed.

Etiology and Pathology.—Equinia is almost invariably produced by infection from horses; hence, a history of contact with these animals is one of the important points in establishing a diagnosis. It has occasionally been transmitted through clothing worn by infected individuals. The infection is produced by *Bacillus mallei,* which was carefully described by Loeffler and Schütz in 1882. It is a small rod, varying in length from 3 to 4 microns and in width from 0.5 to 0.75 microns. It is non-motile, stains readily with ordinary dyes, and is easily destained. It takes stains irregularly and is Gram negative. Cultures readily produce the disease in the lower animals, excepting cattle, hogs, rats and birds. The bacilli are abundant in papules, abscesses, nasal secretions, blood and brain tissue.[3]

Diagnosis.—The history of coming in contact with horses is important and the characteristics of the cutaneous symptoms above described, together with the constitutional symptoms, make the diagnosis plain. In some cases the clinical diagnosis should be substantiated by the experimental inoculation method described by Strauss.[4] This test is made by injecting secretion from the lesions into the peritoneum of a male guinea-pig. Within two to seven days an orchitis develops from which the bacillus may be recovered. The toxin of *Bacillus mallei* is used for diagnostic purposes in animals in the same way that tuberculin is used. Agglutination tests may also be employed, as the serum of patients suffering from glanders contains specific agglutinins.

Treatment.—The management of the disorder is largely surgical and supportive. The general condition of the patient is to be treated in accordance with the symptoms present. As a rule, treatment of any kind is of little avail.

Prognosis.—The prognosis is grave in the highest degree.

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[1] Acute Glanders. Report of a case, with review of recent literature and a complete bacteriological report. Jour. Amer. Med. Assoc., 1907, ii, pp. 1593–1595. A complete report, including reference to 9 additional cases reported to the New York Board of Health during the two preceding years.

[2] The Occurrence of Glanders in Man, Ibid., 1907, i, pp. 1595–1599. A full report, clinical and bacteriological, of 3 cases. Bibliography.

[3] Coleman and Ewing, Jour. Med. Research, 1903, ix, p. 223. Report of case with autopsy, histological and bacteriological findings, and bibliography.

[4] Archives de Méd. expérimen., 1888.

ERYSIPELAS.

Synonyms.—St. Anthony's Fire. Ger., *Rothlauf, Erysipel;* Fr., *Erysipèle, La Rose.*

Definition.—Erysipelas is a contagious and infectious disease of the skin and subcutaneous tissue, characterized by redness and swelling of the parts attacked; at times associated with vesicular and bullous lesions, and accompanied by constitutional symptoms.

Symptoms.—This disease is usually preceded by a prodromic period of malaise (lasting for twenty-four hours or less), which may be ushered in by one or several chills, followed by febrile symptoms. The latter are accompanied by anorexia and often by vomiting, with general depression and headache.

The eruptive symptoms are generally first displayed at a given point, from which the disease progresses. It is commonly first noticed in a nut- or egg-sized patch, the integument of which is tumid, slightly elevated, irregular in contour, distinctly circumscribed, without peripheral islands (these are of importance in the diagnosis of erysipeloid), and which presents a rosy or crimson-reddish color, with a peculiarly smooth, characteristic shining or glazed appearance. The sensations awakened may be those of moderate itching, of pain, heat, or burning. To the touch the affected part is tender, moderately firm, and perceptibly hotter than normal. The color fades under pressure to a yellowish white.

In typical cases the erysipelatous blush and swelling spread over an area which may be of the size of the palm, or may even cover the surface of an entire limb or large area of the body. In cases of moderate grade, the inflammation attains a maximum of extent and severity within a week, remains apparently unaltered for a day or more, and then begins to abate, with amelioration of all the concomitant symptoms. The fever, which often precedes the eruption, continues unabated during its progress, the temperature rising to 105° or 106° F., with nocturnal exacerbation, cephalic and lumbar pain, dryness of the tongue, gastric distress, and occasional delirium. As involution of the disorder is accomplished, the redness is replaced by the brownish, bluish-red, and dirty-white shades often seen after the disappearance of erythema multiforme, the epidermis finally desquamating in various degrees, according to the extent of the preceding inflammation.

In other cases, in which the exudation of serum beneath the epidermis has been rapid, the latter is raised in the form of vesicles, pustules, or bullæ, more often the latter; and, precisely as in the severe forms of dermatitis calorica, with which erysipelas presents a certain analogy, gangrene of the skin may result in the part affected. This complication is particularly liable to follow the disorder when it attacks the seat of surgical wounds and injuries.

Not infrequently new areas become involved every few days, with a corresponding increase of the constitutional symptoms; and thus the disease may travel over a large part of the cutaneous surface, with, however, only moderate-sized areas showing activity at any particular time.

Surgical accidents aside, the face is the commonest seat of the disease, on which the blush may be first seen upon one side of the nose, a cheek, a lip, or an eyelid. It often attacks the lobe of the ear after the operation of piercing the lobule for the insertion of ear-rings in women; thence it may extend over the whole face, inclusive of the mucous linings of the mouth and the nose, which present a dry, tumid, and glazed appearance, suggestive of the symptoms displayed upon the skin. The inflammation may extend to the hairy parts, but in many cases it exhibits a species of reluctance to transgress the limits there presented. It may be noticed in cases of mild grade, in which no applications have been made to arrest a local progress, that the elevated border spreads symmetrically to within a few lines of the male beard or the hairs at the edge of the forehead, and there is arrested. In severer grades these limits are exceeded, and then, as a rule, the extension is rapid and formidable. In this way the entire head may become enormously swollen, suggesting to a casual observer that it is twice its normal size. The patient then is greatly disfigured; his lips are scarlet, swollen and parted, permitting the escape of saliva; the ears, as usual when greatly enlarged, project in a marked degree from the side of the head; the eyelids are edematous and incapable of separation; the face is disfigured by bullæ or crusts; and the mind is disordered in the violence of the fever or the accesses of delirium. When recovery ensues the hairs generally fall.

All regions of the body may be invaded, such as the vaccinated arm, the leg the skin of which is involved in venous varicosities, the scrotum or the umbilicus of the infant, the genitalia of the newly-delivered woman, the breast of the nursing-mother, and every surface which has been the seat of punctured, incised, contused, or poisoned wounds, or other accidents of the integument, to which the germs of the disease may have had access.

The febrile symptoms are, throughout, persistent and characteristic of a specific toxemia. The body-temperature, as has been said, may reach 105° to 106° F., with vespertine exacerbations and remissions; it may also become subnormal. If not relieved in the course of seven or eight days, complications may be expected, namely: edema, abscess, phlegmonous inflammation, gangrene, or inflammatory accidents involving the membranes of the brain, lungs, heart, bowels, kidneys, peritoneum, or joints, together with coma and delirium. Death may result from the complications or from shock, exhaustion, or pyemia.

Erysipelas Ambulans.—This is a term used to describe that form of the affection which, after involving a given area, spreads with greater or less rapidity to the parts in the vicinity, either by direct extension and uniform advancement in one direction of the tumid and distinctly circumscribed border, or by linear, digitate, or irregular prolongations radiating from the inflammatory focus. As the blush and swelling advance in one direction, there is usually a correspondingly rapid disappearance in the other. At other times the disease, while extending to a new area and abandoning the old, is relighted in the latter; and thus an irregularly involved and irregularly extending inflamed surface presents for weeks the varying phenomena of the disease. In yet other cases, chiefly those in which there has been a history of traumatism, a linear streak or band may spread from the site of the traumatism in one direction or another, suggesting the indurated lines observed in lymphangitis.

Chronic Erysipelas.—Habitually recurrent and indolent erysipelatous attacks, the identity of which with the disease here described is difficult to establish, occur frequently. Some of these cases are due probably to repeated infection with bacteria which may be attenuated or less virulent forms of the cocci found in the severe types of erysipelas. Many cases, however, reported as "chronic or recurring erysipelas" are instances of eczema, dermatitis, or rosacea, which are subject to acute exacerbations. Instances occur in which the face, wholly or in part, is the seat of a low grade of inflammation, with local heat, swelling, redness, considerable infiltration, and some tenderness, the skin being irritable and worse after exposure to a high wind or after excesses at the table. But most of such cases fail to exhibit the distinct imprint of erysipelas. They are not only chronic in course, but are also exceedingly indolent, often lasting for years. They are unaccompanied by fever; are limited distinctly in all accesses of aggravation to the same part of the face; are characterized rarely by a bullous efflorescence; many occur in the subjects of chronic alcoholism; and the specific germs of erysipelas are not present.

Etiology.—Erysipelas is caused by the streptococci of Fehleisen, or other organisms, which gain admission to the tissues through some lesion of the surface. The site of infection may be a surgical or other wound, or it may be a slight scratch or an unrecognized abrasion of the skin or mucous membrane.

In the face, catarrhal and ulcerative processes involving the mucous membrane of the mouth, ears, and nose are often the cause of erysipelas, these processes occurring in a wide range of disorders, from syphilis of the nasal bones to caries of the teeth. Tuberculous and other ulcers, as well as eczema and several other skin-diseases, frequently furnish a means of ingress to the streptococci. Injuries of, and surgical operations upon, the scalp not conducted with antiseptic precautions, and the common piercing of the lobe of the ear in women and female children for the insertion of ear-rings, may be followed by the appearance of the disease upon the scalp, as a result of which the hair often falls. Fistules, vaccination, lesions of the tender umbilicus of the newborn infant, and railway accidents may be named as common causes of the disease in other regions.

Predisposing causes of this disease are to be sought for in cachexia, general debility, alcoholism, kidney-disease, epidemic influences, traumatism, violation of hygienic rules, idiosyncrasy, and occasionally the recurrence of previous attacks.

Jordan[1] and others have demonstrated apparently that the disease, in both mild and severe forms, may be produced by staphylococci as well as by streptococci. Jordan has shown that typical erysipelas may be produced in the rabbit by a number of different cocci.

Pathological Anatomy.—The disease is an acute inflammation of the skin and of the subcutaneous tissue. Unna, whose examinations were made largely of the skin of children and infants, found invariably a simultaneous invasion of both the cutis and the hypoderm in erysipelas, the former recovering far more rapidly than the latter, and rarely reaching such a grade of activity. The venous capillaries were all enormously distended, as if paralyzed by the poison present, and the collateral lymphatics with the lymph-spaces were equally dilated. All the cutaneous vessels swarmed with streptococci, both in the central and the marginal zones.

Diagnosis.—Erysipelas is to be distinguished from the erythemata, from dermatitis of various grades, from eczema, and from scarlatina. As a rule, its recognition is readily effected when the presence of the fever in erysipelas is kept in view, as also the peculiar shining, swollen, and rosy-reddish to damask hue of the affected parts. The redness is never produced, as in scarlatina, by multiplicity of reddish puncta, nor is it also widely diffused as in that disease. Erysipelas may at times be accompanied by a pruritic sensation, but the patch which it affects is never by any possibility scratched. By this simple test alone one may often distinguish an erysipelas of the face from an eczema of the same region in a child. From a chronic dermatitis, with thickening of the affected tissues and redness of the surface, erysipelas is to be distinguished by its tendency to spread, by its acute course, by its frequent association with bullous or vesicular lesions, and by the color, outline, and raised border of the affected patch. However, it must be understood that to these localized patches of chronic dermatitis several authors have given the name "chronic erysipelas," the difference between the views held on this point being chiefly one of titles.

Treatment.—The method of treating erysipelas by the administration of tincture of iron internally has long been popular among American practitioners, but its efficiency is questionable. This preparation is given in full doses, from 10 to 50 drops, day and night, every two to three hours, irrespective of the febrile state.

The constitutional treatment is important, but is solely symptomatic, and should be directed to lowering the temperature, to obtaining proper functional activity of all the organs of the body, and, in prolonged cases, to sustaining the strength of the patient. Locally, no matter what application is made to the surface, the affected area should be covered with gauze and bandaged. Equal parts of ichthyol, lanolin, and vaselin make a very satisfactory dressing for the average ease. It may be applied once or twice daily. Fraser[1] uses pure phenol. As soon as the skin becomes white, it is mopped with absolute alcohol. The purpose of such treatment is to limit extension of the disease. It is true that these measures will not always succeed, but it is erroneous to assert with some authors that they always fail. Certain it is that, whether effective or not in the production of the result, the advancing border of the disease will often fail to exceed the limits thus artificially described. Heppel[2] recommends the painting over the surface of a 10 per cent. solution of phenol in alcohol, as an abortive treatment, for which Braithwaite[3] substitutes a solution of tannin of the same strength.

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[1] Münch. med. Wchnschrft., 1901, p. 1371.

Good results have been reached in the local treatment of erysipelas, first, by attempting to limit the extension of the disease by the application of the tincture of iodin over the peripheral zone; and, secondly, by retaining over the entire affected surface neatly applied compresses saturated with a solution of sodium hyposulphite in the strength of about 1 drachm (4.) to the ounce (30.), or with 95 per cent. alcohol.

Attempts to limit extension of the disease by local applications of an irritating sort (corrosive sublimate, silver nitrate, phenol, tar, or turpentine) are sometimes positively injurious. Dry heat applied by the aid of cotton or wool, cold compresses, or iced lead-lotions, with intermissions of application, salicylic acid, boric acid, iodol, resorcin in solution, or iodoform in powder, may be used. A 95 per cent. alcoholic or a saturated solution of boric acid often gives good results if painted frequently over and for an inch or more beyond the affected area, or if applied on compresses.

Koch applies 1 part of creolin, 4 of iodoform, and 10 of lanolin, covered with gutta-percha. Hallopeau praises 1 part to 20 of sodium salicylate in aqueous lotions upon folds of linen. Tabit claims to abort the disease with a 10 per cent. solution of iodol in collodion. Avata and Woodyatt[4] used collodion successfully by painting a strip ½ inch in width 1 inch in advance of the border of the disease. Injections of antistreptococcic serum have been used with varying success. Guy[5] found injections of a polyvalent antistreptococcic serum of much value. He recommended the subcutaneous injection of 2 c.c. as a desensitizing dose followed in one hour by 20 c.c. injected intravenously. In severe cases 40 to 100 c.c. may be employed. Iced solutions of boric acid or magnesium sulphate were of more service than other local measures.

It is needless to add that all surgical indications are to be fully met when they are present: pus is to be evacuated, crusts removed, and drainage secured.

Prognosis.—Under favorable circumstances erysipelas, even of severe grade and extensive invasion, terminates in complete resolution. Reserve should be made, however, in every case, as a serious complication has often transformed the simplest into the gravest form of the disease. The very young, the cachectic, the victims of drink, the aged, the inmates of hospital wards depressed by other illness, and those mentally distressed by destitution and neglect, are particularly liable to suffer from grave and fatal forms of the malady.

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[1] Brit. Med. Jour., 1901.

[2] Arch. of Derm., April, 1881, p. 147.

[3] Brit. Med. Jour., April, 1881.

[4] Jour. Amer. Med. Assoc., September 14, 1918.

[5] Jour. Cut. Dis., 1919, xxxvii, p. 399; Treatment of Erysipelas.

ERYSIPELOID.

Synonyms.—Erysipelas chronicum, Progressive phlegmon, Crab cellulitis, Erythema serpens, Erythema migrans.

Definition.—This term is employed by Rosenbach[1] to designate a special inflammation of the integument occurring as a complication chiefly of traumatisms.

Symptoms.—When a wound is infected with the special poison of the disease, a peripherally spreading, tumid, and empurpled halo encircles the site of infection, which slowly disappears in the part originally attacked, while it extends progressively to another area. The advancing border of the disease is distinctly circumscribed, and may be festooned or scalloped. New points may appear, from which the violaceous redness spreads, while others are in a state of apparent inactivity. This affection may be complicated with furunculosis, but scaling is said never to occur. Itching and burning sensations are usually present. The disease is unaccompanied by general symptoms and tends to spontaneous recovery in a few weeks. The disorder affects chiefly the fingers and hand (according to Elliot, also the scratched toes) of scullions, meat-dressers, fish-dealers, poultry-cleaners, and persons of similar occupations.

Rosenbach believes that the source of this disease is a microörganism of the order *Cladothrix*, existing in putrid flesh and cheese, from pure cultures of which organism he is reported to have induced the disease. In a careful study of 323 cases, Gilchrist[2] found the disease followed a bite or other injury from a crab, the process beginning about two days subsequent to the injury. In only five cases was there any glandular involvement.

Pathology.—Gilchrist found in sections an acute inflammatory process involving the corium, and to a slight degree the subcutaneous tissue. There was a small lymphoid cellular infiltration about the bloodvessels, with numerous polynuclear leukocytes throughout the corium. The sweat-apparatus was particularly involved. The epidermis was edematous and contained some polynuclear leukocytes. Carefully performed inoculation experiments were negative, and no microörganisms were found to account for the disorder. He concluded that the disease might be due to a ferment inoculated into the skin by the crab. Adamson,[3] in two cases examined, found no microörganisms.

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[1] Verhandl. der deutsch. Gesell. f. Chir., 1887, xvi, p. 75.

[2] Jour. Cut. Dis., 1904, xxii, p. 507 (complete literature).

[3] Brit. Jour. Derm., 1908, xx, p. 367.

Diagnosis.—The disorder is to be distinguished from eczema, erysipelas, and dermatitis repens. The activity of the symptoms, the presence of vesicles, and marked itching in eczema make the distinction. From erysipelas it is differentiated by lack of constitutional symptoms, and by the presence in the former of the streptococcus. Dermatitis repens is a much more chronic disorder, and is characterized by a raised vesicular border.

Treatment.—Treatment is efficient with local application of formalin, ichthyol, resorcin, pyoktanin-blue, pyrogallol, potassium permanganate, or the mercurials in salves or in lotions. Gilchrist found a 25 per cent. salicylic-acid plaster firmly applied was curative in a few days.

SOLID EDEMA OF THE FACE.[1]

Synonym.—Recurrent eysipelatoid attacks on the face (Fox).

Definition.—This disorder was originally described by Sir Jonathan Hutchinson, in 1883. It occurs as a solid and edematous swelling upon the face, particularly the eyelids and upper lip, although the entire face may be involved. It is characterized by recurrence at varying intervals, each attack being preceded by inflammatory changes somewhat suggestive of erysipelas. Eventually, the swelling becomes permanent.

Etiology.—The disorder is believed to be due to streptococcic infection, the latter gaining entrance through some portion of the mucous membrane of the nose. In addition, many staphylococci are found, but their causative relation is not determined. Other areas through which infection may come are the eyes, mouth, and gums. In certain cases no infection atrium can be determined.

Diagnosis.—The disorder is most likely to be confused with angioneurotic edema, myxedema, or leprosy. The characteristic symptoms described above, which include the recurrent attacks and inflammatory symptoms, with temporary swellings at first, which later, after organization, become permanent, point to the nature of the disorder.

Treatment.—This has been unsatisfactory. A search for and removal of the infectious agents in the nose and other places is essential. Tonic treatment, also, is suggested, and in view of the parasitic nature of the disorder vaccine is recommended.

DERMATITIS REPENS.

Under this title Crocker first described an inflammatory disease of the skin (usually a consequence of injuries) spreading with a marginate border, and, as a rule, beginning over the upper extremities.

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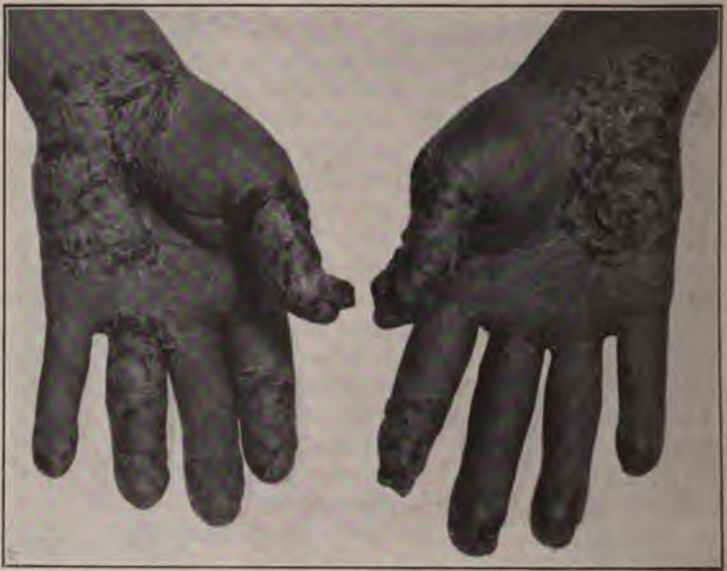
[1] Fox, Allbutt and Rolleston's System, 1911, ix, p. 183; Mackenzie, Brit. Jour. Derm., 1896, viii, p. 138 (quoted by Fox); Foster, Jour. Cut. Dis., 1909, xxvii, p. 72. Pollitzer, ibid., p. 176; Pusey, Archiv, 1912, cxi, p. 41 (report of three cases; associated lesions in the nose; staphylococcus albus and aureus found in one).

Cases have since been reported by Garden and Nepveu, Hartzell,[1] Sutton,[2] Wallhauser,[3] and others.

The inflammation spreads from a traumatism, eventually producing a raw, reddish surface denuded of epidermis and oozing at several points, the serous exudate also undermining the apparently sound cuticle. The disease spreads with uninterrupted regularity, lasting for months, and in cases invading the larger part of an upper extremity. Extension occurs by the appearance at the periphery of new vesicles or small blebs, or by the elevation of the adjacent epidermis with the fluid exudate. In either event, detachment of the epidermis leaves the characteristic denuded, red surface. There is a definite margin to the affected patch. The disease may begin with the formation of vesicles or bullæ.

Fig. 121



Dermatitis repens.

The disease has originated in cicatrices after amputation of a finger, from burns, from irritation of the feet after walking barefoot on sand, and from splinters under the nail. Crocker believes that the dermatitis results from peripheral nerve-irritation, and that there is a secondary parasitic involvement of the part. In one of Sutton's cases the disease occurred by transference of the virus from one part to another; an occurrence which, in his opinion, ruled out the peripheral nerve-irritation. The disease seems to be an infectious dermatitis, the traumatism being simply an initial factor of the process. The parchment-like epithelium often left after healing shows that the process may be one of considerable destruction of epidermal and dermal tissues, which may result in superficial atrophy and cicatrization.

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[1] Jour. Amer. Med. Assoc., 1902, ii, p. 1581 (brief summary of reported cases).

[2] Jour. Cut. Dis., 1911, xxix, p. 325.

[3] Jour. Cut. Dis., 1916, xxxiv, p. 186; Dermatitis Repens; Report of two rare cases.

Two cases of this disease were supposed to have originated in the minute traumatisms of the finger-nails occurring in farm laborers engaged in husking Indian corn by hand; and one well-marked case followed the amputation of a finger. In another the infection began near the finger nail following an injury and spread over the entire hand and later developed on the opposite hand. This proved to be a long continued and persistent case.[1] An excellent illustration of the disease is given in a colored lithograph accompanying the report of a case by Stowers.[2]

The diagnosis from eczema depends chiefly upon the recognition of the limited outline of the disease, the entire denudation of the surface, the undermined edge of the lesion, and the thinned, shining epidermis after healing. In a histological study, Sutton[3] found very slight inflammatory changes in the superficial part of the corium, the chief, and practically the only, change of importance being found in the granular layer of the epidermis. Here abscesses containing cocci were numerous. Pudor[4] also found the chief change in his case in the stratum granulosum, which he described as edema. In Sutton's comparative study of dermatitis repens and acrodermatitis perstans, he concludes that the disorders are identical.

The affection is to be treated as a parasitic dermatitis. It is very resistant to all forms of local treatment. A 5 per cent. ammoniated mercury ointment is of service, also the modified Wilkinson salve. *x*-rays may be employed. Crocker recommends a strong solution of potassium permanganate. In some instances the disorder persists for long periods even under the most careful attention.

Acrodermatitis Perstans (*Acrodermatite continue* (*Hallopeau*)).—Under this title Hallopeau, Audry,[5] Crocker, and others describe a condition very similar to that of dermatitis repens, in that it begins on, and often is limited to, the extremities; it originates frequently in a traumatism; begins often as a vesicle, spreading peripherally; and is rebellious to treatment. Hallopeau describes vesicular, bullous, and purulent types.

The disease begins frequently on a finger, to which it may be limited for weeks before it commences to spread. On rupture of the vesicles or pustules, a reddened surface is left similar to that seen in dermatitis repens. The condition differs from dermatitis repens in the frequent appearance of secondary eruptions, often pustular in form, even on portions of the body at a distance from the region first affected, and larger areas frequently are formed by the coalescence of a number of foci. The secondary eruption may be in the form of an exfoliative erythema, and may involve symmetrically considerable portions of the body. The disorder further differs from dermatitis repens in the tendency to recur frequently in the same place. It is also more persistent, and occasionally terminates fatally. The author had under observation a case of this sort for a period of one and one-half years. Constant recurrence in the primarily affected areas was noted. Temporary improvement frequently occurred, but eventually the disease resisted all treatment. From the description given by the attending physician, the patient appeared to have succumbed to a generalized exfoliative dermatitis.

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[1] See Jour. Cut. Dis., 1918, xxxvi, pp. 321-407 and 470.

[2] Brit. Jour. Derm., 1896, viii, p. 1.

[3] Loc. cit.

[4] Jour. Cut. Dis., 1913, xxxi, p. 473.

[5] Annales, 1901, s. iv, ii, p. 913 (with summary of reported cases).

This disorder is allied closely, both etiologically and pathologically, to dermatitis repens. Sutton[1] believes they are identical, and his sections show little difference histologically. Vignolo-Lutati[2] believes the disease is due to the arthritic diathesis which induces a lowered local resistance to external irritants (microörganisms).

Treatment.—The treatment is practically the same in both disorders. For the latter, Hallopeau recommends a solution of silver nitrate, 1 drachm (4.) to the ounce (30.).

DYSIDROSIS (TILBURY FOX).[3]

Synonyms.—Pompholyx, Cheiropompholyx (Hutchinson); Fr., *Dysidrose*.

Definition.—Dysidrosis is an acute or subacute inflammatory process occurring on the hands and feet, characterized by deeply situated vesicles and occasionally by bullæ, and having a tendency to recur.

The disorder was first described by Hutchinson and Tilbury Fox in 1875, since which time it has been the subject of much study. A vesicular and bullous type of ringworm closely resembling dysidrosis occurs on the hands and feet, which has undoubtedly been recorded in many instances as the disease under consideration. Dysidrosis is probably a rare affection.

Symptoms.—The early lesions of dysidrosis are deeply seated vesicles, which develop singly or in groups and are accompanied or preceded by sensations of tingling and burning. As a rule, they are found on the palms and palmar surfaces and between the fingers; also on the plantar surfaces of the feet. The lesions usually are symmetrically disposed, though one side may present more marked symptoms. The vesicles when well developed are likened to boiled sago grains embedded in the skin. Their contents early are clear but later become turbid. Bullæ varying in size from a split pea or larger develop by coalescence of groups of vesicles. These are semi-globular and project well above the surface. The lesions are deeply situated and do not rupture spontaneously, but undergo resolution by absorption of the fluid, which is followed by exfoliation of the overlying epidermis, leaving a temporary reddened tender area. The disorder may occur on both the hands and feet, or only on the former. All observers record the more frequent and more severe involvement of the hands, which in some cases may become swollen, stiff and painful. The disorder is usually mild and individual lesions run their course in one to two weeks. By recurrence it may last for weeks or months, and hyperidrosis may be associated. In long-continued cases the nails may show dystrophic changes. They were shed in two cases reported by Leviseur. In exceptional cases secondary pus infection occurs in the lesions and, rarely, an inflammatory process may extend up over the forearm or occur on other parts. This condition is said by Pringle and Adamson[1] to be due either to pyogenic complications or an associated miliaria rubra or prickly heat, conditions closely allied to dysidrosis. All of the early observers record accompanying general disturbances of mild character, exhibited as malaise, nervous depression and other symptoms. As a rule, no constitutional symptoms are present.

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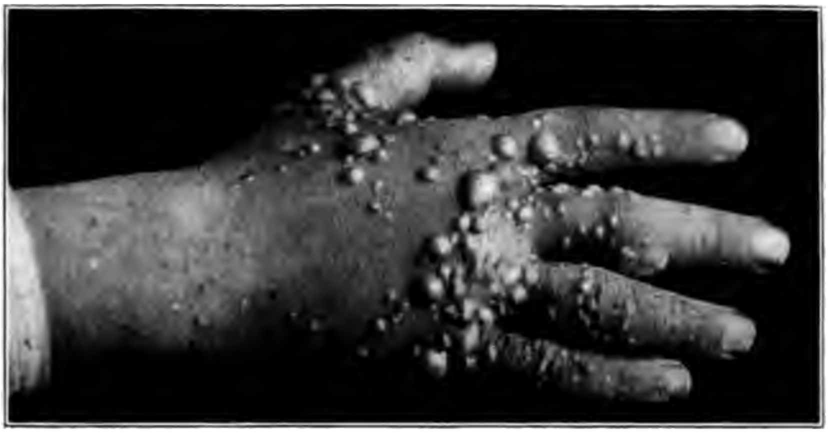
[1] Loc. cit.

[2] Giorn Ital d Mal Ven e della Pella, 1918, fasc iii, p. 153; Acrodermatitis Perstans (Hallopeau) a new contribution to the study of the arthritic dermatoses.

[3] Amer. Jour. Derm., 1873, p. 1; also Brit. Med. Jour., September 27, 1873; Hutchinson, J., Ill. Clin. Surg., 1876, i. p. 49; Robinson, Amer. Archiv. Derm., 1877, iii, p. 289; also Morrow's System, iii, p. 182 (Art. Pompholyx, by Robinson); Crocker, Trans. Path. Soc., London, 1878, x, 19; Leviseur, Contribution to Clinical Aspect and Treatment of Pompholyx (Dysidrosis, Cheiropompholyx), 14 cases, Jour. Cut. Dis., 1905, xxiii, pp. 432-439.

FIG. 122



Dysidrosis. (Howard Fox.)

Etiology.—The disorder occurs in both sexes, but more frequently in women than men. It is said to occur in persons of nervous temperament and in those who are in a depressed mental state ascribed to worry and overwork. It is considered by most observers to be a neurosis; by some to be of toxic origin, the toxin being produced in the gastro-intestinal canal.[2]

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[1] Allbutt and Rolleston's System, London, 1911, ix, p. 667.

[2] Little, Brit. Jour. Derm., 1915, xxvii, p. 324.

Pathology.—Difference of opinion has existed among those who worked on the pathology of the disease as to whether the vesicles were connected with or in the line of the sweat duct. Fox, Crocker, and others found the vesicles either directly connected with or in the line of the sweat duct. At a later date the latter found it in other situations. Hutchinson, Robinson, Williams,[1] Santi,[2] and, more recently, Sutton[3] found the vesicles in the superior portions of the rete over the papilla and not over the rete pegs which pass below to meet the ducts of the coil glands. Unna[4] believes that a microörganism resembling the tubercle bacillus is responsible for the disorder. These findings have not been confirmed by other observers. Darier[5] believes dysidrosis in most instances is due to infection with epidermophyton inguinale.

The histology as described by Robinson and others, shows changes similar to those found in some varieties of eczema. There is a mild inflammatory condition of the papillary layer of the corium evidenced by a perivascular cellular infiltration with exudation of serum which extends upward into the rete, producing vesicles in the upper part. By compression, degeneration and destruction of the cells large vesicles and bullæ form and no connection with the sweat ducts is noted.

In a more recent study Nestorowsky[6] concludes that the disease is intimately associated with the sweat apparatus, thus confirming the original view of Tilbury Fox. He asserts that the process originates in augmentation of the secretion of the sweat glands, with dilatation of the excretory canal; but that dysidrosis may occur in persons whose hands never sweat, and also that in many cases of hyperidrosis there are no symptoms of dysidrosis. While on the other hand, Sutton, in a histological study of 9 cases, found no connection between the lesions and sweat-ducts or glands, and states that his findings correspond with those of Williams, Santi, Robinson and others. He concludes that the disease is probably a neurosis, the direct exciting cause being a toxin other than local microbic in origin.

Diagnosis.—Dysidrosis is to be distinguished from eczema. The important points of distinction are the presence in the former of the deep-seated persistent vesicles, lack of itching, uniformity of lesions, absence of any marked inflammatory symptoms and serous exudation. The latter, however, does not occur in eczema of the palms, as the erythematous type is the form that is usually seen in this situation. Pemphigus is readily distinguished by the presence of bullæ in situations other than the palms and soles, and by the frequent associated symptoms. Dysidrosis is also to be distinguished from vesicular and bullous dermatitis of the hands and feet produced by epidermophyton inguinale. In the major portion of the cases examined over a long period this microörganism has been demonstrated. A microscopic examination reveals the fungus and immediately places the disorder. (Cf. section devoted to Ringworm.)

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[1] Brit. Jour. Derm., 1891, iii, p. 303

[2] Monatschefte, 1892, xx, p. 93.

[3] Jour. Amer. Med. Assoc., July 26, 1913, lxi, p. 240.

[4] Histopathology, p. 179.

[5] French Supplement to the Lancet, September 27, 1919, p. 578.

[6] Zeitschrift, 1906, xiii, pp. 183, 357, 421.

Treatment.—In cases not due to local infection the general care of the patient is important. Stimulants, such as tea, coffee and alcohol, are to be interdicted. Tonics are usually suggested and if the nervous system is involved, as is commonly described in this connection, a change of climate with proper hygienic environment is advisable. Various local applications have been employed successfully. Crocker recommended the oleate of lead or zinc. Leviseur recommended xeroform powder applied on cotton and firmly bound over each finger. Lotions, such as diluted black wash, lead water, oleated lime water, with zinc oxid or bismuth subnitrate, or the calamine or zinc oxid and liquor calcis lotion may be used. In some instances it is well to combine lotions with ointments, using the latter at night. Any of the soothing ointments suggested in the section devoted to Eczema may be used. Equal parts of Hebra's diachylon alba and the official oxid of zinc ointment, or 2 per cent. ichthyol in Lassar paste, are serviceable.

ACRODERMATITIS VESICULOSA TROPICA.

This is a rare cutaneous disorder described by Castellani and Chalmers[1] as occurring in Ceylon. These authors describe the condition as follows:

The skin of both hands, especially the fingers, appears glossy and tense, and the fingers assume a tapering shape. Millet-seed-sized or larger vesicles, deeply imbedded in the skin and having clear contents, are noted. These may remain unchanged for a long time, then slowly disappear or rupture, leaving small, superficial ulcers, which may be discrete and heal spontaneously. There is associated pain in the hands and fingers, which may be severe and continuous, or of a neuralgic, intermittent type; or there may be diffuse hyperesthesia. Itching and anesthesia have not been noted. No changes have been discovered in the nerves of the arm, and the general health of the patient is not affected. The disorder progresses for several months, occasionally for two or three years, with periods of improvement and ultimate spontaneous disappearance. The chief characteristics are the pains in the hands and fingers, the presence of deep-seated, dysidrosis-like vesicles in the glossy skin, and the long course of the disorder.

The disease is to be differentiated from dysidrosis, leprosy, dermatitis repens (Crocker), and acrodermatitis perstans (Hallopeau).

The treatment suggested is the regular application of an ichthyol ointment, 2 to 5 per cent., and the internal administration of the same drug, 3 grains three times daily.

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[1] Manual of Tropical Medicine, 2d Ed., p. 1621.

KERATOLYSIS EXFOLIATIVA.

Under the above title Wende[1] described a superficial, non-inflammatory condition of the skin which he believed to be not very rare. It is characterized by circumscribed exfoliating patches of the superficial epidermis more or less symmetrically disposed. The lesions begin as minute spots which gradually enlarge peripherally, detaching the superficial epidermis in variously-sized flakes, causing exfoliation. There is no inflammatory reaction and the disorder is unaccompanied by subjective symptoms. The disorder is recurrent, several attacks occurring during the course of a year in some instances. It apparently is more prevalent during the summer season. It occurs chiefly on the palms, less often on the soles, the dorsi of the hands and feet and, rarely, on the arms and legs.

The disorder not infrequently occurs in association with eczema, dermatitis seborrheicum, hyperidrosis, dysidrosis and ringworm of the extremities.

Lane[2] says he has long been familiar with this affection and has classed it as "desquamation estivale en aires des mains" described by Carayon.[3]

Etiology and Pathology.—The disorder occurs frequently in association with inflammatory diseases, which may be a factor in its production. It also occurs independently. Microscopically Wende found a mild inflammatory process. In the epidermis there was moderate edema, with parakeratosis. In the corium a slight cellular infiltration occurred about the vessels of the papillary layer.

Treatment.—Soothing applications are suggested and in case of persistence of the affection a mild treatment with roentgen rays may be employed.

DERMATITIS GANGRENOSA (SPHACELODERMA).

Gangrene of the skin may occur as the result of many causes, both internal and external. It may follow a dermatitis originally due to the action of either excessive heat or cold; it may be produced by the external application of chemical agents, such as caustics, strong acids and alkalies; it may be due to ergot and other substances ingested; to infectious diseases, such as lepra, tuberculosis, syphilis, erysipelas; to disorders of the bloodvessels (embolism, thrombosis, acute and chronic endarteritis obliterans, arteriosclerosis); to central nervous diseases, such as Raynaud's disease; to compression of vessels by ligatures, by tumors, by constricting scar tissue[4] or by inflammatory products; or to local infection by various microörganisms.

Multiple Gangrene of the Skin, due to local infection of the tissues having diminished power of resistance may complicate typhoid fever,[1] malaria,[2] pneumonia,[3] erythema multiforme,[4] and other diseases.[5] Independent of such disorders, infectious cases have been recorded by Hartzell,[6] Wende,[7] Waelsch[8] and others. In these cases the lesions were auto-inoculable, presenting macular and bullous lesions, followed by gangrene, ulceration and scar formation. Hartzell's case followed local infection of the hand produced by injury with a meat-hook. He found both in the bullæ and gangrenous sloughs a bacillus and *Staphylococcus aureus*. Wende's case originated from a chronic leg ulcer, and here the subsequent lesions, which were auto-inoculable, were bullous and gangrenous, and in these he found streptococci, staphylococci, diplococci, and bacilli. Sailer and Bernard and Jacob report cases of gangrene due to the diphtheria bacillus. Hallopeau[9] describes a form of gangrene in which the infection occurs on the face, especially on the forehead, as an acne-like papule, the epidermal covering of which quickly exfoliates, leaving an ulcer, which may remain superficial and disappear, leaving a scar; or may be covered with an eschar, which may persist unchanged for a long time; or the process of ulceration may extend under the eschar; or groups of new papules may develop. The same disease may affect the mucous membrane of the mouth, and the lymphatic glands may become involved.

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[1] Jour. Cut. Dis., 1919, xxxvii, p. 174.

[2] Ibid., p. 223; "Some Observations on Keratosis Exfoliativa."

[3] Annales, 1903, s. iv, v, iv, p. 283.

[4] Thompson, Med. Record, 1916, xc, p. 1103.

Single areas of gangrene have been reported in children. A local variety of gangrene has been described, under the title of *Erosive and gangrenous balanitis*, by Corbus and Harris[10] and Corbus[11] in which marked destruction of tissue may at times occur. These authors describe as the cause of the disorder a symbiosis of a vibrio and a spirochete, the latter being identical with that found in Vincent's angina.

A case of extensive gangrene involving the lower abdomen, inguinal regions, and upper and inner surfaces of the thighs was recorded by Lancashire.[12] The symptoms developed here in an area the seat of chronic eczema. The case terminated fatally. *Bacillus pyocyaneus* and *Staphylococcus aureus* were found abundantly in the skin, less so in the spleen, liver and kidney. *Bacillus pyocyaneus* was also obtained from the heart's blood.

The author has seen two cases resembling the above. Both of these had extensive gangrene involving the entire side of the abdomen, extending from near the lower margin of the ribs to the groin and from near the median line in front to beyond the posterior axillary line. In one of these, a patient aged sixty-three, the lesions began in a traumatic injury from a truss; in the other, infection followed a post-operative appendicitis wound. In each the lesions were pustular and gangrenous, characterized by rapid spread and intense destruction of tissue. Much bacteriological work was done in these cases, but no unusual organisms were found.

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[1] Stahl, Amer. Jour. Med. Sci., 1900, p. 251.
[2] Osler, Johns Hopkins Hosp. Bull., 1900, p. 41.
[3] Zuppinger, Wien. Klin. Wnschr., 1899, No. 13.
[4] Polland, Archiv, 1906, lxxviii, p. 247.
[5] Thompson, Loc. cit.
[6] Amer. Jour. Sci., July, 1898, p. 1.
[7] Jour. Cut. Dis., 1906, xxiv, p. 445.
[8] Archiv, Bd. xxxix, Heft 2, 1897, p. 173.
[9] Annales, 1895, s. viii, vi, p. 213.
[10] Jour. Amer. Med. Assoc., May 8, 1909, p. 1474.
[11] Ibid., June 7, 1913, p. 1769, with résumé of foreign cases.
[12] Brit. Jour. Derm., 1910, xxii, p. 215.

An unusual case of multiple gangrene of the hands and feet following poisoning with carbon monoxid was reported by Briggs[1] in which the early lesions were bullæ of the type seen in a second degree burn.

The clinical course of these cases is similar. They begin as bullous lesions which crust, undergo gangrene and the slough separates, leaving an ulcer which heals with scar formation, the size, configuration and extent varying greatly in different cases.

Treatment.—Surgical excision was necessary in Hartzell's and one of the author's cases. Hallopeau recommended dusting powders and ointments rather than wet dressings. Harris and Corbus found dressing with hydrogen peroxid to be specific in gangrenous balanitis. Arsphenamin applied locally is of value in this type and Sutton[2] successfully treated a severe case with subcutaneous injections of oxygen. In the major portion of cases mild, antiseptic wet dressings, such as boric acid and aluminum acetate until the slough separates, then antiseptic dusting powders and ointments until healing occurs; excision in rapidly spreading and extensive cases is at times demanded.

Prognosis.—Recovery usually takes place, even in extensive cases.

Diabetic Gangrene.—Gangrene occurs occasionally in diabetic subjects, usually of advanced age. It involves chiefly the extremities, hands, fingers, feet and toes, although it may involve the genitals and other portions of the cutaneous surface. Both moist and dry forms are seen. It may develop spontaneously, or follow a trauma, or be implanted on a previously existing dermatitis. The affected part may rapidly fall into a state of gangrene, or the latter condition may be preceded for a time by circulatory disturbances exhibited by active hyperemia, flushing, or passive congestion lividity. Neuralgic pains or paresthesia may precede. Vesicles and bullæ may form before the terminal process sets in. The process spreads peripherally and may extend and involve the deeper structures. Extensive sloughs form, at times involving one or several fingers or toes, or the entire foot. A serpiginous variety, originally described by Kaposi, may occur in advanced cases of diabetes mellitus. In these cases groups of bullæ form which dry and become covered with dark crusts surrounded by an area of inflammation. The lesions spread peripherally and after some days the crusts fall, leaving a gangrenous area beneath. This later separates and a red, granulating surface is exposed. By the peripheral enlargement and junction of these lesions large, serpiginous areas are formed. When gangrene follows a trauma the process is similar to the above. The part rapidly undergoes gangrene, or the latter process is preceded by inflammatory changes.

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[1] Jour. Amer. Med. Assoc., 1919, lxxxiii, p. 678.

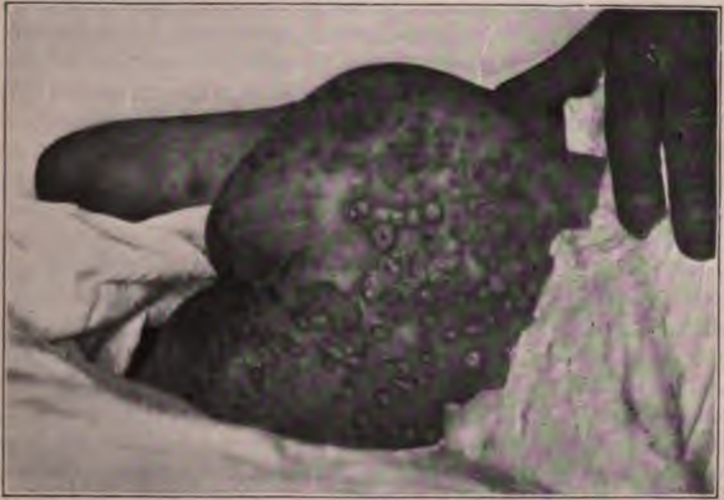
[2] Ibid., 1918, lxx, p. 675.

Etiology and Pathology.—The disorder occurs usually in well-marked cases of diabetes, in patients of advanced years. Infection of tissues of lessened resistance explains those cases following trauma and various forms of dermatitis. The spontaneous cases may be due to changes in the vessels and perhaps also to some extent to infection. Wallace[1] found atheromatous changes in the arteries in the major portion of patients suffering with diabetic gangrene.

Treatment.—General treatment of the patient and the underlying disease is of prime importance. Locally, wet dressings of boric acid or aluminum acetate may be employed until the slough separates, then antiseptic ointment or powders may be used. Curettage or amputation may be indicated in certain cases.

Prognosis.—Local areas recover. In the extensive cases sepsis may supervene, and in widespread cases the outlook is grave.

FIG. 123



Dermatitis gangrænosa infantum.

Dermatitis Gangrenosa Infantum.—**Synonyms.**—Multiple disseminated gangrene of the skin in infants, Varicella gangrenosa, Pemphigus gangrenosa, Rupia escharotica, Gangrenous infantile ecthyma. Fr., *Ecthyma térébrant.*

Definition.—A gangrenous affection of the skin in children, developing in the lesions of varicella or other of the exanthemata, or independently.

Symptoms.—The lesions of the disorder may occur during or following an attack of varicella, variola, vaccinia, rubeola, or independently. When occurring with varicella ulceration occurs beneath the varicella crusts and they present a pustular border with a red areola. The process extends peripherally and into the deeper tissues, producing a gangrenous area varying in size from a small pea to that of a silver half dollar or larger. By coalescence of several lesions large, irregular areas become involved. After progressing for a variable length of time the gangrenous areas separate and are cast off, leaving sharply defined, superficial or deep ulcers, which heal with more or less scar formation. These lesions occur chiefly about the head and upper part of the body. When unconnected with the lesions of varicella the early lesion is a vesicle or pustule, which enlarges, ruptures and becomes covered with a crust which desiccates in the center and has a pustular border and a surrounding areola of inflammation. A black slough falls, which is succeeded by ulcers, as above described. The lower portion of the trunk and buttocks are attacked more frequently in this variety. The lesions may be few or numerous, but usually only a moderate number are present. They may occur in successive crops. While the ulcers usually are superficial, at times they become deep and extensive, in which case there may be marked elevation of temperature, diarrhea, exhaustion and convulsions, and at times pulmonary complications, pyemia and tuberculosis supervene. Crocker[1] reports hemorrhagic vesicles in a grave case. In a fatal case (Fig. 123) studied by the author, vesicular and vesico-pustular lesions, which rapidly became gangrenous and sloughed, occurred over the hands, arms, lower back and buttocks, and lower limbs. *Staphylococcus aureus* was obtained here in pure culture from unruptured bullæ, and after death from the heart's blood, by Dr. Le Count.

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[1] Lancet, 1899, p. 1730.

In the cases following vaccination, lesions develop on the arm independently of the vaccination lesion, and run the course above described.

Etiology.—The disorder occurs in infants and young children from three months to several years of age, the major portion occurring in girls. While varicella and vaccinia are the most frequent antecedents, it has followed variola, rubeola, scarlatina, enteric fever, rickets, tuberculosis and congenital syphilis.

Pathology.—The lesions are induced by some microörganism or group of microörganisms usually occurring in tissues of lowered resistance. *Bacillus pyocyaneus* has been found most frequently but *Staphylococcus aureus* alone was found in our case above mentioned.

Diagnosis.—The occurrence in infants and young children of gangrenous areas, preceded by vesicles or pustules which become crusted and surrounded by an inflammatory zone, are characteristic and do not resemble other affections.

Treatment.—General tonic treatment is indicated. Quinin is recommended by Crocker. Local mild antiseptic dressings, such as boric acid, aluminum acetate, or mercuric chlorid 1 : 4000, followed after the separation of the slough by aristol, europhen, or other similar preparation in powder or ointment form, are of value.

Prognosis.—Mild cases recover. Occasionally severe and extensive cases terminate fatally.

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[1] Diseases of the Skin, 1903, p. 537.

Symmetrical Gangrene of the Extremities (*Local asphyxia, Raynaud's disease*).[1] **Definition.**—This is a paroyxsmal, vascular disorder usually affecting the extremities, characterized by local syncope, asphyxia or gangrene.

Symptoms.—The fingers, toes, ears and tip of the nose are most frequently attacked. Local syncope is the earliest and most important symptom. On exposure to cold or otherwise the affected part becomes white, bloodless, cold and appears "dead." The parts feel stiff and motility is temporarily interfered with. Moderate pain and a feeling of numbness or pricking sensations accompany this stage. This condition lasts for a few minutes or hours and is followed by asphyxia or cyanosis, or the latter may occur without preceding syncope. The part becomes moderately swollen and cyanotic. The color varies from bluish-red to deep blue or even black. The nails of the affected part are deeply colored. Painful, tingling and pricking sensations are present and some throbbing may be noted. When the circulation is restored following the stage of syncope, or, more often, that of asphyxia, the color becomes bright red and the throbbing sensations are increased. Osler states that while the sequence of syncope, asphyxia and hyperemia is the rule, exceptions occur. When the hand is affected the three processes may be simultaneously present in different fingers. The disease may present the above symptoms or part of them in recurrent attacks over a long period and may be mild or moderately severe. When the final stage occurs the circulation is not restored and gangrene supervenes. This may follow either stage, but more commonly that of asphyxia. In this stage the part remains cold, the color deepens and small areas become black and gangrenous. Gangrene may be preceded by the formation of bullæ and is commonly limited to small areas. Pain may be severe and extend far beyond the affected region. Occasionally the process is painless. Separation of the slough is slowly accomplished and the amount of destruction is slight or moderate. Occasionally a finger or toe or a part of a foot may be lost, or portions of the rim of the ear. This requires, as a rule, many weeks. The nose suffers least. The disease is symmetrical, as a rule, but exceptions occur. After repeated attacks the fingers may become pointed or clubbed and much distortion of the nails occurs.

Hemoglobinuria, epileptiform seizures, amblyopia and abdominal pains occur not infrequently as complications and occasionally scleroderma begins with Raynaud's symptoms. Galloway[2] observed temporary paralysis of both lower extremities in a case with Raynaud's phenomena.

Etiology.—The disease occurs more often in the female sex and usually in the second and third decades of life. It may occur in several members of a family. Prolonged exposure to cold, fright and sudden shock are predisposing factors. Recently many cases presenting the above symptom-complex have proved to be of syphilitic origin. As organic vascular changes are absent in true Raynaud's disease these cases may not be identical with the original conception of Raynaud.

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[1] For bibliography and more complete description of the disease see monograph by Monro, 1899, and chapter by Sée, La Practique Derm., t. i., p. 436, and Osler, Osler's Modern Medicine, vol. vi, p. 625.

[2] System of Medicine, Allbutt and Rolleston, vol. ix, p. 70.

Pathology.—The disorder is believed to be due to trophic disturbances the nature of which has not been determined. Spasm of the vessels shuts off the blood supply, producing syncope and when the circulation is entirely shut off for a sufficient length of time gangrene follows.

Diagnosis.—Raynaud's disease is distinguished from other conditions producing gangrene by the location of the lesions, the age of the patient, and the peculiar associated symptoms—syncope and asphyxia.

Treatment.—Systemic treatment is employed according to the individual indications. Protection from the cold by suitable clothing and if possible by a change of climate during the cold season. In mild cases, before gangrene occurs, electricity, either galvanic or the high frequency current, are recommended in addition to massage and hydrotherapy. For the pain analgesics may be necessary and the gangrenous parts are to be treated with mild antiseptic lotions.

Prognosis.—The disease may continue over long periods of time through successive attacks and moderate destruction of tissue sometimes follows. Satisfactory recovery follows in a certain proportion of cases. In a limited number the outcome is grave.

DIPHTHERIA OF THE SKIN.[1]

Until recently, diphtheria of the skin referred to a secondary infection of wounds with the *Bacillus diphtheriæ*, when a false membrane was produced, in which the causative organism was found. These cases at times became extensive and fatal. In recent years numerous cases have been published showing that the organism may produce acute or chronic invasions of the skin, which clinically have no resemblance to the above. Several types of lesions are described, among which may be mentioned gangrenous patches, with severe constitutional disturbance; ulcerative lesions, with or without the development of false membranes; whitlows; and vesicular and bullous eruptions resembling pemphigus and dermatitis herpetiformis. In addition, eczematous, impetiginous, ecthymatous, and pustular types, as well as tumors and abscesses, have occurred. A description of the individual types is unnecessary, as the lesions are practically identical with those produced in the diseases to which the several types correspond.

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[1] Bibliography: Brit. Jour. Derm., 1908, xx, p. 239: Condensed abstracts of articles by Labbé and Demarque—Rev. Mens. des Mal. de l'Enfance, February, 1905, xxii, p. 49: "Impetigo and Ecthyma Due to Bacillus Diphtheriæ;" A. B. Slater, Lancet, January 4, 1908, p. 15: "Diphtheria of the Skin of Three Years' Duration Treated by Antitoxin;" A. Eddowes, ibid., February 1, 1908, p. 284: "A Case of Severe Ecthyma from which Diphtheria Bacillus has been Isolated;" and A. Schucht, Archiv; Neisser's Festschrift, 1907, ii, p. 105: "Diphtheritic Dermatitis (Case 4, with the Clinical Picture of Infantile Ecthyma)." For complete bibliography and historical, clinical, and bacteriological description, *Cf.* Knowles and Frescoln, Jour. Amer. Med. Assoc., 1914, lxiii, p. 398.

A bacteriological examination is essential to the diagnosis. Knowles and Frescoln called particular attention to the differentiation between the Klebs-Loeffler bacillus and the pseudo-diphtheria bacillus. The disorder is contagious, and is contracted through inoculation of the organism from an infected patient; it usually occurs secondarily from the throat, nose, or larynx. It may, however, occur independently of such infection and be limited to the skin. The disorder may last for a long period of time or may be rapidly fatal.

Treatment.—In the ulcerative type, as previously noted in the chapter devoted to Gangrene, the lesions rapidly clear up upon administration of diphtheria antitoxin. This remedial agent should be used in all cases where the Klebs-Loeffler bacillus has been demonstrated. The local treatment will depend upon the character of the lesions present.

HERPES SIMPLEX.[1]

Synonyms.—"Fever blisters." Fr., *Herpès;* Ger., *Bläschenflechte.*

Definition.—Herpes simplex is an acute eruptive disorder, characterized by the appearance of grouped vesicles on a mildly inflammatory base, accompanied by sensations of heat and burning, and usually occurring about the face and genitalia.

Symptoms.—Herpes simplex is ordinarily a simple affection completing its evolution within one week. Any portion of the cutaneous surface may be attacked, though the face and genital region are areas of predilection. The lesions are commonly few and localized but extensive distribution occasionally occurs. It is frequently associated with infectious diseases, such as pneumonia, malaria, and cerebrospinal meningitis. It is said to be present in 40 per cent. of these cases. Sensations of heat and burning are always present, and mild constitutional disturbance is often noted in those cases involving the face and mucous membranes. It is characteristically a recurrent disorder.

Herpes facialis, Herpes febrilis, Herpes labialis, "Cold sores."—The lesions occur most commonly about the lips and nose; less frequently on other portions of the face. In the area where the lesions are to appear, there occurs a sensation of heat and burning, followed by the development of a group or cluster of vesicles situated upon a mildly inflammatory base. The vesicles are pinhead to small pea-sized, usually contain clear serum, which later may become lactescent. After accidental or spontaneous rupture there remains a superficial excoriation which may become covered with a light crust, under which healing takes place within a few days. One or several groups may be present and by coalescence different sized patches may occur. The mucous membrane of the oral cavity, pharynx, larynx, esophagus, and nasal cavity may be the site of the eruption. Here vesicles are rarely seen, their place being represented by patches of grayish epithelium or superficial erosions. Mild constitutional symptoms frequently accompany the disorder when it occurs in these situations.

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[1] Schamberg, Jour. Amer. Med. Assoc., March 2, 1907, p. 746; "The Nature of Herpes Simplex, with a Consideration of its Diagnostic and Prognostic Significance in Various Infectious Diseases;" Knowles, New York Med. Jour., August 7, 1909; "Herpes Simplex," discussion and references.

Herpes Progenitalis.—(*Herpes genitalis, Herpes preputialis*).—These titles are given the disorder when it occurs in the genital region. The lesions in men occur on the inner face of the prepuce, especially upon its upper limb, on the glans, on the balano-preputial sulcus, or in the adjacent integument. In women on the hood of the clitoris, the labia minora and the inner face of the labia majora, or adjacent surface The eruptive manifestations here are commonly preceded by itching and the sensation of heat and, rarely, by pain which is followed by the appearance of one or several pinhead-sized vesicles situated upon a tumid and hyperemic base. Within the preputial sac the lesions rupture and assume the features described in connection with those occurring upon the mucous membrane of the mouth. Considerable swelling and edema of the prepuce or the labia minora may occur. In these localities the floors of the ruptured vesicles are liable to be irritated and become infected, discharging pus or blood, or become crust covered, the crusts having a dark hue owing to the admixture of blood. In the course of a few days the crusts fall and the disease is terminated. Successive crops of vesicles may prolong the disorder for several weeks.

Generalized Herpes is noted particularly in France. A generalized herpetiform eruption occurring as a sequelæ to infectious disorders, such as diphtheria, may occasionally occur.[1] Cohen[2] reported a case of recurrent herpes in which lesions recurred over a period of two years. The lesions were situated on the lips, nose, ears, chest and wrists, and occasionally on the leg and forearm. A streptococcus was isolated and the disease terminated under vaccine therapy.

Herpetic Fever.—Savage[3] and Seaton[4] recorded two epidemics of herpes associated with marked elevation of temperature occurring in schools for boys. The disorder was characterized by sudden onset, chills, headache, elevation of temperature, nausea and vomiting, with the appearance of herpetic lesions on the skin on the third day. The lesions were chiefly on the lips and other portions of the face with occasional involvement of the extremities, throat and pharynx.

The recurrence of all varieties of herpes should be recognized and emphasized. Not infrequently the lesions reappear in the same areas over long periods of time; these areas may be conspicuous places on the face, or regions under the clothing, as on the genitalia, or over the buttocks. Several cases which recurred over a period of years have been noted on the faces of children. The same is true of those lesions occurring about the buttocks at or near the menstrual period. Adamson[1] reported herpes attacking the fingers. He further[2] noted a group of recurrent herpes of the buttocks.

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[1] Pflugbeil, Derm. Zeitschr., 1910, xvii, p. 307.

[2] Jour. Amer. Med. Assoc., 1916, lxvi, p. 1598; "Recurrent Generalized Herpes of Infectious Origin."

[3] Jour. Cut. Dis., 1883, i, p. 253.

[4] Trans. Clin. Soc., London, 1886, p. 26.

Etiology.—The disease occurs as the result of irritation or inflammation of the peripheral nerves or ganglionic centers induced by various agents. A connection between labial herpes and rigors was emphasized by Hutchinson and Symonds in patients predisposed to the disorder. It may occur as the result of local irritation, exposure to the sun's rays, drafts of cold air, or traumatism. It occurs in association with coryza and gastric and intestinal disturbances; focal infections in the mouth may also be a factor. Hartzell[3] records a case in which labial herpes invariably followed the eating of cheese. Herpes is seen in connection with acute, infectious disorders as follows:[4] Cerebrospinal meningitis, lobar penumonia and malaria (about 40 per cent.); scarlet fever (6.5 per cent.); influenza (6 per cent.); typhus (5 per cent.); diphtheria (4.01 per cent.); typhoid fever (1.33–3.5 per cent.); relapsing fever (2.5 per cent.); variola (2 cases in 3000). Herpes simplex frequently occurs in women near the menstrual period. The disorder is recurrent and the lesions may appear on the buttocks and other cutaneous locations, as well as upon the genitalia. Herpes progenitalis in some instances has the same causative factors as the other forms. It is common in prostitutes. In Hamburg, Unna observed a large number of cases in this class, and believed that 25 per cent. of them suffered with herpes. It also occurs occasionally in other women. Congestion of the parts is thought to be a cause of its development. Other causative factors are uncleanliness or irritation from decomposing secretions, local heat, friction and trauma.

Pathology.—A single pathological condition can hardly explain the diverse manifestations of herpes. Irritation or inflammation of the terminal nerves or their centers probably explains most cases. Schamberg[5] states that apart from traumatism to nerve structures most cases of herpes are of infectious origin. Many observers believe herpes to be of toxic origin. Degeneration and inflammatory changes in the ganglionic centers supplying the areas involved upon the face have been demonstrated in cases occurring with pneumonia and cerebrospinal meningitis by Howard[6] and Mallory and Wright.[7]

The pathological anatomy, according to Unna, is characterized by a true coagulation necrosis in the prickle layer of the epidermis. The vesicles had thick walls, were deeply situated, and were formed by elevation of the prickle cells from the papillary body. In the corium marked edema was present and in addition there was dilatation of the bloodvessels with perivascular cellular infiltration consisting of leukocytes, connective-tissue cells, and a few mast-cells.

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[1] Brit. Jour. Derm., 1909, xxi, p. 323; four cases in children.

[2] Ibid., 1911, xxiii, p. 322.

[3] Diseases of the Skin, 2d Edition, p. 156.

[4] Schamberg, loc. cit.; Knowles, loc. cit.; Rolleston, Brit. Jour. Derm., 1907, xix, p. 309; Idem., ibid., 1910, xxii, p. 309; Adamson, Brit. Jour. Children's Dis., 1916, xiii, p. 193.

[5] Loc. cit.

[6] Amer. Jour. Med. Sci., 1903, p. 256; and 1905, p. 1012.

[7] Mass. State Board of Health Reports, 1889.

Diagnosis.—The recognition of recurrent herpes in the genital region and about the lip is unattended with difficulty. Occasionally, when there is extensive involvement on the face the resemblance to herpes zoster is marked. The pain of the latter, the striking asymmetrical distribution and non-recurrence are important features. In herpes febrilis there is elevation of the temperature, absence of pain and the lesions may be symmetrically disposed; there will probably be a history of recurrence. Vesicular eczema and dermatitis venenata are readily differentiated. Herpes of the genital region is to be distinguished from chancroid and chancre. In chancroid the disorder is ulcerative in tendency from the beginning, auto-inoculable, and usually accompanied by coincident inflammation of the inguinal glands of the same side. The induration of the primary lesion of syphilis is absent in herpes. Balanitis with its puriform secretion and superficial patches of reddened epithelium or, in severe cases, deep destructive process, is readily distinguished from herpes progenitalis, although the two diseases may be coexistent.

Treatment.—The mild forms of herpes occurring about the lips and the genitalia require the simplest treatment. Sponging with pure water as hot as can be comfortably tolerated is of value, followed by the local application of a weak lead solution, spirits of camphor, or lotio alba, recommended by Duhring:

℞—Zinci sulphatis,				
Potassii sulphuret.,	āā	grs. xx–ʒj;	1.33–4	
Alcoholis,		ʒj;	4	
Aquæ dest.,	q. s. ad	℥j;	32	M.

Sig.—Shake and apply freely and frequently.

Alcohol or spirits of camphor applied locally will sometimes abort the lesions. Equal parts of tincture of benzoin, alcohol and glycerin is an effective combination. On the lips, after rupture of the vesicles, the abraded surface may be protected by frequent applications of compound tincture of benzoin. Crusts may be removed by the application of simple ointments, to which tincture of benzoin 1 dram (4.) to the ounce (30.) may be added. For lesions at some distance from the mucous surface dusting powders may be employed. If seen early, before rupture has occurred, the lesions may be painted with collodion. When present upon the genital regions the lesions are to be protected by the interposition of a pledget of lint, borated or salicylated dusting powder. Lotions containing tannin, phenol, or mercuric chlorid may be employed. As a rule, ointments are contra-indicated in this region. Prophylaxis by the local use of aromatic wine, or tannin and brandy, with proper sexual hygiene that will prevent congestion of the genitals, is a matter of importance. In cases in which recurrences continue it is necessary to investigate the general health of the patient, and correct whatever defect may be found. Arsenic is occasionally of value in preventing recurrences. Adamson[1] advises quinin early as an abortive agent. For immediate relief, as well as for the prevention of repeated attacks, radiotherapy offers the best method of treatment.

In several cases of recurrent herpes on the face, particularly on and about the lips in children, and in a large number of cases on the genitalia of men and women, the author has employed radiotherapy successfully.

Prognosis.—Herpes simplex is a self-limited disorder and, except for recurrences, is not serious. In the genital cases, and occasionally in those occurring on the lip, an infection atrium is presented through which syphilis may be contracted. This point should be remembered in giving prognoses to patients.

HERPES ZOSTER.

Synonyms.—Shingles, Zoster, Zona, Ignis sacer, Hemizona. Ger., *Gürtelflechte, Gürtelausschlag;* Fr., *Zona.*

Definition.—Herpes zoster is an acute disorder characterized by the occurrence of groups of vesicles situated on an inflamed base, accompanied by neuralgic pains or itching sensations, of unilateral distribution, and usually not recurrent.

Symptoms.—The eruption in herpes zoster may be preceded for a variable length of time by hyperesthesia, itching or pain. Pain may be limited to the future site of the eruption or extend beyond these limits; not infrequently the glands in the vicinity of the eruption are enlarged and painful. The lesions occur along the course of the affected nerve and are usually tense vesicles grouped upon an inflammatory and edematous base. Each group may have two or three, or many, vesicles and there may be from one to several groups or patches separated by areas of normal skin. The vesicles are grape-seed to coffee-bean in size, contain clear fluid and develop rapidly from pustules or macules. The groups or patches of vesicles may be few or numerous, and at times coalesce, forming large areas. The lesions develop in successive groups for several days, when the older ones begin to desiccate, then become covered with a yellowish-brown crust, which after a week or ten days falls, leaving temporary hyperemia or pigmentation.

The vesicles are deeply situated and have a thick roof-wall. They do not rupture spontaneously but undergo resolution by desiccation and crust formation. Occasionally the centers become purulent or hemorrhagic, in which case the crusts are yellow or dark colored. While as a rule the lesions terminate without sequels, ulceration with subsequent scar formation follows gangrene or pyogenic infection occurring in the areas.

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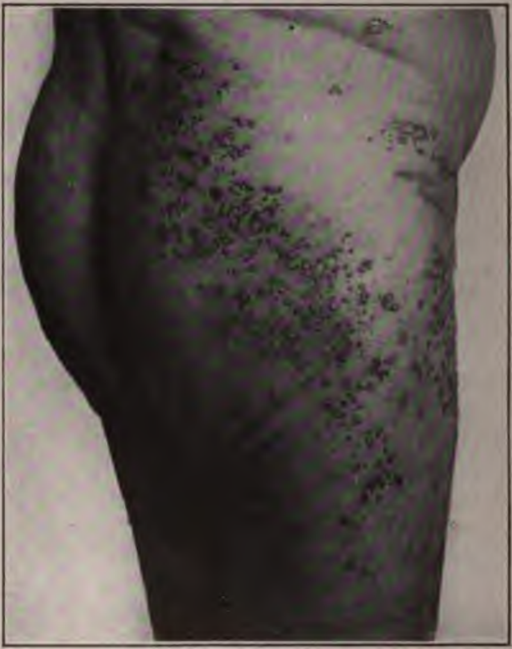
[1] Loc. cit.

The average duration of the disorder is from ten days to three we
although it may be prolonged.

The pain of zoster varies greatly in intensity and duration. It m
precede the lesions by a day or two and diminish or disappear with t
appearance of the eruption, or it may continue during the attack a
be prolonged for months or years afterward in elderly subjects. Ma
patients experience no discomfort and in children itching is apt
replace pain.

As a rule the eruption is unilateral and the disease seldom recurs

FIG. 124



Herpes zoster in Hodgkin's disease.

Variations.—The vesicles in mild cases may be limited to a
group, or they may be abortive and transitory; they may develo
bullæ, or become filled with blood (zoster hemorrhagicus, "
herpes"), or violent inflammation may occur resulting in gar
followed by the formation of eschars which leave disfiguring
(zoster gangrenosus). Recurrent zoster[1] is rare, but many cas
recorded in which an individual had two or more attacks, either
same or different regions of the body. The simultaneous occu
on two sides of the body may be symmetrical or asymmetri

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[1] For a résumé of the literature *Cf.* Grindon, Jour. Cut. Dis., 1895, xiii, p. 191
rent zoster; also see Vörner, Annales, 1906, s. 14, vii, p. 888; and Hartzell, Am
Med. Sc., April, 1890.

development. In either case it is of rare occurrence. Fox[1] reported a symmetrical case in an infant of five months.[2] Mobley[3] recorded a case of bilateral herpes zoster occurring in the region of the left facial and posterior auricular nerves, and along the course of the intercostal nerve at the right seventh interspace. MacKee and Fordyce[4] recorded a bilateral zoster occurring simultaneously over the right thigh and leg, along the course of the crural nerve, and over the third and fourth intercostal spaces on the left side. Areas widely separated on one side and supplied by different nerves are occasionally involved simultaneously. Little[5] reported simultaneous involvement of the third and eighth dorsal segments of the left side in a boy of eleven.

Occasionally in the course of herpes zoster a generalized eruption occurs[6] associated with fever, hematuria, and other signs of grave systemic infection. The author[7] recorded a case of extensive herpes zoster involving the frontal region and scalp in which on the fourth day there developed a generalized varicella-like eruption. Pernet[8] and others report similar cases. Weber[9] describes two cases with a generalized vesicular eruption and one with paralysis and quotes many illustrative cases of both types found in the literature.

Paretic and paralytic phenomena with muscular atrophy sometimes occur.[10]

The following types of zoster are generally recognized according to the regions involved:

Zoster Capillitii.—This depends upon involvement of the second branch of one of the fifth pair of nerves, and its lesions occupy the anterior and posterior portions of the scalp.

Zoster Frontalis.—Zoster frontalis occurs in the area supplied by the supraorbital nerve, which springs from the first branch of the trigeminus. Its lesions extend from the upper eyelid to the vertex, and spread in a fan-shaped figure over one-half of the brow, forehead and scalp.

Zoster Ophthalmicus.—This form may be a severe and dangerous manifestation of the disease, being often complicated by agonizing neuralgia, formidable involvement of all parts of the eye, even resulting in panophthalmia, ulcerative keratitis, pyemia, meningitis and death. Typical cases of zoster of this region may not, however, exhibit a single untoward symptom of the disease.[11]

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[1] Brit. Jour. Derm., 1898, x, p. 252.

[2] See Kraus, Centralblatt, 1905, viii, p. 226.

[3] Jour. Amer. Med. Assoc., September 14, 1912, lix.

[4] Jour. Cut. Dis., 1915, xxxiii, p. 319.

[5] Proceedings Royal Soc. of Med., London, 1914–15, viii, Derm. Sect., p. 130.

[6] Beyer, Monats., 1906, xlii, p. 415.

[7] Jour. Cut. Dis., 1911, xxix, p. 308 [8] Brit. Jour. Derm., 1914, xxvi, p. 399.

[9] International Clinics, Phila., 1916, 26, s., iii, pp. 185–202, Herpes zoster; its occasional association with a generalized eruption, and its occasional connection with muscular paralysis.

[10] Weber, loc. cit., a review of the literatue regarding paralysis following zoster, showing the many muscles that may be involved.

[11] Osterroht, "Herpes zoster ophthalmicus," Carl Marhold, Halle a. S., 1907; Monats., 1907, xliv, p. 46.

Zoster Facialis.—This depends upon involvement of the sensory nerve fibers of the trigeminus distributed to the face. Its lesions are displayed over one cheek, the side of the nose, the half of the lip or of the chin. The facial and seventh nerves may chiefly be affected.

Zoster Nuchæ, seu Collaris.—Zoster nuchæ occupies the region extending forward from the cervical vertebræ to the clavicle, or upward toward the occipital region and the auricle.

Zoster Brachialis.—Zoster brachialis occupies the region from the last cervical and first dorsal vertebræ over the supraspinous scapular region and the contiguous portions of the upper arm. Rarely, even the skin of the fingers and that over the first and second ribs are involved. It is a common and usually mild form of the disease, and is characterized by a peculiar isolation of the vesicular groups. It occurs also with lesions of exclusively brachial distribution. Thomson, of London, reports brachial zoster, with involvement of the right internal cutaneous nerve, in which two groups of vesicles appeared in the palm. Pernet[1] recently recorded zoster on the arm and hand of an infant.

Zoster Pectoralis.—Zoster pectoralis is the most frequent form of the disease, from which the common name "shingles" originated. The eruption occurs below the first dorsal vertebra and may cover the skin of the thorax as far down as the lumbar vertebræ, and extends from the spinal column behind to the sternal region in front. Two, three or more of the intercostal nerves in this region are commonly involved, and the neuralgia resulting has frequently been mistaken for the pain of pleurisy. Children more often display this form than any other variety of zoster.

Zoster Abdominalis.—The area here involved extends from the lumbar vertebræ to the median line of the abdomen. Zoster abdominalis is usually much less pronounced in its features, and the exanthem is less abundant, than in the variety of the disease just described.

Zoster Femoralis.—Zoster femoralis covers the buttocks and sacrum, and extends along the thighs, sweeping from behind forward and above downward as far as the popliteal space; in some cases involving the leg and foot. The penis, the scrotum, the labia, the vestibulum vaginæ, and perianal region may then exhibit unilaterally arranged vesicles.

Etiology.—Herpes zoster occurs in both sexes and at all ages, though it is rare in infancy. More cases are seen in the spring and autumn than in other seasons. Infections, traumatism and chemical poisons are the chief causes. It is probable that the idiopathic variety is always due to some infection. No specific microörganism has been isolated as the cause and it appears that different ones may be effective. Rosenow and Oftedal[2] produced lesions of zoster in animals by injecting streptococci isolated from tonsils and pyorrheal pockets in patients suffering with zoster. Hay[3] called special attention to the presence of adenopathy in zoster and concluded that true zoster is of infectious origin. Epidemics are not infrequently recorded. Sachs[1] reported on an epidemic in Breslau in 1901 (69 cases). It is recorded also as occurring with other epidemic disorders such as influenza and varicella.[2] Its association with or following varicella has recently been emphasized.[3] It occurs with other infections such as malaria,[4] eclampsia,[5] focal infections,[6] pyemia, septicemia, cerebrospinal meningitis and syphilis.[7] Curtin[8] reported ten cases in which zoster accompanied inflammation of serous membranes. Traumatism is a factor. Cases are recorded following gunshot wounds, spinal injections,[9] extraction of teeth, tapping of hydatids, osteopathic manipulation of the spine[10] and other injuries. It may follow the administration of arsenic and this factor is to be recognized in some of the recent cases reported occurring in syphilis. Poisoning with carbon dioxid and monoxid have also been causative factors. Recurrent cases are probably due to arsenic, trauma, or local disease in the vicinity of the affected nerve.

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[1] Brit. Jour. Children's Dis., London, 1916, xv, p. 30.

[2] Jour. Amer. Med. Assoc., 1915, p. 1968.

[3] Jour. Cut. Dis., 1898, xvi, p. 1, "Etiology of zoster; review and bibliography."

Pathology.—Pathological changes occur in the ganglia of the posterior roots of the spinal nerves. The most frequent finding is an interstitial inflammation of the ganglionic centers, less often a descending neuritis. In some cases a peripheral neuritis without involvement of the ganglia occurs. Head and Campbell[11] found inflammatory and degenerative changes in the ganglia of the posterior roots, in the posterior roots, and in the root fibers of the posterior columns of the cord, as well as in the peripheral nerves. These observations are important, as they explain the presence of the eruption in areas beyond the distribution of a single nerve which sometimes occurs. These inflammatory and degenerative changes are usually due to infection, but occasionally are produced by arsenic, trauma, or to some local irritant such as pressure from tumors or abscesses. Sunde[12] found on microscopic examination of the Gasserian ganglion on the affected side in a patient suffering with herpes zoster frontalis, active inflammation with multiple small hemorrhages and round cell infiltration in the area around the ganglion, with round-cells between some of the nerve-fibers and a sticky, fibrinopurulent exudate. In all sections gram-positive cocci, chiefly diplococci, but some also in small chains, were found. The ganglion on the opposite side was found to be normal. In most cases the products of inflammation in the nerve centers and nerves themselves are absorbed with regeneration of the degenerated area and fibers. Occasionally destruction is complete and restoration impossible and in this case the various paralyses follow.

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[1] Zeitschr. f. Heilkunde, 1906, F. 12, V. 25; Rev. Prat. d. Med. Cut. Syph. et Ven., January, 1907, Nos. 1 and 2, pp. 9 and 219 (full bibliograph to 1904); Weber, loc. cit., gives reference to epidemics recorded in the literature.

[2] Corlett, Jour. Cut. Dis., 1905, xxii, p. 289.

[3] LeFeuvre, Brit. Jour. Derm., 1917, xxix, p. 253; "A plea for the common origin of shingles and chicken-pox;" many cases quoted. Goldberg and Francis, Jour. Amer. Med. Assoc., 1918, lxx, p. 1061, Herpes zoster and chicken-pox. McEwen, Archiv of Derm. and Syph., 1920, ii, p. 205. "The Association of Herpes Zoster and Varicella," a report on zoster and varicella occurring simultaneously in a patient, with discussion of previously recorded cases.—Literature.

[4] Winfield, New York Med. Jour., 1902, lxxvi, p. 191.

[5] Abstr., Archiv, 1907, lxxxii, p. 147.

[6] Lain, Jour. Cut. Dis., 1917, xxxv, p. 486.

[7] Brown and Dujardin, Brain, London, 1919, xlii, p. 86.

[8] Amer. Jour. Med. Sci., 1902, cxxiii, p. 264.

[9] Pautrier and Simon, Annales, 1908, s. iv, ix, p. 124.

[10] Howard Fox. Jour. Cut. Dis., 1916, xxxiv, p. 773.

Brain, 1900, xxiii, p. 362.

[12] Deutsch. Med. Wchnschr., May 1, 1913, 18, p. 849.

The cutaneous changes are found both in the epidermis and corium. In the epidermis the early change is an interstitial edema which separates the epithelial cells and ruptures the protoplasmic bridges, thus producing vesicles. The epithelial cells undergo a peculiar change termed by Unna "ballooning degeneration." The vesicles are situated in the deeper portions of the rete, as a rule, although occasionally they lie between the epidermis and corium. This is more often the case in zoster of the frontal region and is followed by scars. They are unilocular, as a rule, though occasionally by coalescence of several a large multilocular vesicle is formed which may in turn by rupture of the dividing walls become unilocular, in which fragments of dividing septa may persist. Within the vesicles and about them in the edematous rete are large, round, or pear-shaped epithelial cells which have lost their prickles, and in which the protoplasm is changed into a cloudy, homogeneous mass which stains with acid dyes. They frequently contain a number of nuclei and resemble protozoa. They were once thought to be parasitic, but this was disproved by Unna, Hartzell[1] and Gilchrist.[2]

In the papillary portion of the corium there is edema and dilatation of the bloodvessels, with moderate leukocytic infiltration. The fibrous elements are moderately swollen and separated and stain poorly. In severe cases necrosis occurs which destroys the papillæ and results in scar formation.

Diagnosis.—Eczema is readily distinguished from herpes zoster by the presence of itching, the short life of the individual vesicle, its tendency to rupture spontaneously, its superficial character and the distribution of the lesions. Herpes simplex is usually recurrent, is apt to occur about the mucous outlets, often appears on both sides of the body and is unaccompanied by pain. Herpes zoster is readily distinguished from herpes iris by the peculiar concentric circles of the latter disorder and their coloring.

Treatment.—Most patients require no general attention. In severe herpes zoster and in zoster frontalis and ophthalmicus complete protection of the part can only be accomplished by rest in the hospital or at home. Exposure to the outside air sometimes results seriously.

The internal treatment of herpes zoster is, as a rule, directed toward relief of pain. In many cases this is unnecessary. If the pain is moderate, acetyl salicylic acid in 5-grain doses four or five times daily is of great value. Sodium salicylate may be used in the same manner, but is less efficient. Quinin is serviceable at times and may be administered without danger. When the pain is severe opiates are necessary and may be administered hypodermically or by mouth.

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[1] Jour. Cut. Dis., 1894, xii, p. 369.

[2] Johns Hopkins Hosp. Reports, 1896, i, p. 365.

Local Treatment.—The local treatment varies according to the stage of the disorder. If seen very early the lesions may be aborted by painting with collodion or covering with glycogelatin. If the vesicles are well formed they cannot be aborted, and protection is accomplished by dusting powders and lotions. When feasible, the lesions should be covered with cotton impregnated with a simple dusting powder, such as Anderson's, or one containing lycopodium with opium, boric acid, zinc stearate or orthoform, and held in position with a bandage if possible. When the lesions have ruptured, liniments, such as oleated lime-water with zinc oxid, belladonna, opium, or morphin, may be applied. Other helpful lotions are the lead and opium wash and calamine lotion to which one-half to one per cent. of phenol has been added. In certain instances mild antiseptic ointments are of service, particularly toward the termination of the disorder or if secondary infection has ensued. Phenol, menthol, cocain, and opium have all been used in ointment form when pain is severe. These, however, have to be employed with great care.

A continuous galvanic current of between 2 and 3 milliampères may be applied over the root of the nerve two or three times daily for ten minutes at a sitting, or the high frequency current over the nervous center responsible for the disease. Spraying with ethyl chlorid has also been of value.[1]

Prognosis.—Herpes zoster usually runs a self-limited course, terminating without sequels in a few weeks. The possible prolongation of pain in elderly people, scar formation following gangrene or infection, and destructive effects occurring about the eye are all to be remembered in forecasting the outcome.

DERMATITIS HERPETIFORMIS.[2]

Synonyms.—Herpes circinatus bullosus (E. Wilson), Herpes gestationis (Milton, Bulkley), Pemphigus circinatus (Rayer), Herpes phlyctenoides (Gilbert), Duhring's disease, Hydroa herpetiformis

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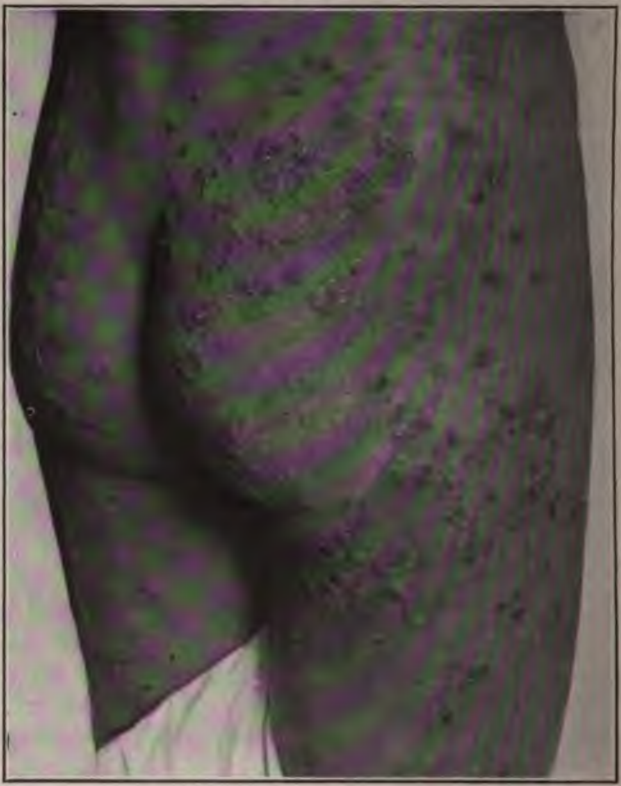
[1] A. Gregor-Penryn, Brit. Med. Jour., 1905, xli, p. 651; Morrow, Jour. Cut. Dis., 1905, xxiii, p. 157.

[2] Duhring's contributions are largely collected and published by the New Sydenham Society, London, 1893: "Selected Monographs in Dermatology." See also Bulkley, Amer. Jour. Obst., February, 1875, vi, p. 580; Fox, Amer. Archiv, of Derm., 1880, vi, p. 16; Radcliffe-Crocker, Brit. Med. Jour., May 22, 1886, p. 966; Brocq, Annales, 1888, s. ii, ix, pp. 1, 65, 133, 209, 305, 433, and 493; Tenneson-Lyon, ibid., 1888, s. ii, ix, p. 328; Stelwagon, Jour. Cut. Dis., 1890, viii, p. 50; Elliot, New York Med. Jour., May 28, 1892; Melot, Thèse de Paris, December 19, 1894; Leredde, Annales, 1895, p. 59; Darier, Annales 1896, s. iii, vii, p. 842; Leredde, ibid, s. iii, vii, p. 846; Kromayer, Zeitschrift, July, 1897, p. 475; Corlett, Trans. Amer. Derm. Assoc., May 31, 1898, p. 22; Recurrent Bullous Eruptions Limited to Certain Areas; Jamieson, Brit. Jour. Derm., 1898, x, p. 75; Dermatitis Herpetiformis; MacLeod, Brit. Jour. Derm., 1915, xxvii, pp. 201 and 249; Pemphigoid Eruptions; a thorough discussion of the subject of dermatitis herpetiformis; bibliography; opening paper of the discussion on pemphigoid eruptions at the Royal Society of Medicine. In the discussion which followed the present English views on dermatitis herpetiformis are expressed.

(Tilbury Fox). Fr., *Maladie de Duhring*, *Dermatitis polymorphes douleureuses* (*Brocq*), *Pemphigus composé* (Devergie), *Pemphigus aigu prurigineux* (Chausit), Pemphigus prurigineux (Hardy).

Definition.—Dermatitis herpetiformis is a somewhat rare cutaneous disorder, characterized by vesicles, bullæ, papules, or pustules, which frequently occur in special arrangement, followed by pigmentation. They are usually accompanied by marked itching and burning sensations.

Fig. 125



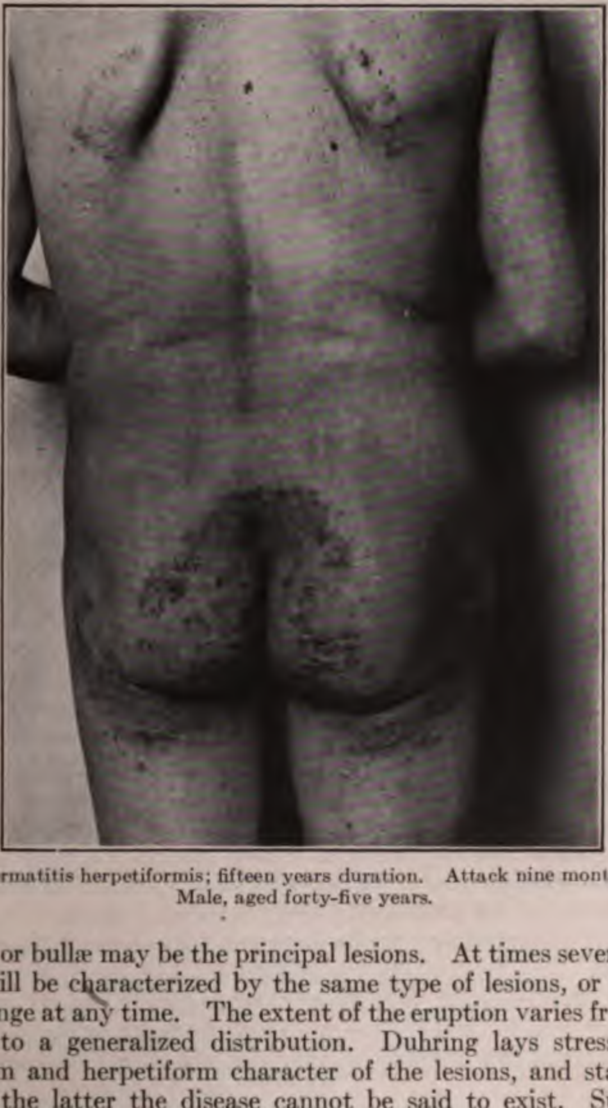
Dermatitis herpetiformis.

The disease has been described under different titles for a long period. The collection and proper placement of the various manifestations comprising this affection were made by Duhring of Philadelphia.

Symptoms.—The disease may begin with mild constitutional disturbance such as malaise, chilly sensations and moderate elevation of temperature. In severe cases these symptoms may be well marked. Similar symptoms may recur with exacerbations or recurrences. Frequently such symptoms are absent and the first indication of the disease is the eruption.

The eruption is usually symmetrically distributed and occurs in successive attacks with variable periods of partial or complete remission. The lesions of an attack may be of one type, or at least one type may predominate, and in the next recurrence they may present another type. Very frequently multiform lesions are present during the entire time, or one type of lesion may change into another. At some period in a given case vesicles may predominate, while at others erythema, pustules or bullæ may be the principal lesions. At times several recurrences will be characterized by the same type of lesions, or the type may change at any time. The extent of the eruption varies from a few patches to a generalized distribution. Duhring lays stress on the multiform and herpetiform character of the lesions, and states that without the latter the disease cannot be said to exist. Subjective symptoms of itching, burning or pricking sensations are always present

Fig. 126



Dermatitis herpetiformis; fifteen years duration. Attack nine months. Male, aged forty-five years.

and are often intense. The sensations are marked at the commencement or preceding each recurrence and are greatly relieved upon rupture of the lesions. In long-continued cases the lesions may select special areas which, according to Boeck,[1] are the extensor surfaces about the elbows, the sacral region, about the knees, and the trochanteric and scapular regions. This observation has been repeatedly confirmed by the author. During the period of years, however, through successive invasion of different regions, practically the entire surface may be involved at one time or another. The appearance of the lesions in successive crops and their grouping in fairly well-defined areas is characteristic. The general health is well preserved even in cases of long duration, though in some severe attacks through suffering and loss of rest the vitality of the patient is lowered, but is restored during the next period of remission. While the major portion of cases presents multiform lesions, vesicles predominate, but there are some cases which exhibit for certain periods in comparatively pure form one of the four primary types.

Vesicular Type.—This is the variety most frequently seen. The vesicles are pinpoint to pea-sized, flat or elevated, angular, irregular or stellate in contour. Pearly, pale yellow and translucent, firm or tensely distended, and often without areola. They may be irregularly disseminated, or occur in groups over a given area. The groups may be small, consisting of only a few discrete vesicles, or large, with closely set vesicles, which may coalesce and form bullæ or multilocular lesions. They usually develop for a period of several days, during which time itching or burning may be severe. These subjective symptoms are frequently greater than the extent of the eruption would indicate and are relieved to some extent upon rupture of the lesions. The vesicles have firm walls and tend to refill after rupture. After existing for some time they disappear by desiccation or absorption and new ones develop.

Erythematous Type.—This variety is characterized by erythematous patches of different sizes and configurations. Not infrequently it closely resembles multiform erythema. The color in the early stages is brown, red, yellowish, or brownish-red, or later variegated. Some pigmentation follows. The erythematous patches may be small coin to palm-size or larger, discrete, or coalesce to form gyrate configurations, or the erythema may be more or less diffuse. Edema in patches with urticarial characteristics may also occur. Associated with this there may be maculo-papules, or papulo-vesicles of circumscribed or diffuse form, followed by infiltrations differing in size and shape. In addition to itching and burning sensations there may be some associated general symptoms.

Bullous Type.—Bullæ are usually associated with other lesions, particulary vesicles and pustules. They are tense or flaccid, usually grouped and rather angular or irregular and of many sizes. There may be a few groups which consist of two to four or more, or the eruption may be extensive. Itching and burning sensations may be severe.

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[1] Monatshefte, 1907, Bd. xlv, p. 277.

Pustular Type.[1]—Pustules occur with vesicles and bullæ, as well as in uncomplicated form. In several successive attacks or recurrences the lesions may be pustules. They differ in size and shape; they may be small or large, acuminate, oval or flat, tense or flaccid and range from pinpoint to pea-size. The larger ones are surrounded by an inflammatory areola. They are angular or irregular in shape and occur in groups or may be disseminated. Circles or segments of circles are seen. They may be white, yellowish or, rarely, hemorrhagic. After a variable period a flat, adherent yellowish, or dark colored crust forms. An attack usually lasts for several weeks, when a period of quiescence occurs, followed later by a recurrence of similar lesions. Subjective symptoms are comparable to those in the other varieties and may be severe.

Papular Variety.—In the mild form of the disease the lesions may be chiefly papular. The areas are usually circumscribed and the lesions tend to become vesicular.

In many cases no distinct type can be recognized. All of the lesions above described may be present in various stages of evolution. There is no regularity as to appearance, evolution or recurrence. For long periods this may continue with the lesions changing their type, the older ones clearing up and new ones appearing. A variable degree of pigmentation occurs and is characteristic. The subjective symptoms increase or diminish, depending upon the activity of the process. The mucous membranes may be involved, producing erosions of varying extent, but this is of uncommon occurrence. The course of the disease is chronic, lasting for years with marked tendency to recurrence and relapse. Duhring reported two cases under observation for eleven and thirteen years respectively. The author has a patient under treatment for an acute recurrence who has been continuously under observation for nineteen years. Fairly long periods of inactivity have occurred during this time.

As complications, vegetating lesions occasionally occur and resemble those seen in pemphigus vegetans. Thickening of the skin from long-continued trauma and scar formation following secondary infection may be present. While the general health is usually well preserved, in rare instances serious disturbances of the nervous system result from long-continued distress and anxiety produced by the cutaneous manifestations.

Herpes Gestationis.—This is a variety of dermatitis herpetiformis occurring in pregnant women, and does not differ in any particular from the types of the disease seen in non-pregnant women and in men. The eruption may develop after the conclusion of pregnancy[2] but more often occurs from the third to the fourth week of gestation. In certain instances the various manifestations of the disorder have been observed repeatedly in successive pregnancies in the same woman and have occurred at no other time in this patient. The disorder may disappear before the termination of gestation or at that time, but in some cases it continues indefinitely.

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[1] Wende and Pease, A Case of Dermatitis Herpetiformis Illustrating an Unusual Variety of the Disease; Jour. Cut. Dis., 1901, xix, p. 171. Milian, Annals, July, 1919. Reviewed in Practical Medicine series 1919, vii, p. 100.

[2] Little, British Jour. Derm., 1917, xxix, p. 125; disease developed one week after delivery; other cases quoted.

In *dermatitis herpetiformis in children* the lesions are chiefly of the vesico-papular type with a generalized distribution and the grouping ordinarily seen in adults is lacking. Subjective symptoms are less marked and there is less pigmentation. Knowles[1] made a careful study of the records of 57 cases of dermatitis herpetiformis in children reported by forty-one observers recording the above facts.

Etiology.—The disease occurs in both sexes and at all ages, but less frequently in childhood. It is agreed by a large number of observers that the disease is due to a toxemia[2] of undetermined origin. It has frequently been reported in individuals of neurasthenic type, or in those in whom the nervous system has been subjected to unusual strain. Fright, anger and nervous shock are all recorded as causative, and Duhring states that the disease is due to various causes, some of which are obscure in their origin, but that the nervous system is directly responsible for the cutaneous manifestations. It has also followed infections.[3] Menstrual irregularities, pregnancy and the puerperal state are all factors in certain instances. Defective renal excretion, exposure to cold and physical fatigue have also been cited. Winfield[4] reported four cases with glycosuria as an additional symptom, indicating the neurotic origin of the disease. Cases following vaccination have been reported by Bowen,[5] Pusey[6] and others.

Pathology.—Histological examination shows an acute inflammation in the upper part of the corium, chiefly in the papillary layer. There are dilatation of bloodvessels and perivascular cell infiltration, in which are some plasma cells. Edema, with infiltration of the lymph spaces, is also noted. The vesicles are found chiefly between the basal layer of the rete and the papillary body. Larger vesicles are frequently formed by the confluence of smaller ones, and they are filled more or less with a fine or coarse network of fibrin containing polymorphonuclear with some mononuclear and eosinophile cells, red-blood and epithelial cells, also coagulated albumin. The eosinophiles are found in the vesicles and in the bloodvessels and lymph spaces of the corium, and frequently between the epithelial cells. The deeper part of the corium shows few changes. Eosinophilia is present, as a rule, but its exact significance is not established, as it is also found in other conditions. Gardiner[7] regards eosinophilia as an important means of diagnosis in the case of children. This condition was present in Bowen's[8] case, being increased at the height of the attacks; also in the cases in which a blood examination was made in the group of cases reported by Knowles. Leredde[1] would include in this category, on the basis of the pathological anatomy of the disease and the blood findings, the pemphigus vegetans of Neumann.

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[1] Jour. Cut. Dis., 1907, xxv, p. 247; with forty-eight references to literature.

[2] Hartzell, Jour. Cut. Dis., 1918, xxxvi, p. 497.

[3] Pernet, Brit. Jour. Derm., 1910, xxii, p. 1.

[4] Jour. Cut. Dis., 1893, xi, p. 447.

[5] Ibid., 1901, xix, p. 401.

[6] Ibid., 1897, xv, p. 158.

[7] Brit. Jour. Derm., 1909, xxi, p. 237.

[8] Loc. cit.

Diagnosis.—The diagnosis in classical cases is made readily. In others the distinction between dermatitis herpetiformis, impetigo herpetiformis, and certain forms of pemphigus is exceedingly difficult. It is possible that between the three there may be transitional forms scarcely to be assigned to the one category or the other. The same is true of certain exceptional varieties of erythema multiforme. It is thought by some authors that the benign form of pemphigus vegetans is merely dermatitis herpetiformis with vegetating lesions. The diagnostic features of the disease are; chronicity, with or without remissions; multiformity of the lesions, among which those of herpetic type usually predominate; the tendency of the lesions to appear in groups or patches; the very marked capriciousness and variableness of the recurrences and exacerbations in their times of appearing, and in the nature, extent and severity of the lesions; itching, often intense; and more or less pigmentation.

Treatment.—Internal treatment is employed to meet the indications presented. Proper hygienic measures should be instituted to improve the general health. Excesses of all sorts, excitement, and everything tending to interfere with the equilibrium of the nervous system should be eliminated. Regular habits of living, careful diet and sufficient out-door exercise are all of great value. Johnson[2] advises a vegetable diet. The preparation of most value in controlling the disorder is arsenic. While it is successful in all varieties of the disease, it exhibits its most marked effects on the vesicular and bullous types. The mode of administration is to begin with small dosage and increase gradually to the physiological limit, remaining at this point until the lesions have disappeared. It is frequently necessary to continue this medication over long periods and it is essential that the untoward effects, which consist of hyperpigmentations and hyperkeratoses, should be borne in mind. The preparations commonly employed are liquor potassii arsenitis (Fowler's solution) or the Asiatic pill. Crocker prefers salicin in 15-grain (1.) doses. Most English dermatologists prefer arsenic.[3] Sutton[4] advises thyroid extract in certain cases. Robinson and Whitehouse[5] have had success with antipyrin. Other coal tar preparations are sometimes used. In some instances antimony will relieve itching. Autoserum injections given as described in the chapter on general treatment have been of distinct service in many cases of dermatitis herpetiformis. After a few injections the itching disappears and the lesions are temporarily cleared up within a short time. Engman and Davis employed emetin successfully in four cases of dermatitis herpetiformis in which pyorrhea alveolaris was present.[1]

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[1] Annales, 1899, s. iii, x, p. 836.

[2] Jour. Cut. Dis., 1909, xxvii, p. 41.

[3] Brit. Jour. Derm., 1912, xxiv, p. 148.

[4] Amer. Jour. Med. Sc., cxl, No. 5.

[5] Jour. Cut. Dis., 1909, xxvii, p. 41.

Local treatment is directed toward relieving the itching and overcoming secondary infection. In some instances stimulating preparations are of service and in others sedatives are necessary. Stimulating lotions may contain tar, phenol (1 to 2 per cent.), ichthyol (2 to 10 per cent.) or thymol, 1 to 5 grains, (0.06–0.33) to the ounce (30.). Stelwagon recommends liquor carbonis detergens in a strength varying from 1 to 10 parts of water up to the pure solution. Duhring recommends a strong sulphur ointment in the cases presenting vesicles, pustules and bullous lesions, and advises that the ointment be applied with friction. An ointment combining sulphur and oil of cade has been of distinct service in numerous cases treated by the author. The formula follows:

℞—Acidi salicylici,		grs. iij;		20
Mentholis,		grs. ij;		16
Sulphur. ppt.,		ʒss;	2	
Zinci oxidi,		℥j;	4	
Olei Cadini (Muth & Co.),		ʒj;	4	
Amyli pulvis,		ʒij;	8	
Ungt. petrolati,	q. s. ad	℥j;	30	
M. et ft. ungt.				

In many cases soothing treatment by means of alkaline, bran or other demulcent baths, followed by some of the soothing lotions advised for use in acute stages of eczema, is required. In chronic cases, where lesions constantly recur over a given area, radiotherapy is of distinct value.

Prognosis.—The prognosis is always doubtful, and in each case may be grave. Persistence for years, with periods of aggravation and decline is the rule. Temporary recovery from repeated outbreaks is common, and complete recovery under proper treatment occurs in a certain proportion of cases.

IMPETIGO HERPETIFORMIS.

Definition.—Impetigo herpetiformis is an inflammatory affection of the skin characterized by the occurrence of pustules in groups, accompanied by grave constitutional symptoms, usually terminating fatally.

The disease was originally described in 1872 by Hebra[2] from observations made in 5 cases, 4 of which terminated fatally. Some years later Kaposi[3] recorded additional observations. A few other cases have since been reported in various countries, including America. Competent observers, such as Duhring, Crocker and Dubreuilh in analyzing the reported cases, believe some of them do not conform to the type originally described by Hebra.

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[1] Jour. Amer. Med. Assoc., 1916, 66, p. 492; "Dermatitis herpetiformis." A report of some cases treated with emetin.

[2] Wien. klin. Wochenschrift, 1872, p. 48.

[3] Vierteljahr, 1887, xiv, p. 275.

Symptoms.—The eruptive manifestations begin, as a rule, in the groin, on the inner surface of the thighs, about the umbilicus, over the breasts and in the axillæ. From these regions it spreads over other portions of the cutaneous surface. The early lesions are pinhead-sized, rarely larger, pustules grouped upon an inflammatory base. The pustules are opaque or greenish-yellow and soon become covered with a dirty, brownish crust, beneath which the epidermis is reformed, or the surface remains red, infiltrated, and exudes serum. The process extends by the development of new pustules in a circinate manner about the original patch. Several rings may thus be formed. By coalescence of these peripherally spreading patches from different foci large portions or the entire cutaneous surface may become affected. After some months, when the eruption has become extensive, the skin is described as being swollen, covered with crusts, and presents excoriated and fissured areas with irregularly disseminated circles of pustules. The mucous membranes of the mouth and throat and occasionally the esophagus participate in the process. Well-defined grayish patches with depressed centers, together with erosions, the site of previous pustules, occur.

Constitutional symptoms are always present and as the disease progresses become grave. Fever may be continuous or remittent. Chills and high elevations of temperature mark each new extension of the process. Vomiting, diarrhea and delirium are associated symptoms.

Etiology.—The disease occurs usually during pregnancy or the puerperal period, but cases are recorded of its occurrence in non-pregnant women by Dumesnil and Marks,[1] Hartzell,[2] and others. Instances of its occurrence in male subjects are recorded by Kaposi, Dubreuilh,[3] Whitehouse,[4] Chambers,[5] and others.

The relation of the disorder to pemphigus and dermatitis herpetiformis is shown in cases recorded by Heitzmann,[6] Zeisler[7] and Fordyce.[8] Many observers believe the disorder is the result of an intoxication. Wechselmann,[9] in the absence of positive findings in a typical case, discussed the possibilities of the toxins of pregnancy being etiologically concerned. De Amicis[10] concluded after a carefully conducted bacteriologic examination of the cutaneous lesions and blood that the disease was probably toxic, rather than an infection. While bacteriological examination of the pustules and other cutaneous lesions, as well as the blood, have, as a rule, been entirely negative there are some who believe the disease to be an infection. Kren[1] recorded 2 cases following local infection of the finger in one instance and of the toes in the second. Fordyce regards the disease as the cutaneous expression of a variety of conditions.

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[1] Archiv, 1889, v, p. 567.

[2] Jour. Cut. Dis., 1897, xv, pp. 390 and 506.

[3] Annales, 1892, tome iii, p. 50; Report of a Case and an Analysis of Previously Recorded Cases.

[4] Jour. Cut. Dis., 1898, xv, p. 169, and Twentieth Century Practice of Medicine, 1896, v, p. 425; with résumé of literature.

[5] Brit. Jour. Derm., 1911, xxiii, p. 65.

[6] Archiv, January, 1878, p. 37.

[7] Monatshefte, 1887, vi, p. 950.

[8] Jour. Cut. Dis., 1897, xv, pp. 390 and 495.

[9] Archiv, 1910, cii, p. 207.

[10] Giorn. Ital., 1912, liii, No. 5, p. 711; abstr. Archiv, 1913, cxv, p. 791; with résumé of the literature.

The histology has been examined by Dumesnil and Marks, Dubreuilh and others. They found dilatation of the blood- and lymph vessels, with surrounding embryonic cellular infiltration. These changes were most marked in the superficial portion of the corium at the base of the pustules. Cocci were present in the pustules, the latter being always within the epidermis. The interpapillary prolongations were increased in length (acanthosis). In the papillary layer below the pustules the infiltration was so active that the structure of the corium could hardly be distinguished. The cells extended into the rete, obliterating the line of demarcation between the corium and epidermis. The pustules were found at different levels in the rete mucosum.

At autopsy some cases have shown nephritis; others purulent endometritis and septicemia, tuberculous lesions of the lungs and intestines, while in others no cause of death was found (Duhring).

Diagnosis.—The extreme rarity of the disorder is a distinguishing feature. Its usual occurrence during pregnancy, the presence of small pustules grouped on inflammatory bases and having a circinate arrangement, together with the general symptoms, are features not seen in other diseases. Dermatitis herpetiformis of the pustular type very closely simulates this disorder in rare instances. Pemphigus vegetans also is to be differentiated. In the latter disorder the lesions may begin in the same portions of the body, and the distinction will sometimes depend upon the interpretation of the limits of the disease under consideration. If limited to the cases as originally described by Hebra, there will be found no difficulty in making the distinction.

Treatment.—The treatment is conducted on general principles, which includes the administration of antipyretics and the local employment of alkaline or demulcent baths. In certain instances a continuous water bath is of value, and the water bed is also of benefit. Supportive and general treatment should be used according to the indications. The treatment is practically that of pemphigus. (Cf. this section.)

Prognosis.—The prognosis is necessarily grave, the disease terminating fatally in 50 per cent. of the cases.

PEMPHIGUS.

Synonyms.—Pompholyx. Ger., *Blasenausschlag*.

Definition.—Pemphigus is an acute or chronic disease of the skin occurring in several phases and often accompanied by constitutional symptoms. It is characterized by bullæ which develop in cycles or in continuous succession. The term pemphigus was formerly applied to every disease of the skin presenting bullæ. As knowledge has increased, the group has been greatly reduced. The problem is not yet entirely settled, as certain types of multiform erythema and dermatitis herpetiformis are difficult to distinguish from pemphigus. In the following description pemphigus will be treated as occurring in four forms: *pemphigus acutus*, *pemphigus vulgaris*, *pemphigus foliaceus*, and *pemphigus vegetans*.

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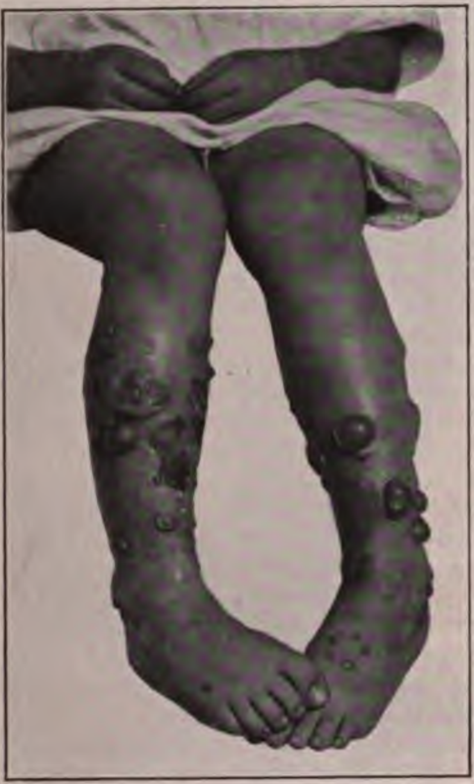
[1] Monatshefte, 1907, xlv, p. 297.

Fig. 127



Pemphigus.

Pemphigus Acutus (*Acute Febrile Grave Pemphigus* (Fox), *Acute Infectious Bullous Dermatitis*).—Under this title the disease will be described as it occurs following septic wounds and vaccination. The former conforms to the type described by Pernet and Bullock[1] and later by Brocq[2] under the title *Pemphigus aigu, grave, febrile à forme Infectieuse*. In America cases have been reported by Bowen,[3] Grindon[4] and Pusey.[5] The latter conforms to the type described by Howe.[6] In this rare form of the disorder the course of the disease is relatively rapid, a few days or weeks being sufficient for the full development and termination of the disorder either in death or recovery.

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[1] Brit. Jour. Derm., 1896, viii, p. 157.

[2] La Pratique Derm., 1902, p. 761.

[3] Jour. Cut. Dis., 1904, xxii, p. 253.

[4] Ibid., 1903, xxvii, p. 439.

[5] The Principles and Practice of Dermatology, 3d Ed., 1917, p. 288. Acute type in a butcher terminated in recovery.

[6] Ibid., 1903, xxi, p. 254.

Symptoms.—There is a history of an injury or previous vaccination in cases of this type. The eruption is usually accompanied by general symptoms, indicated by chills, elevation of temperature, and other signs of constitutional involvement. The lesions may begin in the mouth or about the neck, trunk or other parts of the body. The eruption, which consists of bullæ varying in size from a split pea to that of an egg, spreads rapidly and may cover large areas of the body surface. The bullæ are oval or round, tense or lax, and contain serum which is sterile at first and later becomes purulent or hemorrhagic. The eruption may develop in successive cycles or as a rapid explosion. In fatal cases coalescence of the bullæ occurs, their contents become purulent and hemorrhagic and large areas of the skin are denuded of the outer layers of the epidermis. The group of cases collected by Pernet and Bullock form the type which is representative of acute pemphigus. In many of these cases the disease followed an infected wound, usually on the hand, and occurred most commonly in butchers after bites from animals. The eruption spread rapidly and soon became generalized. A common symptom was extreme fetor present in most cases. Sometimes an areola was present, but this was not constant. Hemorrhagic lesions occurred. The mouth, palate, eyes and nostrils were practically always affected. The constitutional symptoms were usually severe, the temperature ranging as high as 104.9° F. In some cases albuminuria was noted. A varying period of time intervened between the initial injury and the development of the disorder. In this group Bowen places the cases reported by Howe of bullous dermatitis following vaccination. In Howe's cases 9 of the 10 patients had recently been vaccinated. Six died and the average duration of the disease was six weeks from the beginning of the cutaneous lesions until death or recovery took place. The disorder began about five weeks after vaccination. The lesions occurred chiefly on the back of the neck, the region between the shoulders, buttocks and inner aspect of the thighs, and there was a tendency toward grouping. In most of the cases the mucous membranes of the mouth and throat were markedly affected, bullæ and ulcers being present in these sites. Corresponding lesions were also found in the trachea.

In a group recently reported by Mook[1] the lesions occurred about the mouth, neck and extremities, especially over the joints. The bullæ were described as being clear and tense and developing on the normal skin. They were at times arranged in circinate and concentric rings and occasionally became hemorrhagic.

Etiology. From the above description it may be noted that occupation is important with reference to etiology in certain cases. Most of the patients of one group were either butchers or persons who handled

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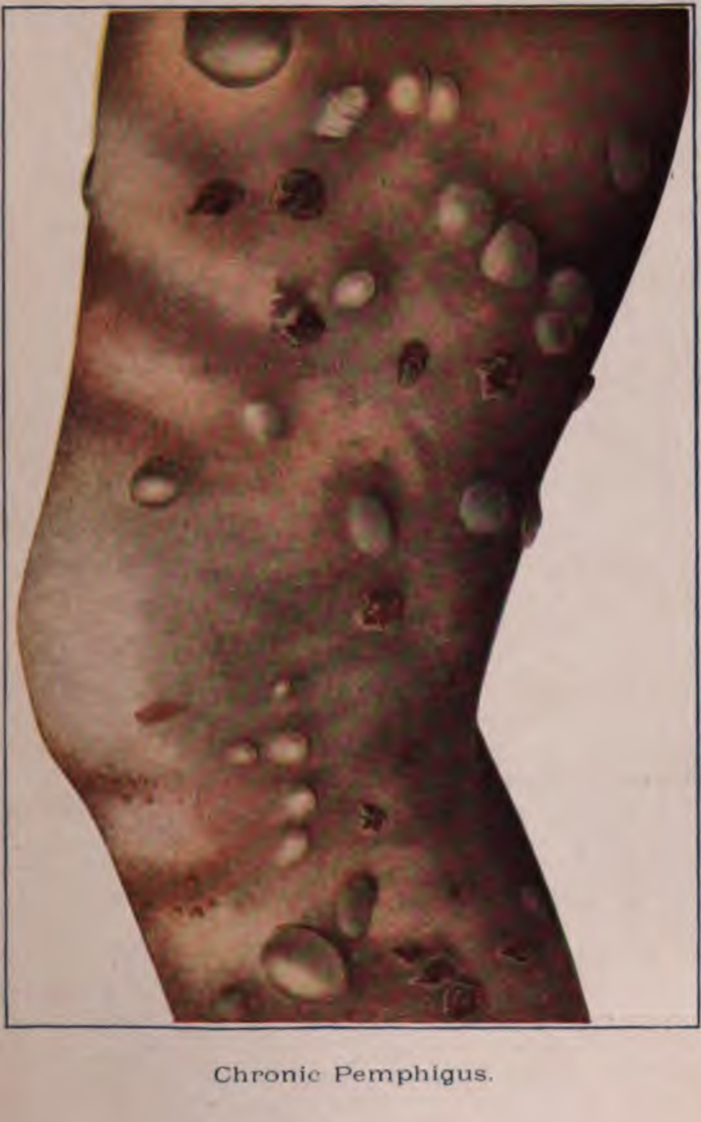
[1] Jour. Cut. Dis., 1915, xxxiii, p. 667.



Chronic Pemphigus.

dead animals, and many of the patients, previous to the appearance of the disease had a wound on the hand where the infection had taken place. In Bowen's case the patient was a meat-cutter, and a septic wound on the hand was the beginning of the disorder. Relative to the group of cases reported by Howe, Bowen discusses the possible connection, through vaccination, of an acute pemphigus of this sort and hoof-and-mouth disease of cattle, the epidemic reported by Howe having occurred immediately preceding such an epidemic. Mook[1] traces no connection between hoof-and-mouth disease and the disorder as it occurred in the group he studied. That the cases are of bacterial origin there seems to be little doubt. The definite organism has not been isolated. In the Pernet and Bullock group a special diplococcus was isolated. This organism proved pathogenic and resembled that described by Demme. Other organisms have occasionally been found, but as a rule the attempt at culture fails.

Pathology.—Acute pemphigus is induced by a toxemia, probably of bacterial origin. Hartzell[2] says: "The toxic character of the post-vaccinal eruptions and those which occur as a sequel of septic wounds is generally admitted." The histological picture shows an inflammatory process involving the superficial layers of the skin. In the Pernet and Bullock group there was well-marked cellular infiltration in the corium, extending rather deeply, and presenting the appearance of a chronic inflammatory process. There was some vascular dilatation, with perivascular cellular infiltration, the latter involving to a certain extent the sweat glands. In the epidermis the bullæ were found between the stratum corneum and the rete. The bullæ contained a fibrinous network, throughout which were entangled cells of the rete. In addition, they contained small, round cells, with large deeply-stained nuclei, a few red blood corpuscles, and some micrococci.

Treatment.—The treatment resolves itself into two factors, the first prophylactic, which is the more important. Immediate attention to wounds suffered by butchers and other people working with animals should be urged and thoroughly carried out. When the disease is well started, the general treatment suggested by the patient's condition should be instituted. Quinin in large doses; vaccine if an organism can be isolated; and supportive treatment are indicated. Locally the continuous bath would be of value, and soothing and emolient preparations should be employed for the comfort of the patient. Close attention should be paid to cleansing of the skin in order to prevent absorption from secondary infection. Pernet[3] reported the successful management of a case of acute septic pemphigus, developing upon dermatitis herpetiformis, with opium and liquor arsenicalis.

Prognosis.—From the above it will be seen that the prognosis is very grave.

Pemphigus Vulgaris.—The term pemphigus vulgaris is applied to the common form of the disease and it has been employed generically by many authors to include all varieties of the disorder. Mixed types do occur and one type not infrequently develops on another. In the present state of our knowledge it seems best to describe separate types.

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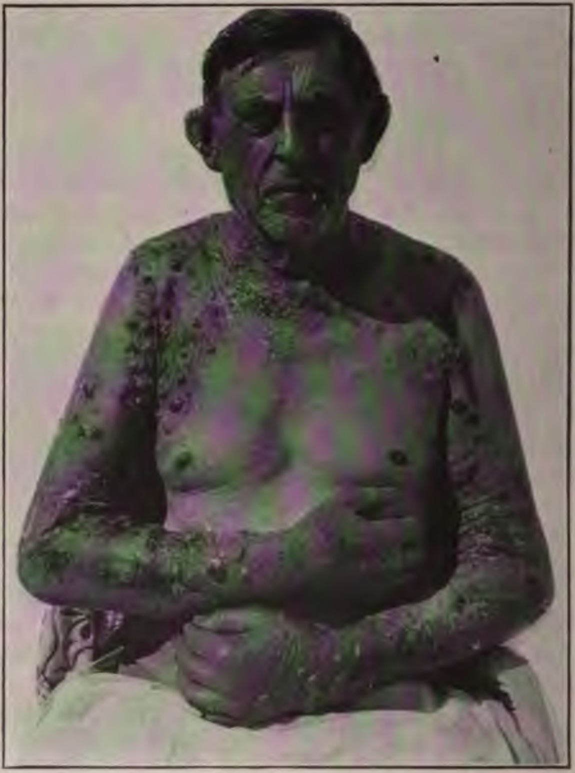
[1] Loc. cit.

[2] Jour. Cut. Dis., 1912, xxx, p. 119.

[3] Brit. Jour. Derm., 1910, xxii, p. 1.

Symptoms.—The lesions of pemphigus are bullæ of various types and their sequels. The lesions may be located on any part of the body. The mucous membranes, face, neck, extremities and trunk are all invaded to a greater or less degree. The bullæ occur in successive groups or continuously. They may be widespread or comparatively limited. There may be periods of intermission or remission. In the common forms the bullæ are usually tense and contain clear serum. They are oval or rounded and vary in size from that of a split pea to the palm or larger. In certain instances they may be elongated, and they not infrequently are flaccid instead of tense. Both types of lesions may be present simultaneously. The contents may be serous, seropurulent or hemorrhagic. They may be irregularly distributed, or a certain tendency to grouping may be noted. A circinate arrangement of the bullæ is not uncommon in typical pemphigus. The bullæ

FIG. 128



Pemphigus with rapid and fatal termination.

may disappear by rupture and the formation of crusts, or they may undergo involution without rupture. Crusting is followed by pigmentation. The crusts may be comparatively light or heavy and their color depends upon the contents of the bullæ. In some instances the superficial epidermis is removed and various-sized areas of denuded surface are left. This frequently occurs in serious cases where there is apparently no attempt on the part of the lesion to heal. In the favorable cases a single bullous lesion may run its course in a week or two, leaving a newly formed, delicate epidermis which is violet, purplish or bluish-red, and which later becomes pigmented. By successive crops the disorder is continued over weeks, months, or years. The mucous membranes of the mouth and vagina may become invaded, and not infrequently the first manifestations of the disease are presented in the former situation. The mucous membrane of the mouth becomes denuded of epithelium in varying sized areas, the bullæ in this location rupturing early. In consequence of the tenderness thus produced the vitality of the patient suffers soon on account of inability to take proper nourishment. The course of the disease may be rapid, two or three months being sufficient to result in a fatal termination. The cases of grave portent are those in which the first manifestations occur in the mucous membrane of the mouth, where because of the trauma, the roof of the bullæ becomes removed, leaving a raw, denuded surface which shows little tendency to heal. The term *Pemphigus malignus* has been used in the past to designate these cases. At other times the course of the disease is slow, the number of lesions varying from a few to a great many and even when extensive areas are involved the general health may remain good over a long period. Some cases have a complete remission for some time, though recurrence is the rule.

Constitutional symptoms vary. Some cases are ushered in with symptoms of this type. Others have mild or severe constitutional symptoms at each recurrence, whereas in many cases the constitutional symptoms are slight or entirely absent over a long period.

A mild form of pemphigus has been described under the term *Pemphigus benignus*. The eruptive manifestations in any pemphigus may be of this type for a time, but it is doubtful if the major portion of the cases so described are pemphigus. *Pemphigus solitarius* has also been described. Such cases are probably local infections and should not be considered as a type of pemphigus. The terms *Pemphigus disseminatus*, *Pemphigus circinatus*, and *Pemphigus hemorrhagica* are only descriptive of certain phases of the disease.

That certain examples of pemphigus belonging to this group are acute in nature seems to be a fact. A number of cases have been under observation during the past few years. One of these was reported conjointly by the author with Dr. Peter Bassoe.[1] In this case the lesions originated in the mouth and the disease proved fatal in two months. Extensive cutaneous involvement occurred with the formation of bullæ and severe and extensive erosions of the skin. There was practically no attempt at healing in any of the denuded areas. This history could be repeated many times by patients seen during the last few years.

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[1] Jour. Cut. Dis., 1905, xxiii, p. 294.

Etiology.—The theories formerly held as to the origin of the disease have been the neuropathic, toxic and infectious. In Europe males apparently are affected more commonly than females, but the reverse seems to be true in this country. It has been observed not infrequently in debilitated patients who are suffering from neurasthenia, worry and exhaustion, visceral disorders and impairment of nutrition.[1] The neuropathic origin is held by many but the proof in most cases is lacking. Occasionally there seems to be a connection between the disease and changes in the trophic nerves and nerve centers, but practically no findings have been made at the postmortem table which directly connect the nervous system with the disease. The opinion of many competent observers now is that the disease is of toxic origin, the nature of which is not yet explained.

Hartzell[2] believes that pemphigus is of toxic origin. Johnston[3] favors a toxemia of metabolic infectious origin. Highman and Goldenberg[4] believe all varieties of pemphigus are phases of a single disease which is of toxic origin through some undiscovered infectious agent.

From the standpoint of infection, a number of microörganisms have been brought forward, but in view of the fact that the bullæ when formed are usually sterile, no definite results have been obtained. Secondary infection with staphylococci commonly occurs. *Bacillus pyocyaneus* was obtained in pure culture from the blood before, and the heart's blood after death in a case of chronic pemphigus by Petges and Bichelonne.[5] This microörganism has been demonstrated by other observers and in other forms of pemphigus.

Max Joseph[6] noted pemphigus following infection occurring in the gums after the extraction of teeth. Other similar cases are reported. In a case of our own the disease apparently followed a treatment of the gums.

Lipschütz[7] describes organisms which he found in cases of chronic pemphigus, and which he believes are connected etiologically with the disease. The first, a small organism termed *Cytoplasma oviforme*, which measures from 1.5 to 2.7 microns, is egg-shaped and has an eccentric nucleus, extending through the margin of the cytoplasm or just bordering the periphery, giving it a signet-ring shape. This organism appears to have a cyclic existence. A second and smaller one he terms *Anaplasma liberum*. This occurs extracellularly and has practically no cytoplasm, being made up of chromatin or nuclear substance. Lipschütz also found these organisms in cases of dermatitis herpetiformis.

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[1] Vollmer, E., Zeitschrift, 1901, viii, p. 138; White, C. J., Boston Med. and Surg. Jour., 1903, cxlix, p. 297.

[2] Jour. Cut. Dis., 1910, xxviii, p. 111.

[3] Brit. Med. Jour., October 6, 1906, p. 839.

[4] Trans. of Derm. Sec., Amer. Med. Assoc., 1918, p. 217.

[5] Annales, July, 1908, p. 417.

[6] Archiv, 1911, cx, p. 399; abstr. Jour. Cut. Dis., 1912, xxx, p. 222.

[7] Ibid., 1912, cxi, p. 675; abstr. Jour. Cut. Dis., 1913, xxxi, p. 183.

Radaeli[1] described a bacillus cultivated from the bone marrow, heart blood, liver and spleen postmortem, and from the cutaneous lesions and blood during life. Inoculations of these microörganisms into a Rhesus monkey induced vesicular and bullous lesions. He believes this bacillus is important etiologically.

Pathology.—The location of the bullous lesions in pemphigus occurs at various depths in the skin. They are seen immediately beneath the stratum corneum at various depths in the rete and between the epidermis and corium. The contents of the bullæ consist of a fluid of similar character to that of the blood serum in which fibrin-forming bands occur, with leukocytes, both polymorphonuclear and eosinophiles in various stages of degeneration, together with epithelial nuclei and débris of epithelial cells.

Gilchrist[2] describes a section from a bullous lesion as follows: "The contents of the bleb consist of fibrin, coagulated albumin, a few lymphoid cells, numerous polymorphonuclear leukocytes, and a few epithelial cells. The papillary layer of the corium shows the presence of acute inflammatory changes, together with marked serous exudation, particularly around the vessels. The uncovered papillæ form the base of the bleb. The reticular portion of the corium is normal."

In the histology of the lesions studied by the author in a comparatively acute, fatal case, ulceration was found extending through the epidermis. In the corium the vessels were dilated and filled with red cells. Throughout the corium were mast- and round-cells of varying appearance. Masses of staphylococci were noted in the infiltration. The deeper layer of the corium was less involved and the subcutaneous tissue free.

Diagnosis.—Pemphigus is to be differentiated from other bullous disorders. In the bullæ of lepra there is usually coexisting cutaneous anesthesia, and the involution of the bleb is followed by a strikingly characteristic atrophic patch, usually pigmented and insensitive.

No difficulty should be experienced in differentiating this form of pemphigus from the bullous syphiloderm. The latter occurs only in infants, in hereditary lues, and is usually limited to the palms and soles.

From bullous impetigo, particularly that variety formerly classed as pemphigus neonatorum, it is distinguished by the history of contagion and the autoinoculability of the lesions in impetigo.

No difficulty will be found in differentiating pemphigus from such diseases as erysipelas, dysidrosis, hydroa estivale, and other such disorders, when the associated symptoms of these diseases are recognized. The greatest difficulty arises, however, in the differentiation between certain cases of erythema multiforme and dermatitis herpetiformis and pemphigus. In fact, there are certain cases that cannot, even after prolonged study, be positively placed in any one of the categories. As a rule, in dermatitis herpetiformis, the lesions are more multiform, vesicles, pustules, and other lesions being present at some time or other during the course of the disease. From multiform erythema, pemphigus usually is readily distinguished by the peculiar characteristics of the former. In certain bullous varieties of multiform erythema, however, the resemblance is close and, indeed, at times a differential diagnosis is impossible.

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[1] Giorn. Ital. d Mal. Ven. e della Pelle, 1917, v, p. 332; abstr., Brit. Jour. Derm., xxix, p. 294.

[2] Duhring's Cutaneous Medicine, Part 2, p. 461.

From herpes iris, a subdivision of multiform erythema, it is distinguished by the lesions of the latter being more vesicular than bullous and more transitory. They are concentrically arranged, vary in color, and are situated more frequently upon the extremities, especially the backs of the hands.

Finally, some ingested medicaments are capable of producing bullous lesions; for example, potassium iodid. Such a possibility should always be borne in mind when establishing a differential diagnosis. In rare instances, scabies in infants and older children, when characterized by the formation of blebs, might cause confusion, but a history of contagion, the discovery of the parasite, and the location of the lesions will point to the real nature of the disease.

The external application of cantharides, mezereon, the stronger acids, alkalies, and other chemicals may be followed by blebs produced either by accident or intention, with a view to feign disease. The intentional production of such symptoms is usually affected upon the anterior surfaces of the lower extremities, regions within easy reach of the right hand, and the lesions produced are readily distinguished from true pemphigus.

Treatment.--The internal treatment of pemphigus is important because of the marked effect of the disease on the patient's general health. Hutchinson[1] believed that "arsenic is a specific for the state of health upon which relapsing pemphigus depends." This drug is still employed by a number of people, but with indifferent results. In our own experience arsenic has been of but little value. It is a remedial agent of the greatest service in dermatitis herpetiformis of the bullous as well as other types, and its reputation in pemphigus may have been gained through its use in these cases before they were separated from the pemphigus group. Tonics, such as iron, quinin, strychnin and the mineral acids may be given in connection with a nutritious diet. Cod-liver oil and various malt preparations may be of service. Salicin (Crocker) in a dosage of 15 grains (.1) three times daily has been found useful. Cassäet and Micheleau[2] report curative results in the treatment of pemphigus by exclusion of salt from the dietary. Leszcznyski,[1] Kyrl[2] and others report improvement with intravenous injections of quinin. Leszczynski[3] further reports the treatment of twelve cases with quinin, all showing improvement. A 0.5 per cent. solution in normal salt was employed. Approximately 1 gram was administered in 250 c.c. of normal salt solution intravenously every second day.

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[1] Lectures on Clinical Research, London, J. and A. Churchill, 1878, p. 49.

[2] Archiv. de Méd., gén. January 16, 1906.

Strumphe[4] reports favorable results in two cases from treatment with intravenous injections of salvarsan. Sutton[5] has had a similar experience. Fischer[6] injected 20 c.c. of heterogenous blood serum in a case of pemphigus with exacerbation of the symptoms of the disease. This was followed by an injection of neosalvarsan, which apparently cleared up the disorder. Arsphenamin in our own experience has been of no value in the treatment of pemphigus.

The local treatment of pemphigus includes the opening of large bullæ and their subsequent dressing with lotions or ointments, equal parts of olive oil and lime water, or olive oil containing 15 grams of ulmus pulvis in 240 grams of oil. Useful ointments consist of equal parts of Hebra's diachylon ointment and the official oxid of zinc ointment, or cornstarch 25 parts, zinc oxid 25 parts, naftalan 50 parts, or a simple ointment containing boric acid or ichthyol. A simple dusting powder such as those recommended in eczema may be used at a later period. In a severe case under observation over quite a period of time the dry treatment recommended for burns was employed with success. In severe cases a continuous water-bath may be employed for some hours each day, or for some days at a time, using in the interim some of the above-mentioned preparations.

Prognosis.—Every case of pemphigus should be regarded as grave. Remissions occur and complete arrest of symptoms over varying periods are reported.

Pemphigus Foliaceus.—Pemphigus foliaceus is a rare form of the disease, which may originate in one of the common dermatoses or in a grave form of pemphigus vulgaris, or may at the onset present characteristic features. Hallopeau and Fournier,[7] Low,[8] Biddle,[9] and others have reported cases which began as a dermatitis herpetiformis.

Symptoms.—The lesions are flaccid bullæ, which are developed upon the apparently normal skin, and which speedily rupture and discharge their contents, leaving beneath an excoriated, reddish or purplish, and at times inflammatory, surface. Often the blebs are defined so poorly that the epidermis seems scarcely raised from the tissue beneath, the

condition resembling that of the skin to which a blister has been applied with the result of imperfect vesication. The contents, at first pellucid or lactescent, become later purulent or sanguinolent. Occasionally, successive blebs form in the same area, producing many layers, the condition resembling an exfoliative dermatitis.

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[1] Archiv, 1912, cxiv, p. 129, and cxviii, p. 633; abstr., Brit. Jour. Derm., 1912, xxiv, p. 447.

[2] Archiv., 1913, cxv, p. 636

[3] Ibid.

[4] Berlin klin. Wochenschrift, July 1, 1912, xlix, No. 27, p. 1267; abstr., Jour. Cut. Dis., 1912, xxx, p. 626.

[5] Boston Med. and Surg. Jour., 1911, lcxiv, p. 336

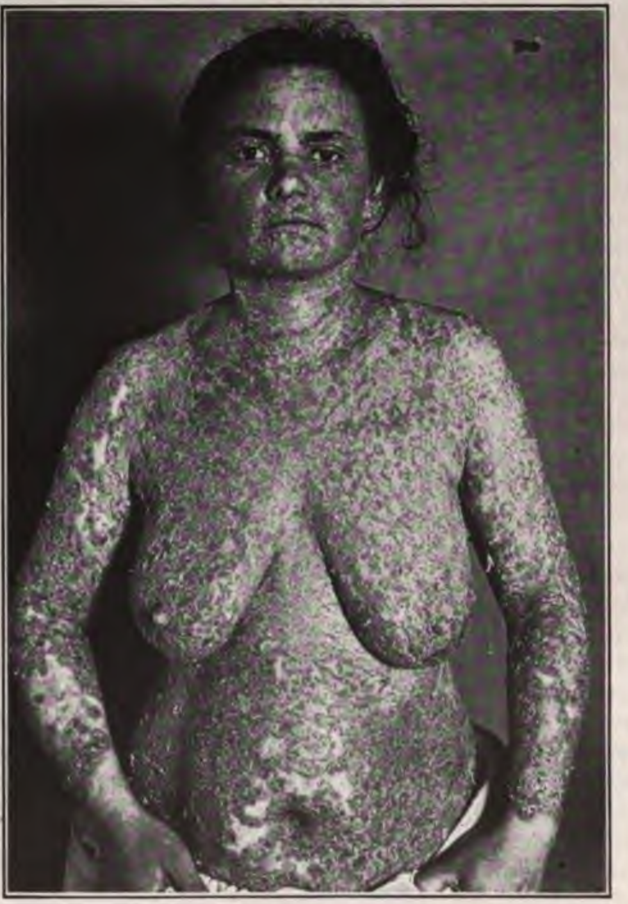
[6] Derm. Wchnschr., 1916, lxii, p. 25.

[7] Bull. Soc. franç. de Derm. et de Syph., 1892, p. 470.

[8] Brit. Jour. Derm., 1909, xxi, pp. 101 and 135 (full bibliography).

[9] Jour. Cut. Dis., 1897, xv, p. 203.

Fig. 129



Pemphigus foliaceus. (Fordyce and MacKee.)

When rupture of the blebs occurs, there form yellowish-brown crusts, attached at the center, while their edges remain free. These edges, visible over the affected surface in polycyclic or irregular outlines, incompletely hiding the raw and sodden epidermis, present a characteristic picture. The crusts have often been likened to flaky piecrust. In this variety Nikolsky's sign is characteristic. This is the ready removal of the skin following the slightest injury, or, as has been stated, the skin may be brushed off with little effort.

The disease spreads gradually until it becomes symmetrical and universal, a peculiarity which distinguishes it from other varieties of pemphigus. As the disorder advances, the patient lies in a pitiably helpless condition, the remaining epidermis being completely undermined by the serum exuded, in places exposing large areas of denuded skin and in a condition of inflammation of a low grade. Even, however, when the disease is fully generalized, the appetite and bowel function are at times unimpaired.

In its later stages, after it has become generalized, it may lose its pemphigus characteristics. In these instances large masses of greasy scales are exfoliated from the surface, the moisture exuding from which is scarcely sufficient to attract attention. The odor from the body becomes offensive; fissures form in the infiltrated skin; the facies of the patient become repulsive; the hands swollen and the fingers contractured.

The disease affects the mouth and throat, denuding the mucous surfaces of the epithelium. The scalp becomes affected along with portions of the body. The hairs remain attached for a long time, but eventually they are completely lost. Over the face, at first merely reddened and scaling, occur retractive processes, which at times produce ectropion and consequent conjunctivitis. Over the body, especially at points pressed upon when reclining, ulcerations may destroy the skin. The palms and soles are infiltrated and fissured rather than the seat of much exudation. The nails are commonly furrowed and distorted; occasionally, they are shed. The subjective sensations are those of burning, smarting, and soreness, rather than of itching.

There may be no fever or there may be a rise of body temperature, with recurrence of lesions, which, in a late stage of the disease, appear in the sites of those which have been followed by imperfect attempts at repair, a thin and glazed epidermis forming, in cases of chronic type, in the sites of former bullæ. In other cases the temperature remains above normal for weeks at a time, especially in advanced stages of the disease. The disorder may complete its course in a few months or may persist for years. Adamson[1] recorded a case of sixteen years' duration. Death usually results from exhaustion; occasionally, an intercurrent pneumonia or diarrhea concludes the history.

Pemphigus foliaceus commonly attacks adults, but Brand[2] reports the occurrence of the disease in a newborn child.

Etiology.—The disease may be such from the beginning, or may develop on a dermatitis herpetiformis or a chronic pemphigus. In certain cases a mental or physical shock has preceded the disorder. It has developed during the course of pregnancy (Hardaway). Leredde considers it a blood disease due to the action of some toxic substance on the blood-forming organs, particularly the bone-marrow. The toxic theory, which is the one accepted by most observers, covers a comparatively large field. The toxins may be produced either with or

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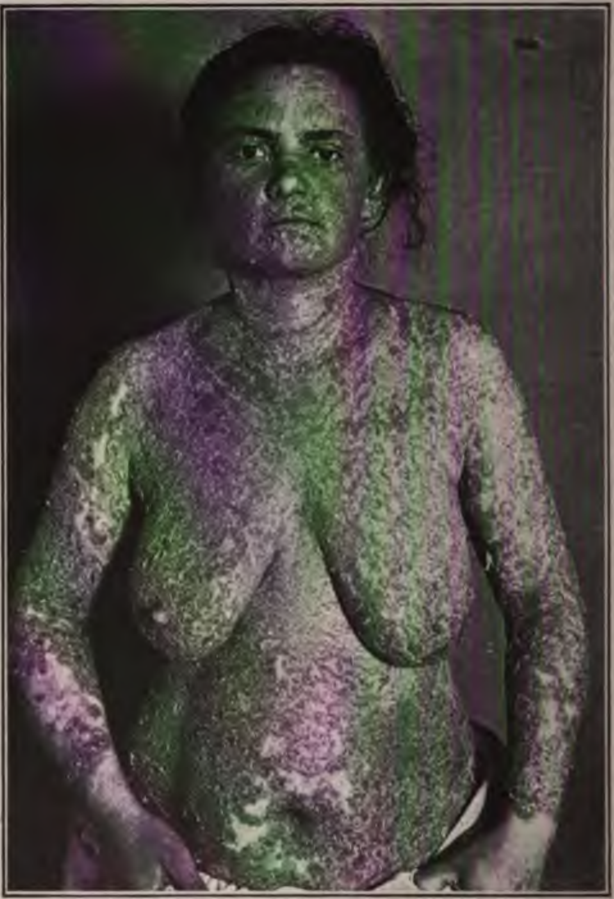
[1] Brit. Jour. Derm., 1910, xxii, p. 381.

[2] Brit. Med. Jour., June 7, 1902.

FIG. 129



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The disease spreads gradually until it becomes symmetrical and universal, a peculiarity which distinguishes it from other varieties of pemphigus. As the disorder advances, the patient lies in a pitiably helpless condition, the remaining epidermis being completely undermined by the serum exuded, in places exposing large areas of denuded skin and in a condition of inflammation of a low grade. Even, however, when the disease is fully generalized, the appetite and bowel function are at times unimpaired.

In its later stages, after it has become generalized, it may lose its pemphigus characteristics. In these instances large masses of greasy scales are exfoliated from the surface, the moisture exuding from which is scarcely sufficient to attract attention. The odor from the body becomes offensive; fissures form in the infiltrated skin; the facies of the patient become repulsive; the hands swollen and the fingers contractured.

The disease affects the mouth and throat, denuding the mucous surfaces of the epithelium. The scalp becomes affected along with portions of the body. The hairs remain attached for a long time, but eventually they are completely lost. Over the face, at first merely reddened and scaling, occur retractive processes, which at times produce ectropion and consequent conjunctivitis. Over the body, especially at points pressed upon when reclining, ulcerations may destroy the skin. The palms and soles are infiltrated and fissured rather than the seat of much exudation. The nails are commonly furrowed and distorted; occasionally, they are shed. The subjective sensations are those of burning, smarting, and soreness, rather than of itching.

There may be no fever or there may be a rise of body temperature, with recurrence of lesions, which, in a late stage of the disease, appear in the sites of those which have been followed by imperfect attempts at repair, a thin and glazed epidermis forming, in cases of chronic type, in the sites of former bullæ. In other cases the temperature remains above normal for weeks at a time, especially in advanced stages of the disease. The disorder may complete its course in a few months or may persist for years. Adamson[1] recorded a case of sixteen years' duration. Death usually results from exhaustion; occasionally, an intercurrent pneumonia or diarrhea concludes the history.

Pemphigus foliaceus commonly attacks adults, but Brand[2] reports the occurrence of the disease in a newborn child.

Etiology.—The disease may be such from the beginning, or may develop on a dermatitis herpetiformis or a chronic pemphigus. In certain cases a mental or physical shock has preceded the disorder. It has developed during the course of pregnancy (Hardaway). Leredde considers it a blood disease due to the action of some toxic substance on the blood-forming organs, particularly the bone-marrow. The toxic theory, which is the one accepted by most observers, covers a comparatively large field. The toxins may be produced either with or

condition resembling that of the skin to which a blister has been applied with the result of imperfect vesication. The contents, at first pellucid or lactescent, become later purulent or sanguinolent. Occasionally, successive blebs form in the same area, producing many layers, the condition resembling an exfoliative dermatitis.

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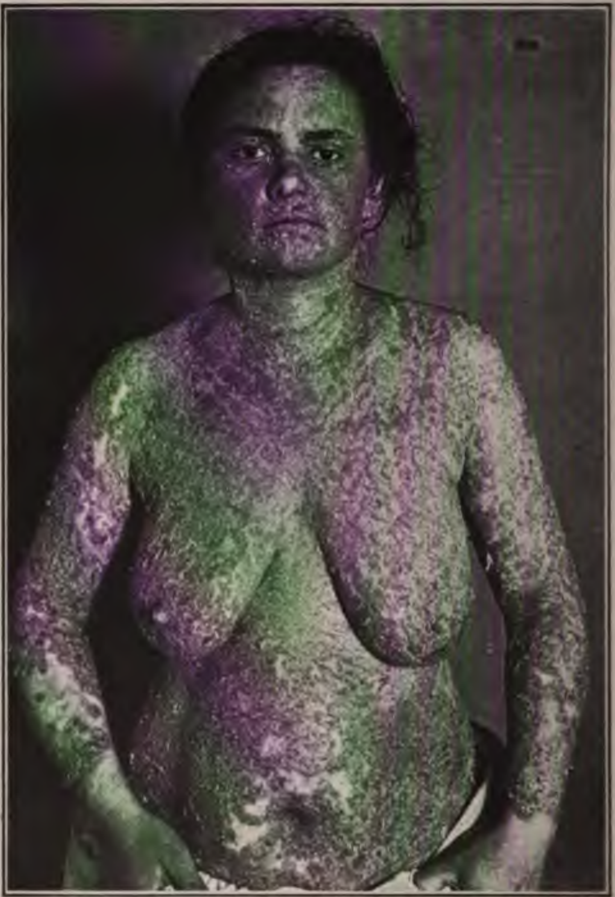
[1] Brit. Jour. Derm., 1910, xxii, p. 381.

[2] Brit. Med. Jour., June 7, 1902.

Fig. 129



Pemphigus foliaceus. (Fordyce and MacKee.)

When rupture of the blebs occurs, there form yellowish-brown crusts, attached at the center, while their edges remain free. These edges, visible over the affected surface in polycyclic or irregular outlines, incompletely hiding the raw and sodden epidermis, present a characteristic picture. The crusts have often been likened to flaky piecrust. In this variety Nikolsky's sign is characteristic. This is the ready removal of the skin following the slightest injury, or, as has been stated, the skin may be brushed off with little effort.

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without the aid of microörganisms, and to a certain extent the nervous system is associated with such changes. Hartzell,[1] Low,[2] and many others record their opinion in favor of the toxic theory.

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[1] Brit. Jour. Derm., 1910, xxii, p. 381.

[2] Brit. Med. Jour., June 7, 1902.

The neuropathic theory, as in other varieties of pemphigus, stands first in the estimation of many. In repeated examinations, no gross changes have been found in the nervous system.

The parasitic theory has several supporters. Hazen[3] attributes the disease to *Bacillus pyocyaneus*, which he found in the blood, urine, and vesicles of the cutaneous surface in a patient suffering with pemphigus foliaceus. The same author[4] concludes that some cases of pemphigus foliaceus are undoubtedly due to *Bacillus pyocyaneus*, and that general infection and death may take place from the same organism. Low,[5] after investigating the microbic theory, believes that it cannot be substantiated, but after an extensive study concludes that the disease is toxic.

DuMesnil de Rochemont[6] reported a case of typical pemphigus foliaceus following a whitlow produced by a thorn, which subsequently led to infection of the arm, with lymphangitis and suppuration.

Pathology.—The histology of this disease has been well described by Unna,[7] Nikolsky, Leredde,[8] Gilchrist,[9] and others. In the corium great vascular dilatation is present, both in the papillary and deeper layers. The lymphatic vessels and spaces, especially the former, also show dilatation. There is a cellular infiltration, composed of lymphocytes, polymorphonuclear leukocytes, and a few eosinophiles. Leredde and Fabry noted pigment-cells and free pigment, also some deeply situated mast-cells. Leredde found many eosinophiles in the dilated vessels of the papillæ. In the epidermis the rete-pegs are greatly elongated and the epithelial cells over the papillæ are thin. In these situations the horny layer lies close to the edematous papillæ, which are naturally elongated to correspond with the interposed rete pegs. The epithelial cells show edema, the prickles often being destroyed and the individual cells separated by spaces, with occasional microscopic vesicle formation. Migratory cells are found interspersed. The bullous formation occurs at different levels, and has been described as occurring all the way from between the epidermis and the corium to just below the stratum corneum. Gilchrist's description of an early lesion is important. In this case the bleb was situated in the rete, and contained coagulated serum, fibrin, and numerous polynuclear leukocytes, many of which were seen migrating through the basal epidermis. The interepithelial spaces were enlarged, and the corium showed acute inflammatory changes, indicated by vascular dilatation and perivascular infiltration of polynuclear leukocytes and lymphoid cells. The papillæ beneath the bleb were flat.

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[1] Loc. cit.

[2] Loc. cit.

[3] Jour. Cut. Dis., 1910, xxviii, p. 118

[4] Ibid., 1912, xxx, p. 325.

[5] Loc. cit.

[6] Archiv, 1895, xxx, p. 163.

[7] Histopathology, Walker's Translation, 1896, p. 174.

[8] Annales, 1899, p. 601.

[9] Duhring's Cutaneous Medicine, Part 2, p. 463.

The blood changes described are variable. Eosinophiles have been found increased in a number of cases, but in many others no increase was noted. Leredde[1] has chiefly emphasized this point. Schalek[2] noted a moderate eosinophilia in his case.

Diagnosis.—The disease is to be differentiated chiefly from dermatitis exfoliativa. As certain cases develop into a general exfoliating stage, the differentiation is difficult. The presence of flaccid bullæ or a history of such, the moist or oozing surfaces, the crust formation, and the peculiar odor exhaled by the patient are important in diagnosis. Dermatitis exfoliativa is a dry, scaling disorder, as a rule. Nikolsky's sign (the stripping of the epidermis) is usually present. Jamieson's statement that pemphigus foliaceus presents the appearance of a general exfoliative dermatitis with pemphigus implanted is very apt in many cases.

The disease also must be differentiated from a generalized eczema. The large crusts, peculiar odor, and presence of flaccid bullæ are usually sufficient to distinguish the two. However, some observation may be necessary in certain cases to determine the above characteristics.

The disease must also be differentiated from certain cases of chronic pemphigus and dermatitis herpetiformis, but by recalling the essential symptoms of these disorders the diagnosis can usually be made, though at times pemphigus foliaceus is a sequel to these maladies.

Treatment.—A water-bed is advisable, and at times a continuous water-bath. Careful cleansing of the skin is necessary. Arsenic and quinin may be tried, but as a rule they are of no value. Autogenous vaccine also should be used, as it has proved of some value. Owing to the great chance for absorption, the local treatment must be carefully watched and only the blandest and non-toxic applications made. Oleated lime-water, plain unguentum aquæ rosæ, and simple dusting-powders are suggested. Caution is advised with the latter, however, as they tend to dry and form crusts, beneath which infection may spread. The alternate use of a continuous bath, if available, for one or two days at a time, followed by a dusting-powder of simple borated talcum or a mild boric-acid ointment, keeps the skin clean and the patient more comfortable than other methods.

Prognosis.—The disease, as a rule, terminates fatally, hence any treatment is only palliative.

Pemphigus Vegetans (*Erythema bullosum vegetans, Herpes vegetans, Condylomatosis, Pemphigoides maligna*).—Neumann[3] in 1886 was first to describe the disease which has since been studied by a number of observers. Crocker published an excellent monograph, giving tabulated results in some 18 cases, and Hyde[4] published a report of the first case recorded as such in the United States. Winfield,[5] in 1907, reported a typical case, and in a review of the literature records 58 authentic cases up to that date.

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[1] Loc. cit.

[2] Jour. Amer. Med. Assoc., July 2, 1910, p. 4.

[3] Vierteljahr, 1886, Bd. xiii.

[4] Jour. Cut. Dis., 1891, ix, p. 412.

[5] Ibid., 1907, xxv, pp. 17 and 71.

Symptoms.—The onset of the disease may be marked by languor, malaise, febrile symptoms of moderate severity, and ill-defined symptoms of impaired health, followed by the development of lesions in the mouth or in the skin; or the first manifestation of the disease may be the mucous membrane or cutaneous lesions accompanied by other symptoms. In the mouth, white patches, which are ill-developed blebs and exhale an unpleasant odor, are visible upon the mucous surface. The detached membrane forming each spot finally is loosened and leaves behind equal-sized, excoriated patches, which produce extreme soreness of the mouth, and which as some heal are succeeded by others. In severe cases they render mastication and deglutition painful; and in patients in whom this becomes a prominent feature of the case the nutrition of the body as a consequence is seriously impaired.

The skin lesions may precede or follow those in the mouth. They commonly are seen in women first about the vulva, spreading over the ano-genital region and umbilicus as closely-set bullæ, covered with a mucoid, whitish secretion, the features thus strongly resembling the appearance of condylomata of the same region. In connection with the mouth lesions, the suggestion that syphilis is present is very striking, and has led to this error of diagnosis in a number of instances. In other cases the scalp, hands, feet, axillæ, and other parts are involved primarily. Pollitzer[1] reported a case, which proved fatal in six months, in which the disease began as a condylomatous lesion about the anus. Wilfrid Fox[2] fully describes a case in which the lesions began upon the neck.

The bullous or vesico-bullous efflorescences, which at first resemble those of other forms of pemphigus, speedily exhibit erosions, ulcerations, or more commonly, vegetating masses, the change from the bleb to a fungoid, papillomatous growth being scarcely appreciable. Botelli[3] describes the evolution of the lesions as follows: First, a bulla, tense if clear, flaccid if purulent, that ruptures instead of healing. Papillomatous granulations spring up, which discharge a fetid, purulent liquid. The vegetations become covered with a brownish-yellow crust, are well defined and are surrounded by a red halo.

The lesions may coalesce and tend to become grouped about the axilla, the circle at the root of the neck, the bend of the elbow, the hand, the foot, and the scalp; but they have no tendency to become universal, even when extensive. The nails may become affected, as the result of the formation of blebs in the matrix. A singular change in the skin, where typical well-formed bullæ have developed and healed, is a deep pigmentation, in puncta resembling comedones, with pinpoint-sized verrucoid elevations of the surface. In some regions the sequence of the closely packed blebs, followed by vegetating masses, resembling that seen in pemphigus foliaceus, in which, especially over the back after long decubitus, there form large, granulating erosions, exquisitely painful and conducive to a rapidly fatal issue. Indelible scarring may result.

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[1] Festschrift zur Vierzigjährigen Stiftungsfeier des Deutschen Hospitals, New York, 546; abstr., Brit. Jour. Derm., 1911, xxiii, p. 335.

[2] Brit. Jour. Derm., 1908, xx, p. 181.

[3] Giorn. ital. d. Mal. Ven. e della Pelle, lii, p. 498; abstr., Brit. Jour. Derm., 1911, xxiii, p. 370.

The disease is characterized by periods of partial remission and exacerbation, lasting for months and occasionally for years. It is in the large majority of cases eventually fatal. Variations occur, chiefly in the degree of febrile temperature, probably always reactive; in the severity of the buccal lesions; and in the extent of the eruption.

In the case reported by Zumbusch[1] nut-sized and larger tumors developed from the papillary excrescences.

A survey of all reported cases emphasizes the view taken by Dubreuilh, Tommasoli, and Winfield, that the cases described by the various authors represent two widely different types. In the true pemphigus vegetans of Neumann, which is probably invariably fatal, lesions of grave significance develop beneath the first-formed blebs. A second group includes the cases in which a few recoveries have been reported, where the bullæ of chronic pemphigus have been complicated by vegetations springing from the seat of bullous lesions, or where vegetating lesions have developed in the course of an attack of dermatitis herpetiformis. Constantin,[2] in reporting a case of pemphigus vegetans developing upon dermatitis herpetiformis, ventures the opinion that benign pemphigus vegetans is really dermatitis herpetiformis with vegetations.

Etiology.—The cause of the disease has not been demonstrated. A number of bacteria have been isolated, the chief among which are the following: Winfield[3] isolated in pure culture *Bacillus pyocyaneus* and *Streptococcus pyogenes aureus*, and was inclined to place some significance on the finding of the first of these two organisms. He further reports from the literature the finding of *Staphylococcus pyogenes aureus* by Marinelli; a coccus by De Michele; a small bacillus and a streptococcus by Gastou; a diplococcus by Philippson and Fileti; a pseudodiphtheritic bacillus by Waelsch; *Staphylococcus aureus* by Hamburger and Rubel; a bacillus and a coccus by Cheeseman (in Hyde's case); the pseudodiphtheritic bacillus by Stanziale; and *Bacillus pyocyaneus* and *Bacillus coli communis* by Pernet. In some of the above instances the organisms described have been found not only in lesions but in the blood; but as yet the findings are too widely diversified for one to arrive at conclusions.

The neuropathic theory is also brought into account in these cases.

Pathology.—The pathogenesis is not understood, but the disease is best explained by a toxemia of bacterial or other origin. The histology has to be considered from different standpoints: First, as it is found in the early bullous lesion; and, second, in the vegetating masses. Weidenfeld, in an exhaustive study of the histology of the disease, calls attention to the enormous dilatation of lymph vessels always present, together with a more or less extensive perivascular cellular infiltration, edema of the papillary layer of the cutis, changes in the elastic-tissue fibers, and edema in whole or in part of the rete. He believes the dilatation of the vessels to be idiopathic, and thinks there may be a circulating substance in the blood serum doing harm to the vessel walls; and that disturbance of innervation alone could hardly account for the changes.

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[1] Archiv, 1905, lxxiii, p. 121.

[2] Annales, 1907, p. 641; abstr. Brit. Jour. Derm., 1908, xx, p. 204.

[3] Loc. cit.

Hartzell[1] found the blebs situated between the epidermis and the corium, and filled with eosinophiles and a few leukocytes, together with a large number of round cells of uniform size, having a limiting membrane and containing a large cavity. In the papillary and subpapillary layers of the corium was a moderate cellular infiltration, composed largely of eosinophiles. The papillæ were greatly enlarged, especially in the longitudinal direction.

Diagnosis.—Pemphigus vegetans is to be distinguished chiefly from syphilis and the peculiar eruption occasionally produced by the bromids and iodids in which vegetations occur. In a large proportion of cases pemphigus vegetans has been mistaken for syphilis. The close crowding of the lesions about the ano-genital region and their striking resemblance to condylomata, taken in connection with the presence of erosions of the mucous membrane of the mouth, cause the confusion. In pemphigus vegetans the vegetations are more superficial than in syphilis, are of more rapid evolution, and exhibit groups of blebs at the border of the lesion; while the genital condylomata have a smooth border unaccompanied by bullæ. Further, the surface is stippled (Neumann) and never smooth, as in condylomata, and the mouth lesions are far more painful. However closely packed together may be the condylomata of the genital region, they rarely spread, as does pemphigus vegetans, beyond the regions adjacent to the mucous outlets; while the bullæ of pemphigus vegetans, when the disease is fairly advanced, are not only numerous, and closely packed together, but they spread also beyond, high toward the pubes, and low over the inner faces of the thighs. There is commonly a history of fever, no lymphatic involvement, and a distinct uniformity of lesions, each separate element being of a bullous type.

That drug eruptions produce similar lesions should be borne in mind. In the latter case the general health of the patient is unaffected, the mucous membranes rarely involved, and the distribution of the lesions is apt to be about the face and extremities. Early many papulopustular lesions will be present in addition to the vesico-bullous and vegetating variety.

Treatment.—The management of a case of pemphigus vegetans resolves itself into keeping the skin clean, supporting the strength of the patient, and careful nursing. As above stated, in the true cases a fatal termination always occurs, and therefore the treatment has been unsuccessful. The internal treatment consists of the usual tonics—arsenic, iron, quinin and strychnia,—in connection with a nutritious diet. The local treatment of the lesions should consist first, in great cleanliness, which may be accomplished by bathing or by the use of the continuous bath as much of the time as the patient can stand it. The bullæ may be punctured and the parts dressed, when not in the bath, with a wet dressing of boric acid; or a dusting-powder may be applied after the surface has been cleansed; or the surface may be treated with oleated lime-water, in which opium, phenol, or dilute hydrocyanic acid has been added in the same proportions suggested in the treatment of acute eczema. Weak sulphur ointments and salicylic-acid pastes may also be used to advantage. The ordinary lead-and-opium wash, with or without the addition of zinc oxid, is useful. The lesions in the scalp require the cutting short of the hair of the head in order to make the applications.

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[1] Jour. Cut. Dis., 1910, xxviii, p. 111.

In cases where the toxemia is marked, supportive treatment is demanded. Pernet[1] used an autogenous vaccine, made from cultures of a coccus and a diplococcus isolated from the skin and blood of a patient suffering with pemphigus vegetans. In addition, the patient had quinin and arsenic internally, but the case terminated fatally. Schamberg[2] noted no improvement in a case treated with an intravenous injection of 0.4 grams of salvarsan. Weiss[3] reported improvement in a case treated with *x*-rays.

Prognosis.—From the foregoing, it is evident that the prognosis is bad. In the so-called true cases of Neumann, the patients have all succumbed to the disease, and the prognosis, therefore, depends in any individual case on whether it is one of the true type or one with vegetating lesions occurring in some other disease.

HYDROA VACCINIFORME.

Synonyms.—Recurrent summer eruption; Hydroa æstivale.

Definition.—Hydroa vacciniforme is a recurring vesicular disease, occurring chiefly in the summer season, in young adult male subjects, and solely on exposed parts of the cutaneous surface.

The disease was described first in 1861 by Bazin and later by Hutchinson,[4] Jamieson,[5] Brooke,[6] Crocker,[7] Bowen,[8] Graham,[9] White,[10] Brocq,[11] and others.

Symptoms.—The disease usually begins during the first three or four years of life and gradually disappears during the few years following puberty. With but few exceptions, the cases reported have been in boys. The disease is most active in summer, the larger number of patients remaining free from active manifestations during the winter months. The direct cause in most cases is exposure to the sun's rays, though exceptionally warm or cold winds, or even artificial heat, seem sufficient to cause an outbreak.

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[1] Archiv, 1911, cx, p. 509; abstr. Jour. Cut. Dis., 1912, xxx, p. 224.

[2] Jour. Cut. Dis., 1912, xxx, 359.

[3] Ibid., 1913, xxxi, p. 509.

[4] Clinical Soc. Trans., vol. xxii.

[5] Lancet, August 18, 1889.

[6] Brit. Jour. Derm., 1892, iv, p. 128.

[7] Diseases of the Skin, 1893, p. 325.

[8] Jour. Cut. Dis., 1894, xii, p. 89 (with review of literature, and histology).

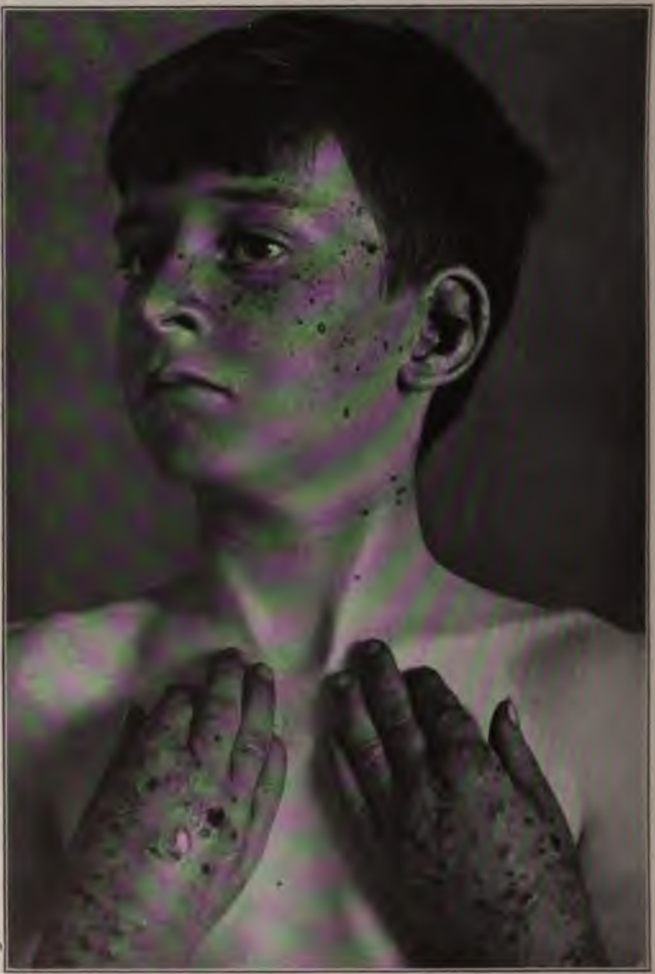
[9] Ibid., 1896, xiv, p. 41.

[10] Ibid., 1898, xvi, p. 514.

[11] Annales, 1894, s. iii, v, p. 1003.

The eruption is symmetrical and is usually limited to the uncovered parts of the body; the bridge of the nose, cheeks, and ears, and the backs of the hands are the parts most affected. Bazin reported cases in which covered portions of the body were slightly involved. In a case observed by Dr. Hyde, a new crop of vesicles and bullæ on the face was accompanied at times by a herpetic keratitis, the resulting scars interfering considerably with vision. The disease occurs in successive outbreaks, each of which lasts for two or three

Fig. 130



Hydroa æstivale. (Fordyce.)

weeks. The intervals between recurrences in the summer may be several weeks, or so brief as practically to be wanting. The lesions often are preceded by sensations of heat or itching, and the first to appear are red macules or elevations, upon which are rapidly formed vesicles or bullæ, varying in size from that of a millet-seed to that of a large pea, and occurring either singly or in groups like herpes. They may coalesce and may be surrounded by a halo. These vesicles may dry in a day or two, or they may rupture and form a crust; but many of the larger become depressed in the center and resemble a vaccination-vesicle. The depressed center is black or dark-blue, and is surrounded by a ring of fluid, while about the whole is a reddened areola. Some of the lesions may become purulent. The dark center is converted rapidly into a thick, black crust, which is very adherent, and which on falling leaves a depressed, reddened scar, that eventually becomes white and practically indistinguishable from that of variola. The duration of an individual lesion from its beginning to the formation of the crust is three or four days. The time required for the crust to fall is variable.

The eruption usually is preceded by some slight constitutional disturbance, and by burning or pain at the site of the lesions. Itching is absent, as a rule, though it was marked in Bowen's case.

Hydroa Puerorum.—Hydroa puerorum, originally described by Unna, presents many features in common with the summer eruptions above described. The disease begins usually in the first year of life, each attack lasting about two weeks. The lesions are erythemato-vesicular, the vesicles frequently coalescing to form bullæ. The lesions may occur on any part of the body, not selecting particularly the exposed surfaces, and undergo involution without subsequent sequels. The eruption is preceded and accompanied by itching and burning sensations. Season is not concerned in the etiology, and spontaneous recovery occurs at puberty. Adding to the above the superficial character of the lesions, which are confined to the rete, Haase and Hirschler[1] make of this disorder an entity.

Etiology.—Exposure of sensitive skins to the sun and wind, especially in the summer season, is the effective cause. We have observed patients in whom the disease was developed not merely in summer, but in winter, when the sunlight was reflected from snow on the ground. Ehrmann[2] showed that light passed through blue glass was just as potent in causing the eruption as the sun's rays; but when the light was passed through red glass, which absorbs the actinic rays, no lesions resulted. He concluded that the eruption is of photo-actinic origin.

Pathology.—The pathology has been studied by Bowen in two lesions taken from a single patient, and by Mibelli.[3] In the primary stage, Bowen found merely vesicle-formation in the middle layers of the rete. In a more advanced lesion he found necrosis involving the lower layers of the stratum corneum, the entire rete, and the corium nearly to the subcutaneous tissue. He concludes that the process begins as an inflammation in the epidermis and upper part of the corium, followed by vesicle-formation in the rete, and later by the necrosis described above. The necrosis is sharply circumscribed, and, showing through the vesicles above, produce the black centers of the advanced lesions. Bowen further calls attention to the points of similarity between this disease and acne necrotica or acne varioliformis.

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[1] Trans. VI Internat. Derm. Cong., New York, 1907, vol. i, p. 413, and Jour. Cut. Dis., 1908, xxvi, p. 199.

[2] Archiv, 1906, lxxvii, p. 163.

[3] Monatshefte, 1897, xxiv, p.

Adamson,[1] in a recent histological study, found vascular dilatation; swelling of the connective-tissue bundles and of the prickle-cells; a serous and cellular exudation breaking through and destroying the prickle-cell layer, forming a vesicle beneath the raised horny layer; and destruction of the superficial part of the papillary layer of the corium.

Diagnosis.—The diagnosis is from erythematous lupus, pemphigus, erythema bullosum, and dermatitis herpetiformis. The limitation of the lesions to the exposed parts of the body, the presence of vesico-blebs, and the scarring, in connection with the age of the patient, all point to the nature of the malady.

Treatment.—The treatment is unsatisfactory. To prevent recurrence, the patient should be guarded from exposure to the sun, and in some cases from hot and cold winds. Veils and coverings which exclude the light may be of service. Crocker recommends treating the eruption by opening the vesicles and applying iodoform in powder or in solution in ether. After removing the crusts with carbolized oil, the surfaces may be dressed with an ointment containing iodoform and boric acid.

Prognosis.—Until adult years are attained, the patient is liable after fresh exposure to recrudescence of the disease.

EPIDERMOLYSIS BULLOSA.

Synonyms.—Acanthosis bullosa; Epidermolysis bullosa hereditaria.

Definition.—This is a rare affection of the skin in which vesicles or bullæ are produced by slight traumatism. Cases have been reported by Goldschneider,[2] Köbner,[3] Valentine,[4] Elliot,[5] Beattie,[6] Bowen,[7] Wende,[8] Engman and Mook,[9] Kanoky and Sutton,[10] Sakaguchi,[11] and others.

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[1] Brit. Jour. Derm., 1906, xviii, p. 125. (On cases of hydroa æstivale of mild type, their relationship to Hutchinson's "summer prurigo" and to hydroa vacciniforme.)

[2] Monatshefte, 1882, i, p. 150.

[3] Deutsche. med. Wchnschr., 1886.

[4] Berlin. klin. Wchnschr., 1885, p. 150

[5] Jour. Cut. Dis., 1895, xii, p. 10; ibid, 1889, vii, p. 539; and New York Med. Jour., April 21, 1900.

[6] Brit. Jour. Derm., 1897, ix, p. 301, resumé of all previously reported cases.

[7] Jour. Cut. Dis., 1898, xvi, p. 253.

[8] Ibid., 1902, xx, p. 537, bibliography; ibid., 1904, xxii, p. 14.

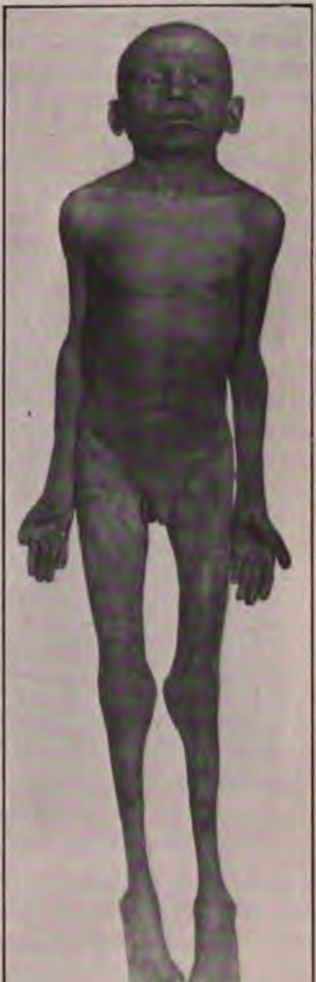
[9] Ibid., 1910, xxviii, p. 276.

[10] Jour. Amer. Med. Assoc., April 2, 1910, p. 1137.

[11] Archiv, 121, p. 379, "Epidermolysis bullosa hereditaria;" an extensive study of the subject with the report of Japanese cases.

Symptoms.—Two forms of the disease are described, the simple and dystrophic. In the simple form bullæ arise after irritation or trauma. They may appear on any portion of the body subjected to such irritation. After healing temporary pigmentation occurs, but no permanent changes follow. In this variety the mucous membranes are rarely involved. The lesions begin shortly after birth or within the first two years and not infrequently subside at puberty. In the dystrophic form the lesions occur particularly on the extremities, chiefly over the hands, feet, elbows and knees, and, like the other form, follow friction or trauma. In this variety the bullæ are not infrequently hemorrhagic. Scarring and pigmentation occur, crusts form over the denuded areas, and the skin about the joints may show atrophic changes. In other locations thickened and hyperemic areas, as well as scaling patches, occur. A sequel which is sometimes seen in pemphigus and has been noted by the author and many observers, is the occurrence of grouped milia occurring particularly on the dorsal surface of the hands and extensor surface of the forearms. The nails show many dystrophic changes and are sometimes destroyed. The mucous membrane of the mouth and tongue may present bullæ, infiltrated areas or patches of leukoplakia. Klausner[1] reports recurring lesions in the mouth in a patient suffering with this disease, who at the age of twenty-five years developed carcinoma in this situation.

FIG. 131



Epidermolysis bullosa hereditaria.

Subjective sensations vary. Itching may or may not be present. In some instances painful erosions occur to such a degree as to interfere with the occupation of the individual. The disorder lasts indefinitely. While the major number of these cases begin shortly after birth, cases of similar nature are reported as beginning later.[2] Two such instances have been under the observation of the author. The first, a man aged thirty-seven years, presented typical lesions of the disorder which had been present for one year. This case was demonstrated before the Chicago Dermatological Society in November, 1911. In acquired cases the clinical manifestations are identical with the hereditary form. Fordyce[1] suggests that there may be transitional forms between this disease and pemphigus.

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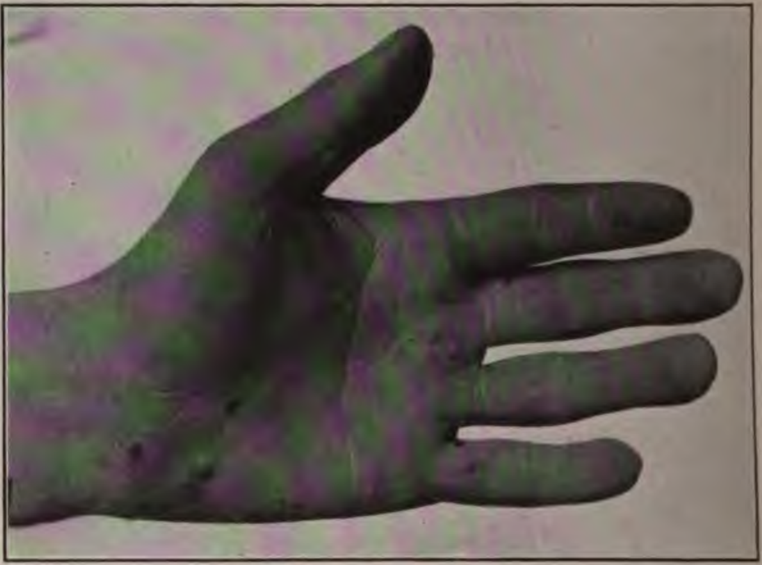
[1] Archiv, March, 1913, cxvi, p. 71; abstr., Brit. Jour. Derm., 1913, xxv, p. 274.

[2] Wise and Lautman, Jour. Cut. Dis., 1915, xxiii, p. 441; "Epidermolysis bullosa beginning in adult life; the acquired form of disease." Report of a case and review of the literature.

Etiology and Pathology.—Aside from the congenital or hereditary character of the disease very little has been ascertained. The occurrence of the disease in several members of a family, over several generations, has been reported. The disorder, however, occasionally begins in adult life. Much difference of opinion exists as to the pathogenesis of this disease. It has been viewed as an angioneurosis, trophoneurosis, a dermatitis, and, more recently, it has been attributed to the congenital loss of elastic tissue. Elliot found the bullæ deeply situated with practically the entire epidermis as the roof wall, and with the papillary layer of the corium as the base. The papillæ were swollen and edematous, and the bloodvessels were greatly dilated but showed no perivascular cellular infiltration. In the sub-papillary region there were edema and dilatation of the bloodvessels and also perivascular cellular infiltration. The cells were chiefly leukocytes, but there was an increase in the connective-tissue cells. From these findings Elliot concluded that the process was an inflammatory one in which the

Fig. 132



Epidermolysis bullosa acquisita.

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[1] Ibid., 1912, xxx, p. 168.

bullous formation was the consequence of the constant serous exudation in an individual with an acquired or hereditarily exaggerated irritability of the cutaneous vascular system. He further states that he regards the affection as due in a predisposed individual to an excessive response on the part of the bloodvessels to an external irritation and the consequent pouring out of an enormous amount of serous exudation which penetrates the rete, carrying it away at its least resistant portion, thus forming the bulla. Engman and Mook, in their studies, found congenital absence of elastic tissue. In two well-marked examples of the disease they found elastic tissue absent or sparsely distributed in the papillary and subpapillary portions of the cutis. This absence of elastic tissue was discovered in regions other than those occupied by lesions. They suggested the possibility of the disease being caused by the hereditary, congenital, or acquired absence of elastic tissue. Kanoky and Sutton made similar observations, and more recently R. S. Weiss[1] has confirmed these observations. According to Wise in the acquired cases the elastic tissue does not show the changes that are present in the hereditary type.

Diagnosis.—In the major portions of the cases the recognition of the disorder is not difficult but in some rare instances it may be confused with pemphigus. Careful observation will soon demonstrate in the latter disorder the spontaneous appearance of bullæ, a circumstance which does not occur in the disease under consideration. The congenital character of the disease, with its location and history of irritation preceding the appearance of lesions, make a classical picture.

Treatment.—No treatment has any effect on the progress of the disorder. The individual lesions should be treated as indicated and everything possible should be done to protect the parts from external violence. Arsenic and other remedial agents have been employed without success. Prophylaxis is, therefore, the most important consideration.

Prognosis.—From what has been stated above it will be seen that the disease rarely disappears. In a few instances activity of the process ceases at the time of puberty and the vulnerability of the skin is overcome. Interference with work when the hands are markedly involved is also to be remembered, and in some instances permanent atrophic changes are seen.

PELLAGRA.[2]

Synonyms. — Lombardy erysipelas, Lombardy leprosy, Risipola lombarda, Lepra italica, La rosa, Mal rosso, Pellarella, Alpine scurvy, Dermatagra.

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[1] Jour. Cut. Dis., 1917, xxv, p. 26; "A case of epidermolysis bullosa showing loss of elastic tissue in an apparently normal skin."

[2] Bibliography: Gemma, Ann. univ. di Med., 1871, p. 564. Winternitz, Vierteljahr, 1876, iii, p. 151. Raymond, Annales, 1889, s. ii, x, p. 627. Lombroso, Die Lehre von der Pellagra, Berlin, 1898. Sandwith, Brit. Jour. Derm., 1898, x, p. 395, and Jour. Trop. Med., 1898, i, p. 63. Babes and Sion, "Pellagra," Nothnagel's Spec. Path. u. Therapie, xxiv, Pt. ii, fasc. iii, Vienna, 1901. Scheube, Diseases of Warm Countries,

Definition.—Pellagra (Lat., *pellis*, the skin; *aeger*, diseased) is a chronic, constitutional, endemic disorder, characterized by symptoms referable to the cutaneous, gastro-intestinal, and nervous systems, and having a tendency to seasonal recurrence.

At present, pellagra is most prevalent in northern and central Italy, southern Roumania, the Austrian Tyrol, southeastern Hungary, and the southeastern part of the United States; also in lower Egypt. In America a transient case was reported by Sherwell[1] in 1883. It is only since 1907 that it has assumed important proportions in this country. Among others who have made important contributions to the subject are Searcy, Babcock, Zeller, Lavinder, and the members of

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Philadelphia, 1903, p. 311. Manson, Tropical Diseases, 4th Ed., 1907, p. 328. Castellani and Chalmers, Manual of Tropical Medicine, 2d Ed. Sambon, Jour. Trop. Med. and Hygiene, 1910, pp. 271, 282, 305, and 319 (Progress report of the British Pellagra Commission). For further foreign and other references, history, and description of the disease, see Hyde, Amer. Jour. Med. Sc., January, 1910: "Pellagra and Some of its Problems."

Some important American literature: Searcy, Jour. Amer. Med. Assoc., 1907, xlix, p. 37: "An Epidemic of Acute Pellagra." Wood, ibid., 1908, liii, p. 274: "The Appearance of Pellagra in the United States." Watson, Jour. So. Carolina Med. Assoc., November, 1908: "The Etiology of Pellagra." Lavinder, reprint Public Health Reports, July, 1908: "Pellagra: a Precis;" ibid., xxiv, No. 43, October 20, 1909: "The Prophylaxis of Pellagra;" ibid., No. 40, September 10, 1909: "Notes on the Prognosis and Treatment of Pellagra." Lavinder, Williams and Babcock, ibid., xxiv, No. 25, June 18, 1909: "The Prevalence of Pellagra in the United States." Sandwith, Jour. So. Carolina Med. Assoc., November, 1909, v, No. 11: "Pellagra." Watson, ibid., November, 1909: "Symptomatology of Pellagra." Sambon, ibid., February, 1909: "Geographical Distribution and Etiology of Pellagra." Jour. Amer. Med. Assoc., 1909, liii, p. 1659: Trans. Conference on Pellagra, held at Columbia, S. C., November 3 and 4, 1909: Dyer, New York Med. Jour., 1909, p. 997: Howard Fox, New York Med. Record, February 5, 1910: Lavinder, reprint Public Health Reports, xxvi, No. 39, September 29, 1911: "The Salient Epidemiological Features of Pellagra." Report of the Pellagra Commission of the State of Illinois, November, 1911: Ormsby, Jour. Cut. Dis., 1912, xxx, p. 589: Lavinder, Amer. Practitioner, 1912, xlvi, p. 568: "The Diagnosis of Pellagra." Deeks, Med. Record, March 23, 1912, lxxxi, No. 12: "Pellagra in the Canal Zone."

The following references are from members of the Thompson-McFadden Pellagra Commission, New York Post Graduate Medical School and Hospital: Siler and Garrison, Amer. Jour. Med. Sc., July, 1913, p. 42: "An Intensive Study of the Epidemiology of Pellagra" (report of progress); and August, 1913, p. 238. Jour. Med. Assoc., 1914, lxiii, p. 1090; Archiv Int. Med., 1914, xiv, pp. 293 and 453; ibid., 1915, xx, p. 98; ibid., 1916, xvi, pp. 173 and 340. Jennings and King, Amer. Jour. Med. Sc., September, 1913, p. 411: A study of the possible carriers of the disease—ticks, lice, bed-bugs, cockroaches, horse-flies, fleas, mosquitoes, buffalo-gnats, house-flies, and stable-flies; and Jour. Amer. Med. Assoc., January 25, 1913, p. 271. Hillmann, Amer. Jour. Med. Sc., April, 1913, p. 507: "Some Hematological Findings in Pellagra." Myers and Fine, ibid., May, 1913, p. 705: "Metabolism in Pellagra." MacNeal, ibid., June, 1913, p. 801: "Observations on the Intestinal Bacteria in Pellagra." Also see Southern Med. Jour., March, 1912, for a series of articles by Albright, Jelke, Harris, Lavinder, Menage, Tuberville, Kimbrough, Leroy, Bass, Cole, Niles, Wolff, and Bronson. Nicolaidi, Southern Med. Jour., 1912, v, p. 465: "A New Treatment of Pellagra with the Organo-mineralized Radioactivated Serum of Dr. Jean Nicolaidi, Paris, France;" abstr. Jour. Cut. Dis., 1912, xxx, p. 756. See in addition text-books by Marie, translated by Lavinder and Babcock, 1910; Niles, 1912; Roberts, 1912; and Wood. Niles, Jour. Amer. Med. Assoc., 1914, lxii, p. 285: "The Treatment of Pellagra: An Optimistic Survey of its Present Status." Mackee, Jour. Cut. Dis., 1915, xxxiii, p. 365: A Case of Pellagra in New York; Wise and Lautman, ibid., p. 760; Pellagra in New York and its vicinity with report of two cases: Winfield, New York Med. Jour., 1916, ciii, p. 1076; Pellagra a Clinical Report on the State of New York.

[1] Jour. Cut. Dis., 1883, i, p. 142.

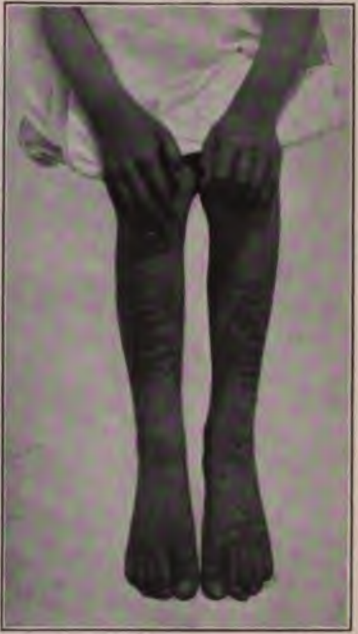
the Illinois State Pellagra Commission and the Thompson-McFadden Pellagra Commission.

Symptoms.—The earliest symptoms may be connected either with the skin or the gastro-intestinal tract. Occasionally, the patient suffers with what is presumed to be a sunburn and is indisposed for a short time, when the symptoms clear up, to return the next year or at some future date. In other cases, a moderately sore mouth, with some gastro-intestinal disturbance, particularly diarrhea, occurs, but is considered to be of no consequence, and in the course of a few weeks the symptoms subside, also to return at some future time. During the winter months the patients are very often entirely well. With each recurrence the symptoms are apt to be exaggerated, although at times they have been noted to be more mild.

A certain proportion of cases begin with gastro-intestinal symptoms, consisting of chronic and often severe diarrhea, with more or less stomatitis. These symptoms in certain cases have lasted for one or two months before the appearance of the eruption. In such cases the latter is usually exaggerated and of the bullous type. In other cases the disease runs a more chronic course, exhibiting mild symptoms. In still others the disease may be acute and severe (*Pellagra typhosis*), the patient having a high temperature, severe diarrhea and stomatitis, delirium, and other evidences of intense intoxication.

Seasonal recurrence has been emphasized by practically all observers. The disease has been noted to occur most often in the spring and autumn.

FIG. 133



Pellagra. (Siler, Garrison and MacNeal, Thompson-McFadden Pellagra Commission.)

In a certain proportion of cases, after an attack has cleared up in the spring a recurrence takes place in the autumn. Annual recurrences have been noted by many.[1] It appears, however, that in South Carolina no evidence was found by the Thompson-McFadden Commission pointing to a spring and autumn recrudescence or seasonal recurrence.

In a study of the cutaneous lesions, the changes are found to be those of an inflammation of greater or less severity and of shorter or longer duration, the lesions necessarily depending upon these factors.

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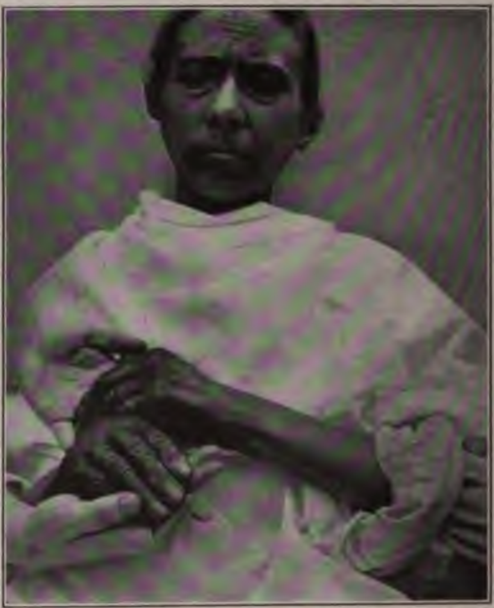
[1] Bacon, Jour. Amer. Med. Assoc., liv, p. 1783 (report of a patient who had seven recurrences in seven years).

In observing the development of these lesions, it is noted that at first there often occur large macular lesions, light- or dark-red in tint, which soon fuse, forming a patch of dermatitis almost identical in appearance with that caused by the sun. As the evolution of the disease advances, the color of the lesions deepens and they assume a reddish-brown or chocolate hue. In from seven to ten days or a little longer, desquamation begins, at which time a roughened, scaling surface is present. Early in the process there is moderately marked swelling. In the more active cases, on the erythematous base, there develop bullous lesions, which often attain a large size, and which after a few days gradually dry, leaving a thickened, crusted epidermis. Ecchymoses not infrequently complicate the process, and secondary pyogenic infection may follow in the vesicular and bullous cases. In severe cases ulceration may ensue; in many the edema is sufficient to produce fissures. Whether erythematous or bullous, the lesions are always well defined. After the eruption has disappeared, the skin in some cases is pigmented; in others the pigment is lessened. In certain cases, where the process is less acute, the appearance of a chronic dermatitis, with marked hyperpigmentation, is presented. After repeated attacks the skin becomes more or less permanently thickened, hard, roughened, scaly, and pigmented; it loses its elasticity, and its normal lines are exaggerated. The terminal

FIG. 134

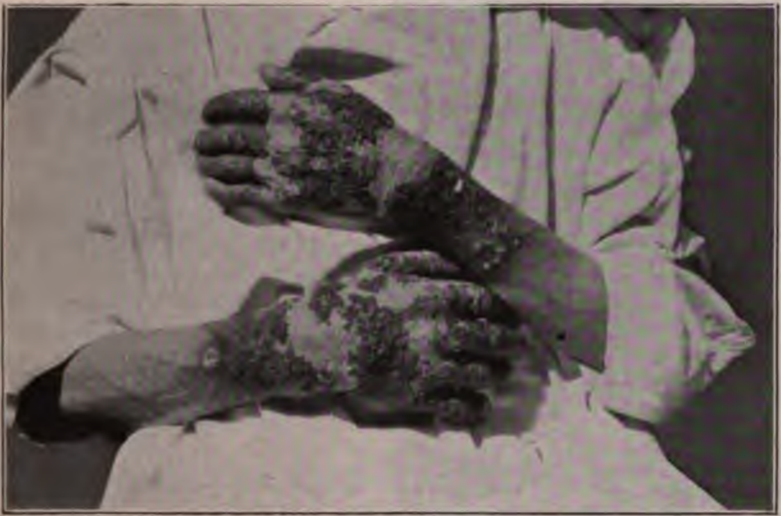


Pellagra. (Siler, Garrison and MacNeal, Thompson-McFadden Pellagra Commission.)

stage is exhibited as a thin, cicatriform, parchment-like integument, presenting striæ parallel with the long axis of the hand.

The lesions are found on the exposed areas of the body chiefly, and their arrangement is characteristic. In the major portion, the dorsa of the hands, the wrists, and some part of the face, neck, or scalp are involved, the feet and ankles more rarely here, but a common site in Europe. The arms and chest, and to a less extent the ears and other parts of the body, including the palms and genital region, are also involved. The symmetry of the lesions is striking and characteristic. On the hands often a solid area extends over the entire dorsal surface, involving the fingers, knuckles, and also the wrists on the extensor side for a distance of about two inches. In this area the lesion frequently sweeps around and involves about two-thirds of the flexor surface, then comes to an abrupt ending. This partial gauntlet has been frequently observed. The peculiar collar described by European observers is occasionally seen here, but is uncommon. That the rays of the sun are a factor in determining the localization of the lesions is accepted by all observers. In suspected cases at Bartonville fenestrated gloves were worn, and the erythema was largely limited to exposed areas. Subjective sensations are, as a rule, not marked, and are exhibited as burning rather than itching, the surface practically never showing evidence of scratching.

FIG. 135

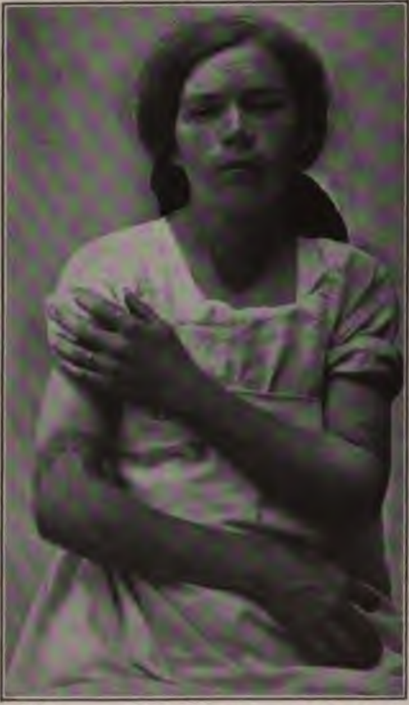


Pellagra. (Siler, Garrison and MacNeal, Thompson-McFadden Pellagra Commission.)

In the gastro-intestinal system diarrhea is a constant concomitant in severe cases; in mild cases this may be absent and constipation be present. The appearance of the tongue is important. This becomes swollen and denuded, presenting a dry appearance, with, in severe cases, more or less superficial ulceration along its edges and upon its under surface, with yellowish sloughings, which bleed easily. The buccal mucous membrane with which it comes in contact also shows in the more serious cases a similar appearance. A resemblance to the aphthous stomatitis seen in other debilitated states is noted. The ulcers are very superficial and heal without scar-formation. In mild cases the tongue is reddened and presents smooth areas, especially at the tip and along the margins, a condition to which the term "bald tongue" has been applied by Sandwith.

The nervous system chiefly presents the symptoms induced by acute intoxication. Singer suggests, in regard to the associative system of the brain, that there is a liability to the occurrence of deliria similar to those seen in other infective and toxic states. An important observation was independently made by Wilgus[1] and Singer,[2] who noted in the terminal stages of pellagra the symptom complex described by Meyer as that belonging to central neuritis. In an examination of pellagrous patients at Peoria, Bassoe[3] found changes in the reflexes suggesting the following: (1) probable pyramidal tract degeneration; (2) posterior column degeneration; and (3) combined degeneration.

FIG. 136



Pellagra. (Siler, Garrison and MacNeal, Thompson-McFadden Pellagra Commission.)

In the recurrences that take place from year to year the general symptoms become more marked. The gastro-intestinal are indicated by dyspepsia, pains in the abdomen, and dysentery. Pains are described as occurring in various parts of the body, particularly the head, epigastrium, and feet, with burning sensations in these same areas, especially at night. Tenderness on pressure over the spinal nerves; muscular weakness and loss of muscular power, particularly in the legs; tremors of the head and arms; mental worry and depression, anxiety and discontent, loss of memory, vertigo, and vague feelings of pressure, weight, or pulsation about the head, all may occur. In some cases the subjects are excitable and irritable, in others stupid and morose.

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[1] Report of the Pellagra Commission of the State of Illinois, November, 1911, p. 53.

[2] Ibid., p. 25.

[3] Hyde, loc. cit.

The knee-jerk, which is early exaggerated, becomes diminished and finally lost. Various paralyses are described, such as spastic paralysis, ptosis, hemianopsia, diplopia, amblyopia, and midriasis.[1] In the terminal stage, extreme weakness and emaciation, from the preceding severe gastro-intestinal disturbances, profound prostration, delirium, with involuntary evacuation of the bladder and bowels, close the scene. A marked feature of this state noted in the South[2] is opisthotonos, a condition in which the patient lies on the bed with the head forcibly extended, and the general musculature in a state of great rigidity.

The duration of the disease extends over periods varying from one to twenty years. Five years is probably an average.

Etiology.—In districts where the disease is endemic, it attacks persons of all ages. Castellani records large numbers of instances of its occurrence in children. Women are affected much more frequently than men, both in Europe and in America. As to occupation, in Europe the field-laborer suffers most. In America, nothing has been determined in this regard except that in the investigation by the Thompson-McFadden Commission a large proportion of cases were found among women employed in housework. The disease seems to be confined to the tropical and warm sections of the temperate zones; otherwise, climate appears to exert no influence in the etiology. All observers agree that even in pellagrous sections the disease is more or less restricted to certain localities. In Europe it is largely rural, occurring chiefly among the poor peasants of Italy and the poorer agricultural classes as a whole. A few cases occur in the cities. These facts have been borne out by American investigators of the European disease. In America, on the contrary, the small towns and mill villages in the Southern States suffer most. These observations, made by several southern physicians, are corroborated by the members of the Thompson-McFadden Commission.

In Europe, poor hygiene, poor food, and poverty are unquestionably important factors in the etiology. While these are factors in the United States, they appear not to be important. In the latter situation the disease not infrequently attacks people in the best walks of life, where poor hygiene or lack of proper food is out of the question. Nicolas and Jambon[3] believe the disease is the result of many circumstances, of which poverty and alcoholism are most constant.

Pellagra not infrequently attacks patients suffering with other disorders. Attention has been called by Singer, Babcock, and other observers to the susceptibility of the insane to the disease.

The various theories regarding the production of pellagra are those concerning maize or Indian corn and other agents. The corn theory has been that most strongly advanced in Italy. The views relative to this hypothesis may be summarized as follows: (1) Indian corn is deficient in or lacks some nutrient principle necessary for health, and pellagra results from a diet limited too strictly to that cereal. (2) Corn contains some toxic substance which in susceptible individuals produces the disease. (3) Corn undergoes some form of decomposition in the intestine of certain individuals, as the result of the growth of bacteria, the toxins thus produced exciting the disease. (The above theories refer to corn in its normal state.) (4) Healthy corn is innocuous, but at some stage in its preparation for consumption, either in the ear, when stored, or after being cooked, it undergoes decomposition, as the result of the growth of certain fungi. Various moulds and bacteria have been isolated and assigned an etiological role in the disease by different authors, such as *Penicillium glaucum*, different varieties of *Aspergillus*, *Sporisorium maydis*, *Ustilago maydis* (smut), *Bacterium maydis*, and *Bacillus pellagræ*. In the growth of these organisms it is supposed that toxins are produced which, when absorbed, induce pellagra. (5) Finally, that some one of the organisms above noted, which are commonly found in mouldy or spoiled corn and which may be eaten with it, directly invades the body and produces the disease. Reed[1] isolated a fungus, *Diplodia zeæ*, from "dry rot," a corn-stalk disease, which he thought might be an etiological factor.

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[1] Castellani and Chalmers, p. 1256.

[2] Hyde, loc. cit.

[3] Annales, July, 1908, p. 385, and August and September, 1908, p. 480.

In the report of the Illinois Pellagra Commission (p. 48), an account is given of two groups of patients, each consisting of sixty members, one of which was given a full corn diet, the other a corn-free diet, for one year. During this period the disease developed among the members of each group equally. Cutaneous anaphylaxis tests with extracts from damaged corn, and animal experimentation with damaged corn, including polenta imported from Italy, resulted in negative findings.

Certain investigators believe the etiological factor is a bacterium of unknown nature and habitat; others believe it to be due to some variety of ameba. The frequency of concomitant amebiasis in pellagra has been emphasized by Siler and Nichols[2] and Long.[3] Alessandrini, in Italy, found filarial infection in certain wells in pellagrous districts. Protozoal infection of the blood in much the same manner as in malaria and tryphanosomiasis has been suggested by others. These views are all based upon the supposed resemblance in the epidemiology, endemicity, and seasonal occurrence of these disorders. It has been thought that the infectious agent, if there be one, is transferred by a carrier. Sambon[4] suggested a species of simulium (the sand-fly or buffalo-gnat). Jennings and King believe that if it is distributed by a blood-sucking insect, *Stomoxys calcitrans* would appear to be the most probable carrier.

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[1] New York Med. Record, January 22, 1910.

[2] Ibid., January 15, 1910.

[3] Jour. Amer. Med. Assoc., 1910, lv, p. 734.

[4] Brit. Med. Jour., 1905, xi, p. 1272.

The disease is regarded by most observers as not contagious. Innumerable examples might be cited to prove this theory. It is said that at the Pellagrosario at Mogliano, Veneto, Italy, where large numbers of pellagrins have been treated for many years, no attendant or nurse has ever been known to develop the disease. This sanitarium accommodates from 400 to 600 patients and has from 60 to 70 employés (Lavinder). In the early history of the disease in America, it appeared most extensively in institutions, particularly in insane asylums. In the investigation of cases by the Thompson-McFadden Commission, in one-half of these a single member of a family was affected; in about one-fourth two members of a family were affected; and in the remainder groups of three, four or five members suffered from the disease. On the other hand, King[1] recorded a group of twelve cases which appeared to have followed the development of the disease in a single case in one family.

Jobling and Peterson[2] state that pellagra presents all the evidences of being in some way conveyed from one patient to another; that it is a family disease or a disease of the house next door.

Attempts at transmission of the disease have usually been unsuccessful. Harris[3] was successful in producing in monkeys symptoms resembling human pellagra, employing subcutaneous, intravenous, and intracranial injections of a Berkefeld filtrate prepared from human pellagrous tissues.

Recently a theory has been advanced by Alessandrini and Scala[4] that silica in collodial solution in potable waters is an etiological factor in pellagra. The dietary origin, exclusive of corn, has been emphasized by Goldberger and Myers and Voegtlin.[5] In the intensive work done on the disease during the past few years more stress has been laid upon an unbalanced diet as being the chief etiological factor. Goldenberger, Waring and Willets[6] conclude that the pellagra-producing diet is (*a*) physiologically defective protein supply; (*b*) inadequacy of water soluble vitamines; (*c*) inadequacy of fat-soluble vitamines; (*d*) defective mineral supply.

Pathology.—The histological changes noted in the skin vary according to the stage in which the examination is made. In a series of cases in the early stages, we found the most marked changes in the superficial part of the corium, consisting of a cellular infiltration, particularly in the region of the bloodvessels, and moderate edema of the collagen. In the epidermis hyperkeratosis was the rule, with here and there areas of parakeratosis, evidenced by the presence of nuclei in the stratum corneum. Increased pigment was noted; otherwise the rete showed no changes except where its integrity was interfered with by the infiltrating cells beneath. No microörganisms were discovered. The process appears to be a reaction on the part of the skin to a local toxic agent, or an angioneurotic process influenced from a distant focus. Corlett and Schultz[1] found in a case in which repeated attacks of dermatitis had occurred evidence of atrophy of the corium and epidermis, with changes in the nerves of the corium of such pronounced nature that they concluded these were primary and that the other findings were secondary. The nerves exhibited evidence of a combined inflammatory and degenerative process, with disappearance of many axis-cylinders.

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[1] Southern Med. Jour., November, 1908.

[2] Jour. Inf. Dis., 1917, xxi, p. 109.

[3] Jour. Amer. Med. Assoc., June 21, 1913.

[4] Jour Amer. Med. Assn., 1914, lxiii, p. 868 (editorial with references).

[5] Ibid., 1918, lxxi, p. 944.

[6] Ibid., p. 1114 (editorial with references).

The most important post-mortem findings have been those connected with the gastro-intestinal tract, chiefly the intestines and liver, and of the central nervous system. In the former, ulceration of the colon was not infrequent, and in the liver changes were noted by Singer which suggested to him the elaboration of a toxin in the intestine, which enters the circulation by way of the portal stream, and induces the liver changes. Singer[2] states that "the nervous system presents a picture of axonal chromatolysis involving especially the Betz and larger pyramidal cells of the precentral convolutions and the cells of the nuclei in the cerebellum, pons, medulla, and cord, as well as the post-root and sympathetic ganglia. Besides these changes numerous cells in most cases show a marked pigmentary degeneration of fatty nature similar to that found in the senile nervous system and in some other conditions. With this there is in most cases but little evidence of connective-tissue reaction, and we would especially emphasize the absence of infiltration of the perivascular sheaths. In some cases there is more or less over-growth of glia cells along the vessels and around the nerve-cells. The picture here described is identical with that published by others, notably Spiller and Anderson,[3] in cases of pellagra, but is also strikingly similar to the picture of central neuritis." Corlett and Schultz[4] found structural changes in the central nervous system involving the nerve-cells and fibers, with marked changes in the chromatin of the ganglionic cells.

Various inflammatory, degenerative, pigmentary, atrophic, and sclerotic changes are described in connection with the viscera and larger bloodvessels.

Marked indicanuria has been noted by Singer,[5] Myers and Fine,[6] and others, indicating unusual intestinal putrefactive changes.

Diagnosis.—Recalling the classical symptoms of the disease, which include a well-defined, symmetrically placed dermatitis on the exposed portions of the body, and stomatitis of varying grades, associated with gastro-intestinal disturbance, chiefly exhibited as diarrhea, little difficulty will be encountered in recognizing the disorder. At a later date the nervous phenomena, including irritability, depression, melancholia, etc., make the picture still more distinct.

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[1] Jour. Cut. Dis., 1911, xxix, p. 193.

[2] Loc. cit.

[3] Amer. Jour. Med. Sc., New Series 141, January-June, 1911, p. 94.

[4] Loc. cit.

[5] Loc. cit.

[6] Amer. Jour. Med. Sc., 1913, cxlv, p. 705.

Eczema and erythema multiforme have to be excluded. In certain advanced cases symptoms of general paresis are presented, which, in the absence of cutaneous manifestations, might cause confusion; but with a history of the latter having been present, with associated gastro-intestinal disturbance, and with laboratory methods, including the complement-fixation test for syphilis, the disorders may be differentiated. A patient suffering with an aggravated but typical attack of pellagra, who developed upon the subsidence of the pellagrous symptoms general paresis of luetic origin, has been studied by the author.

Treatment.—The general treatment, with special attention to improvement of nutrition, is of the highest importance. In Italy, where a mixed diet has been substituted for the exclusive use of polenta, marked improvement has followed. A liberal mixed diet is recommended by Goldberger with an abundance of fresh meat, milk, eggs, peas, beans and other fresh vegetables.

Internal medication has been of little value. Chief among the remedies employed are arsenic, iron, quinin, and other tonics. Atoxyl has been tried without much success, while salvarsan[1] has been used with indifferent results.

Direct transfusion of blood has been of service in a number of cases.[2] Hydrotherapy is recommended by Niles.[3]

Local treatment consists in soothing and mild keratolytic applications in the less severe cases, and soothing and antiseptic lotions in the severe vesicular and bullous varieties.

Prognosis.—In the early cases occurring in Illinois, the mortality was high, but recently the figures have fallen greatly. In 1910 the mortality in American patients was about 35 per cent. Bass,[4] in 1912, gave 5 per cent. as the figure of mortality in New Orleans. Recovery rarely takes place in the more severe grades. In the earlier and mild forms the prognosis is better.

ACRODYNIA.[5]

Synonyms.—Erythema epidemicum; Cheiropodalgia. Fr., *Acrodynie.*

Acrodynia is a disease said to be closely allied to pellagra and ergotism. It first appeared in Paris, in the year 1828, and has since been recognized in epidemic form in France, Belgium, Algiers, Mexico, and a few other countries.

Definition.—The disorder is characterized by an erythema situated chiefly on the palms of the hands and soles of the feet, and by general symptoms referable to the nervous system, exhibited in various parts of the body.

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[1] Nice, McLester and Torrance, Jour. Amer. Med. Assoc., lvi, No. 12 (improvement recorded in three cases); Cranston, ibid., lviii, p. 1509 (eleven patients treated, results not encouraging).

[2] Cole, Southern Med. Jour., 1912, v, p. 167 (review of 31 cases).

[3] Amer. Jour. Med. Sci., 1913, clvi, p. 230.

[4] Southern Med. Jour., March, 1912.

[5] Vidal et Leloir, Traité des Maladies de la Peau, Art. "Acrodynie." Hirsch, Sydenham Society, 1830, ii, p. 217 (quoted by Crocker).

Symptoms.—The lesions on the palms and soles in the beginning are described as erythematous spots, accompanied by swelling, ecchymoses, cyanosis, and occasionally even partial gangrene (Leloir). Vesicular and bullous lesions may be present. According to Alibert (quoted by Crocker), the erythema may be preceded by the bullæ, and the eruption may spread over the limbs and portions of the trunk, being followed by exfoliation, particularly over the palmar and plantar surfaces, where the desquamation may occur as large, horny flakes and casts, similar to those seen in other exfoliating dermatoses; while on the trunk dark-brown or blackish pigmentation occurs. One of the most characteristic features of the disorder is the blackish hue of the skin over the breast, abdomen, flanks, chest, axillæ, and inguinal regions. The subjective sensations in the affected areas are numbness, prickling, tingling, and also lancinating pains, accompanied by marked hyperesthesia and a sensation of intolerable heat. Later on these are followed by anesthesia. In severe cases the limbs become edematous, and there may be cramps, pareses, and toxic symptoms. Associated symptoms are gastro-intestinal irritation, with edema of the face and redness of the conjunctiva.

The disease lasts for from two to four weeks, but relapses are frequent. The whole process terminates, as a rule, in the course of two or three months. It seldom proves fatal except in the case of senile or debilitated subjects. The pathology of the disease is obscure, and the treatment is symptomatic, being directed to meet the special symptoms as they arise.

Under the title acrodynia Weston[1] recorded a group of 8 cases observed by W. T. Patrick (Portland) presenting an unusual symptom-complex. A group of 15 cases observed by Byfield[2] presenting similar symptoms has also recently been recorded. The disorder occurred in young children and all suffered with symptoms referable to the nervous system and the skin together with less marked involvement of the digestive and respiratory tracts.

The hands and feet were cold, cyanotic and presented erythematous areas followed by scaling. Marked subjective symptoms were present causing the patient to rub, scratch and often "chew" these members. Hyperesthesia and pain was frequently noted. On other portions of the cutaneous surface a morbilliform or bullous eruption is described. Among other symptoms are described alopecia due to pulling the hair, falling of teeth, and changes in the nails. Intense sweating was noted continuously. The patients were at times dull and apathetic, or restless and occasionally delirious. Anorexia and thirst occurred and emaciation followed. Photophobia was noted in several. In a child aged four years seen by the author the disorder followed an attack of influenza. In this the hands and feet were edematous, erythematous and scale covered and the seat of intense subjective symptoms causing the child to rub and scratch day and night. In addition there were at times minute vesicles on the hands as well as over the trunk. Erythematous areas and papules also occurred on the trunk and superficial excoriations were noted on the mucous membrane of the mouth. Several teeth dropped out. General sweating occurred and intense nervousness was present with occasional attacks of delirium.

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[1] Archiv of Pediatrics, 1920, xxxvii, p. 513. Acrodynia. Paper read before the Section on Pediatrics of the Amer. Med. Association in 1920.

[2] Amer. Jour. Dis. Children, 1920, xx, p. 347. A polyneuritic syndrome resembling pellagra—acrodynia(?) seen in very young children. This author previously recorded the same disorder under a different title in 1917, Jour. Amer. Med. Assoc., 1917, lxviii, p. 1851.

Byfield suggests that this disorder is a post-influenzal radiculitis or sensory polyneuritis.

Treatment.—Proper feeding, forced if necessary, is recommended. The various soothing local applications gave but little relief.

EXANTHEMATA.

Under this heading an important group of diseases is considered, each of which is accompanied by a characteristic cutaneous eruption. On account of their contagious nature they present an important problem of public health. One attack commonly confers immunity, though exceptions occur. Included in the group are scarlatina, rubeola, rötheln, variola and varicella.

Scarlatina (*Scarlet fever, Scarlet rash, Canker rash.* Fr., *Scarlatine;* Ger., *Scharlach*).

Definition.—Scarlet fever is an acute, infectious and extremely contagious disease characterized by angina, a punctiform cutaneous eruption and constitutional symptoms.

Symptoms.—The period of incubation of scarlet fever is rather short, from twenty-four hours to several days. During this time there may be no disturbance of the health, or a mild indisposition may be noted. The onset of the disorder is abrupt. The attack begins with vomiting, headache and sore-throat, and in children a convulsion frequently occurs in the early stage. The temperature rises rapidly to 102° or 105° F., the pulse is characteristically rapid and bounding, and ranges from 120 to 160 per minute.

Examination of the mouth at this time shows the tongue to be covered with a yellowish-white coat through which the red, fungiform papillæ protrude. The tonsils, uvula and soft palate are congested and swollen, and a red, punctate eruption may be seen on the hard and soft palate. Examination of the maxillary, the posterior cervical and inguinal glands at this time may reveal enlargement. In the course of twenty-four hours the characteristic eruption appears, the general symptoms continuing without abatement after the appearance of the eruption. The latter begins about the neck and clavicular region, spreading rapidly to the face and downward over the trunk and extremities, attaining its complete development in the course of two to four days. The eruption, which appears as a uniform reddened condition of the skin, is made up of innumerable closely set red points, between which the color is less marked. Upon the extremities the eruption may be limited to the punctate lesions above mentioned, but the diffuse redness is usually well seen over the trunk. Occasionally around the punctate spots the erythema is slightly marked, which by contrast gives a patchy appearance somewhat resembling measles. The scarlatina eruption is not infrequently accompanied by small, acuminate papules about hair follicles. These occur chiefly on the extremities, particularly the lower, and are present to a less degree upon the trunk. Miliary vesicles, disseminated or grouped, are often seen, appearing principally over the abdomen and chest and to a lesser degree over the limbs.[1] The face presents a characteristic appearance in that the eruption avoids the area about the mouth. The cheeks are flushed and a few lesions are commonly found on the temples and forehead, thus producing a marked contrast to the pale region surrounding the mouth. The color of the eruption disappears under pressure and after temporary return of the redness a white area remains for a short time. When the fingernail is drawn rapidly over the surface a whitish-yellow line is produced which may persist for several minutes.

After attaining the height of its development the eruption remains for a few hours to a day or two and then begins to fade, first upon the areas earliest attacked. When the eruption is fully developed the tongue presents a characteristic appearance. As previously stated, it is early covered with a grayish fur, through which fungiform papillæ protrude. Shortly afterward the tip and sides become markedly reddened, and later the coat is thrown off, leaving a red, raw or glazed surface, upon which well-defined papillary elevations protrude. These are the enlarged fungiform papillæ and they may be few or numerous. This condition represents the so-called "strawberry tongue" and is characteristic of the disease.

With the decline of the eruption the general symptoms diminish in severity and desquamation begins. This is more pronounced than in any of the eruptive fevers. It may be superficial and furfuraceous in character, or the epidermis may fall in lamellated layers. For example, the sheath of an entire finger or of the palm may be shed. In this way sheets, ribbons, and shreds of the superficial portion of the epidermis fall and expose a new and often tender epidermis beneath. The hairs are rarely shed. The entire period lasts, in uncomplicated cases, from two to six weeks. In more severe cases the mucous membranes may early show fingernail- to pigeon egg-sized grayish ulcers with a dark red halo at the periphery. These may soon be followed by marked adenopathy and this in turn, in serious cases, by abscess formation. As the result of ulceration much destruction may occur in the throat. These cases frequently prove rapidly fatal and have been termed septic (*angiose*) scarlatina.

In the so-called toxic scarlatina (*scarlatiniform typhus;* Fr., *Scarla-*

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[1] Schamberg, Diseases of the Skin and Eruptive Fevers, p. 461.

tine foudroyante), which is a severe type of the disease, the symptoms resemble typhus and the patient may succumb within a few hours after the attack, before the appearance of the eruption. A profound intoxication is exerted upon the nervous system and results in convulsive symptoms, or the patient becomes comatose. The eruption may fail to appear or be poorly developed, and is often deep red and hemorrhagic or petechial in type. Albuminuria, meningitis, severe diarrhea and coma may follow at a later date, followed by fatal termination.

The anomalies and complications of scarlet fever are numerous. The incubation and prodromal stages may be brief as to time or apparently merged by the rapid appearance of the eruption. The latter may occur first upon the extremities and later on the trunk and neck, or may develop simultaneously in a generalized distribution, or be mild, of short duration, or absent altogether, or it may be unusually prolonged. The period of desquamation may appear early or be postponed, or may be prolonged and of an intense type. Among the complications catarrhal and parenchymatous nephritis are the most dangerous and may prove fatal after a relatively benign manifestation of the disease. A moderate albuminuria may be present early in the course of the disease without inducing serious consequences. When developing during the second or third week and accompanied by swelling and edema of the face and extremities with tubular casts in the urine it becomes more serious. Occasionally an acute hemorrhagic nephritis occurs which is rapidly fatal. Other complications are gastro-intestinal disorders, otitis externa, media or interna, pneumonia, pericarditis and pleurisy. Occasionally destruction of the cornea is seen as the result of severe keratitis. Otitis media may lead to mastoid inflammation and thus to meningitis. All observers agree that many of the complications arising in or following scarlet fever are due to streptococcal infection in the throats of these patients.

Etiology and Pathology.—The disease is transmitted either directly or through articles which convey the infective material. Epidemics have followed the use of contaminated milk. The disease occurs at all ages and in both sexes, but is more common in children. Its prevalence in childhood and the immunity thus induced probably accounts for its less frequent appearance in adults. The parasitic cause has not been identified. Streptococci, bacilli, protozoan-like bodies and peculiar inclusion bodies have been observed and frequently reported. These have been obtained from various secretions and organs, and in some instances a disorder resembling scarlet fever has been induced in lower animals by inoculation. Many observers believe the streptococci commonly found are responsible for some of the complications accompanying or following the disease. Klein, Baginsky,[1] Class[2] and Weaver[3] have studied and reported upon the streptococci. Mallory described protozoan-like bodies found in the skin, and his findings were confirmed by Duval.[1] Döhle[2] described peculiar inclusion bodies present in leukocytes. The conclusions arrived at by several observers[3] are that in all cases of scarlet fever, excluding the malignant, fatal, toxic type, these bodies are present during the first four days, but absent after the eighth day. As they are also found in other conditions, their significance is not determined.

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[1] Berlin. klin. Wchnschr., 1900, xxxvii, pp. 588 and 618; Lancet, 1900, ii, p. 1234.

[2] Med. Record, 1899, lvi, pp. 330 and 513; Jour. Amer. Med. Assoc., 1900, xxxv, p. 799.

[3] Jour. Med. Research, 1903, ix, p. 246.

Vipond[4] described a bacillus recovered from the lymphatic glands which was capable of producing the disease in higher apes. Pryor and Kelly[5] reported the discovery of a diphtheroid organism in the blood; also a polymorphous organism which gave a positive cutaneous reaction in convalescent scarlet fever patients when prepared after the method of Gay for typhoidin. A similar organism isolated from the throat in scarlet fever patients by Cantacuzene[6] induced a condition simulating scarlet fever in monkeys. Landsteiner and Levaditi[7] successfully transmitted the disease to higher apes but were unable to determine the nature of the virus. Draper and Hanford,[8] on the other hand, obtained negative results on their attempt at transmission of the disease to monkeys.

The eruption is due to hyperemia caused by marked dilatation of the vessels of the corium. Unna found parakeratosis and scaling, with no evidence of edema in the prickle cell layer, nor of emigration of leukocytes. The corium showed marked dilatation of bloodvessels, with little or no cellular infiltration.

Diagnosis.—The diagnosis of scarlet fever in typical cases is readily made, the characteristic symptoms being the sore-throat, intense fever, punctiform scarlet eruption reaching the border of the inferior maxillæ, the distinct whitish-yellow line traceable by the fingernail when drawn rapidly over the surface, the presence of leukocytosis and the glandular involvement of the inguinal and cervical regions. In measles the macular character of the eruption, its blotchy appearance and often crescentic arrangement in connection with the catarrhal symptoms will usually make the distinction.

From erysipelas it may be distinguished by the absence of the peculiar shining, smooth or glazed and tumid condition of the affected area. It is distinguished from erythema scarlatiniforme by the absence in the latter of glandular involvement, irregular appearance of the eruption, absence of the strawberry tongue and throat symptoms.

[1] Virchow's Archiv, 1905, clxxix, p. 485.

[2] Centralbl. f. Bac. u. Parasit., 1911, lxi.

[3] Kretschmer, Berlin. klin. Wchnschr., 1912, xlix, p. 499; Nicoll and Williams, Archives of Pediatrics, 1912, xxix, p. 350; and Granger and Pole, Brit. Jour. of Children's Dis., 1913, x, p. 1.

[4] Archives of Pediatrics, 1911, xxviii, p. 564.

[5] Jour. Laboratory and Clinical Med., 1918, iii, pp. 267 and 525.

[6] Compt. rend. Acad. de Med., clix, p. 58.

[7] Abstr. in the Jahrb. d. Kinderheilk., 1912, lxxvi, p. 218.

[8] Jour. Exper. Med., 1913, xxii, p. 499.

From all other toxic eruptions, scarlet fever can be differentiated by the constitutional symptoms and resulting desquamation. Drug eruptions producing a scarlatiniform appearance should be remembered. Belladonna has caused scarlatiniform eruptions in children indistinguishable from that occurring in the disease; quinin and some other drugs produce similar eruptions, but all of these resemble scarlet fever only in the eruptive manifestations.

Koessler[1] in a study of the complement-fixation test in scarlet fever, using an extract of lymph nodes as an antigen, obtained positive reactions in 68.6 per cent. of 118 cases examined. Laederlich and Bory[2] employing a cholesterolized antigen, obtained absolutely positive reactions in 16 out of 20, three partially positive and one negative on the first to the fourth day of the disease.

Leede[3] found that by placing a constricting band above the elbows of scarlet fever patients petechial hemorrhages were produced below the constriction. This phenomenon occurred on all but 1 of 200 cases of scarlet fever, and was absent in other diseases, except to a modified degree in measles. It occurs in from five to twenty minutes after the constrictor is applied.

Treatment.—The treatment of uncomplicated scarlet fever consists in the proper regulation of the food, local baths and inunctions, isolation of the patient with complete rest, and an abundant supply of fresh air. To relieve the local irritation, as well as to confine the scaling, inunctions with cold cream salve, almond or olive oil, or vaseline are of service. Oil of eucalyptus can be added and is advocated by Milne[4] to be used as an application over the entire surface of the skin. This application is said to lessen the spreading of the disease. Weaver[5] reported favorable results with the use of serum of convalescent patients. In nineteen cases treated the temperature was reduced, the toxic condition improved and the course of the disease shortened. The other treatment used in scarlet fever is practically limited to the management of complications, which should be treated according to indications.

Walters[6] recorded the successful use of a streptococcic vaccine as a prophylactic measure.

Prognosis.—The prognosis of this disease should always be given with reserve. It is based upon the intensity of the symptoms, the age and general vigor of the subject and the presence or absence of serious complications. Albuminuria is often present but is usually not alarming; anasarca and other evidences of serious interference with renal function are to be considered important. In general, a high temperature, early ulcerative throat lesions, the puerperal state, anomalous associated eruptions, particularly those producing hemorrhagic extravasations, are all dangerous symptoms.

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[1] Jour. Amer. Med. Assoc., 1912, p. 1529.

[2] Bull. et Mem. Soc. Med. d. Hôp. d. Paris, 1919, xliii, p. 504.

[3] München. med. Wchnschr., 1911, lviii, p. 289; abstr. Jour. Amer. Med. Assoc., 1911, lvi, p. 858.

[4] Brit. Med. Jour., October 31, 1908.

[5] Jour. Infec. Dis., 1918, xxii, p. 211; pertinent references.

[6] Jour. Amer. Med. Assoc., February 24, 1912, lviii, p. 546.

Rubeola.—**Synonyms.**—Measles, Morbilli; Ger., *Masern, Flecken;* Fr., *Rougeole;* Ital., *Rosolia;* Sp., *Serampion.*

Definition.—Measles is an acute infectious and contagious disease, characterized by catarrhal symptoms in the upper air passages and a cutaneous exanthem associated with constitutional symptoms of varying grade.

Symptoms.—The period of incubation varies, the major portion of the cases developing from the tenth to the fourteenth day. The earliest symptoms are those exhibited in an ordinary cold. These may be present for a day or two, but ordinarily the disease is ushered in suddenly. There is elevation of temperature, sneezing, a harsh, dry cough, lacrimation, and photophobia. Convulsions occasionally occur in young children and chilly sensations may be present. The temperature on the evening of the first day ranges from 101° to 104° F. The mucous membranes of the mouth and throat become hyperemic and frequently show a punctiform eruption, and the tongue is furred. During this stage the general symptoms continue and the mucous membrane of the mouth shows characteristic lesions. These are described as "Koplik's spots," which are white or bluish-white, fine plaques or points situated upon a reddened base. These occur chiefly along the line of the molar teeth on the buccal mucous membrane. They are practically always present and are considered diagnostic.

Eruptive Stage.—On the fourth day eruptive manifestations occur in the skin and may be first noted either upon the neck or face. According to Welch, the area on the neck back of the angle of the jaw is the first point attacked. From the face and neck the eruption rapidly spreads over the trunk and extremities. It is usually well-marked on the face on the second day and at its height on the third or fourth day on the trunk and extremities, at which time it begins to fade on the face, disappearing in the same order that it develops. The eruption begins as small red macules, which are slightly elevated, a condition which sometimes is more perceptible to touch than to sight. The macules enlarge and rapidly increase in number until the entire surface is more or less completely covered. When the eruption is fully developed the face presents a swollen appearance and the lesions may be confluent, while on the trunk areas of normal skin remain, giving a peculiar blotchy appearance, or the lesions may be arranged in concentric form. Difference of opinion exists as to the value of the last named configuration in measles. The cervical glands may be moderately enlarged and tender. The elevation of temperature and catarrhal symptoms continue and are usually increased with the appearance of the eruption, but on the third or fourth day, when the eruption is fading, the temperature falls and the other symptoms subside. As the eruption fades a yellowish-brown discoloration remains and a furfuraceous scaling may follow, which persists for a week or ten days. Many variations occur in this disease. The time of invasion may be shortened or prolonged and the eruption may be scanty and localized or profuse and generalized. In confluent cases a high temperature and severe general symptoms are present. Very rarely the eruption reappears after having subsided. Occasionally in ordinary cases some lesions may be dark colored from a moderate amount of hemorrhage. In rare instances all the lesions may become hemorrhagic, in which case hemorrhages also occur from the nose and other mucous membranes. The constitutional symptoms in this type are severe and there is not infrequently a fatal termination.

The complications[1] of measles are numerous and varied, the most serious being bronchopneumonia. During the late war many fatalities occurred in the various camps through this complication. In some instances the catarrhal condition is so severe that membranous croup develops.

Other general complications are keratitis, otitis media, and, rarely, nephritis. In some instances pulmonary tuberculosis has followed and whooping-cough, scarlet fever and other exanthemata may occur.[2]

Cutaneous complications are exhibited as local pus infections, such as impetigo, furunculosis, abscess, and, rarely, gangrene. The latter (*noma, cancrum oris*) may attack the mucous membrane of the mouth and prove formidable. Multiple disseminated lupus vulgaris and other forms of cutaneous tuberculosis occasionally follow measles. Erythema nodosum[3] has been noted by several independent observers.

Etiology.—The disease is infectious and contagious, being directly communicable from person to person and also by fomites. The causative microörganism, which has not been positively identified, apparently is of low resistance, as the transmission of this disease by clothing and other objects occurs less frequently than is the case with the other exanthemata. The disease is contagious in all stages of development, including the pre-eruptive stage. The infection seems to occur in the dried secretions from the nose and mouth. It is essentially a disease of childhood although any unprotected person may contract it. Serious epidemics have occurred among all ages in islands where the disease had not previously existed. Infants under three months of age are rarely attacked and it is infrequently seen in advanced years, probably owing to previous immunity. Second attacks are reported, but these are rare.

The infective agent is present in the blood, as shown by inoculation experiments. Hektoen[4] successfully reproduced the disease in two instances, using blood from a measles patient taken on the first day of the eruption, in one case, and in the second thirty hours after the first appearance of the eruption. Anderson and Goldberger[5] have been successful in producing the disease in monkeys by inoculating them with blood taken during the prodromal stage. Hektoen[1] concludes, from results of human experiments, that the cause of measles is present in the nasal secretions, scrapings of the skin (epithelial débris and blood) and the blood during the earlier part of the eruptive stage, and that the disease may be transmitted to animals, the monkey being the only one susceptible. The results in monkeys show the causative factor of measles present in the nasopharyngeal secretions and the blood at least twenty-four hours before the eruption, as well as a day or two afterward.

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[1] Craster, Amer. Jour. Dis. of Cnildren, August, 1913, vi, p. 65; an analytical study of 1000 cases of epidemic measles relative to complications, age, and incidence.

[2] Williams, Brit. Med. Jour., 1901, ii, p. 1797; "Rubeola, scarlatina and fourth disease."

[3] Joynt, Brit. Med. Jour., April 15, 1911; abstr. Jour. Cut. Dis., 1911, xxix, p. 513.

[4] Jour. Infec. Dis., 1905, ii, p. 238.

[5] Jour. Amer. Med. Assoc., 1911, lvi ,p. 113, lvii, pp. 476, 971 and 1512.

Tunnicliff and Brown[2] isolated a Gram-negative diplococcus from the blood, nose and throat secretion, ears and sputum in cases of measles, which may be concerned in the production of the disorder. Hektoen[3] concludes that the chief bacteria concerned in measles are the diplococcus isolated by Tunnicliff, the influenza bacillus and hemolytic streptococci. The last two are important in the pneumonias that frequently follow.

Diagnosis.—Measles must be differentiated chiefly from scarlet fever and German measles. For the distinction of the former see that disease. From German measles it is distinguished by the presence of Koplik's spots, the more marked catarrhal and general symptoms and the character of the eruption. The latter disease is mild, occurs in epidemic form, appears suddenly, often without prodromal symptoms, is transitory, accompanied by mild general symptoms, and has associated glandular involvement in the neck, which is important. Measles is readily distinguished from the various forms of erythema as well as from dermatitis medicamentosa, insect bites, and syphilitic eruption by the specific characteristics of these eruptions and the absence of fever. Serum eruptions sometimes closely resemble measles, but in the former the history of the use of serums and the presence of joint pains are important.

Treatment.—The treatment of measles should be limited to careful hygiene, restricted diet, isolation of the patient, proper ventilation of the apartment and such medical treatment as is individually indicated. A bland oily or fatty substance may be used to relieve the itching. Eucalyptus[4] used as a local application on the skin and also as a spray for the nasal passages has been found of value. The eyes should be protected and, as a rule, it is best to keep the patient in a darkened room.

Prognosis.—The prognosis is generally favorable, but with certain complications becomes grave in the highest degree; the one most to be feared is bronchopneumonia. All the complications above named increase the gravity of the disease, and it also becomes more serious among men crowded together in camps, military and civil, children in public charities, in pregnant women, in the cachectic and those enfeebled from other disease, in infants and old people, and in residents of islands not previously infected.

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[1] Jour. Amer. Med. Assoc., 1919, lxxii, p. 177; "Experimental measles." A review of past work with discussion of the subject to date.

[2] Jour. Inf. Dis., xxiii, pp. 472-572; idem., ibid., 1919, xxiv, p. 76; idem., Jour. Amer. Med. Assoc., 1917, lxviii, p. 1068.

[3] Jour. Amer. Med. Assoc., 1919, lxxi, p. 1201; discussion of the bacteriology of measles, with extensive bibliography.

[4] Elgart, Med. Klinik, Berlin, August, 1913, ix, No. 31, pp. 1238 and 1276; abstr. Jour. Amer. Med. Assoc., September 13, 1913, lvii.

Rötheln.—**Synonyms.**—Rubeola, Rubeola notha, Rubella, German measles, Hybrid or Bastard measles, French measles, Epidemic roseola; Fr., *Rubéole;* Ger., *Rötheln.*

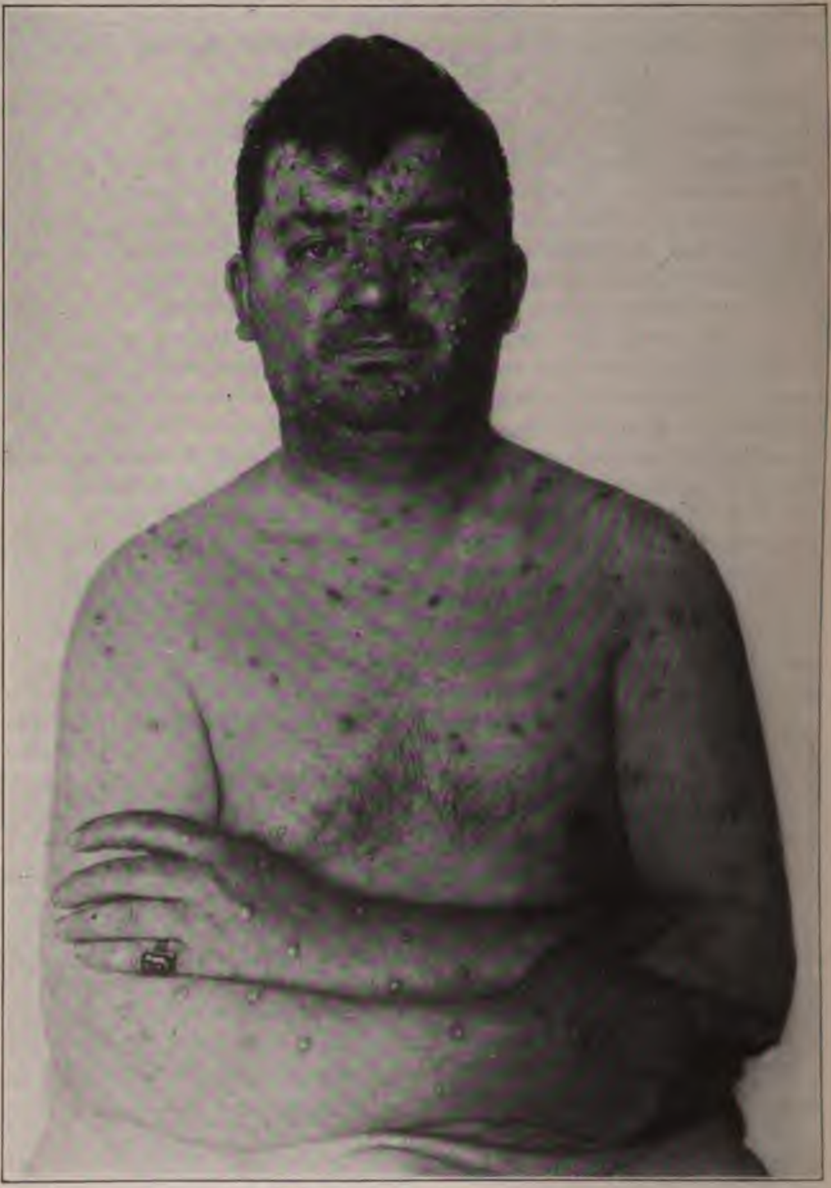
Definition.—Rötheln is an acute, contagious disorder characterized by a macular eruption and mild constitutional symptoms.

Symptoms.—The time of incubation is variable and usually extends over a period of one to three weeks. Mild prodromal symptoms may precede the eruption by a few hours or a day, and consist of malaise, headache and moderate elevation of temperature. With or without these symptoms the eruption appears. It may begin on any portion of the cutaneous surface, but usually the face and scalp are the sites earliest affected. The eruption spreads rapidly and in twenty-four to forty-eight hours has reached its height, after which it begins to recede. It not infrequently is receding in areas first involved as it is appearing in new locations. The lesions are pinhead- to lentil-sized, round or oval, pinkish or rosy-red, smooth or slightly elevated macules. The color is less vivid than that of scarlet fever and lacks the blue or violaceous tinge seen in measles. The lesions are usually discrete and have no characteristic arrangement, but may be grouped on the face or on areas of pressure, such as the buttocks and posterior portion of the thighs. The eruption may be abundant or limited in distribution. Usually it is most marked on the face and neck, the trunk and extremities exhibiting fewer lesions. The palms and soles not infrequently share in the eruptive process (Schamberg). After twenty-four to forty-eight hours the eruption undergoes involution, which is accomplished by the fourth or fifth day. In some instances a furfuraceous desquamation follows. Occasionally the eruption is scarlatiniform in type or through arrangement of lesions may closely simulate that of measles. With the eruption there may be a moderate elevation of temperature and catarrhal symptoms involving the eyes, nose, throat and bronchi. The temperature is usually 100° to 101° F., but may be higher. Angina of moderate degree is generally present. The posterior cervical glands are enlarged, a fact of diagnostic import, and other glands may be involved.

Etiology and Pathology.—The disease occurs more often in children; rarely in infants under six months of age, whereas adults are not infrequently attacked. It occurs in epidemics and the contagium is frequently transmitted by fomites. It is contagious in all active stages.

Diagnosis.—Rötheln is most likely to be confused with rubeola and scarlatina. From rubeola it is differentiated by a more variable period of incubation, a shorter period of invasion, less marked catarrhal symptoms and more marked glandular involvement. The eruption has no particular arrangement, is of shorter duration, frequently disappears in one area as it is appearing in another, and the color is a lighter red. In scarlatina marked general symptoms, including fever,

Fig. 137



Variola, eighth day of eruption. (Heman Spalding.)

nausea, vomiting, headache, angina, glandular involvement at the angles of the jaw and leukocytosis are distinguishing features. The eruptions may be similar, but in scarlatina desquamation is a prominent and almost constant feature, while in rötheln it is poorly marked or absent.

Treatment.—No medication is required. Rest in bed in a well-ventilated room with the diet indicated by the individual case is all that is usually necessary.

Prognosis.—The majority of individuals suffering with rötheln recover. Fatal cases only occur from some complication.

Variola.—**Synonyms.**—Small-pox, The Pox. Fr., *Variole; Petite vérole;* Ger., *Blätten* or *Pockern;* Ital., *Vajuolo.*

Definition.—This is an acute, infectious and contagious disease, characterized by papules, vesicles, and pustules on the skin, with involvement of the mucous membranes, and associated constitutional symptoms.

Symptoms.—The period of incubation varies from eight to fourteen days, during which time practically no symptoms are present. Those of the prodromal stage usually appear suddenly and are characterized by chill, followed by an abrupt rise of temperature, which may reach 103° to 105° F., or higher. This may increase somewhat during the succeeding few days. Associated with this are headache, pains in the lumbar, sacral, and epigastric regions, nausea and vomiting. When the temperature is high and headache severe, delirium may be present, and convulsions are not infrequently associated with this stage in children. The pains in the back may be severe in hemorrhagic cases and are present in about half of all cases (Welch). Hyperemia of the fauces may be present at this time. These symptoms last with varying severity for three or four days, at which time the characteristic eruption appears. In the interval other rashes may occur.

Initial Rashes.—(*Variolous erythema; Variolous roseola*).—These eruptions appear on the second day and may be erythematous or hemorrhagic, generalized or localized. The erythematous type may resemble measles or be scarlatiniform in character. While these rashes may be more or less generalized, they occur more frequently about the groins and pubes, the hypogastric region and the inner face of the thighs. They are also found about the axillæ, the extensor surfaces of the joints and over the lumbar and cervical regions. These eruptions usually disappear promptly and the frequency of their occurrence differs in different epidemics. A petechial eruption is seen less often and may have the same distribution but is usually indicative of a severe type of the disorder.

Small-Pox Eruption.[1]—On the third or fourth day this eruption is noted first on the face and scalp and during the next two days extends over the trunk and limbs, including the palms and soles. Upon the appearance of the eruption marked improvement is noted in

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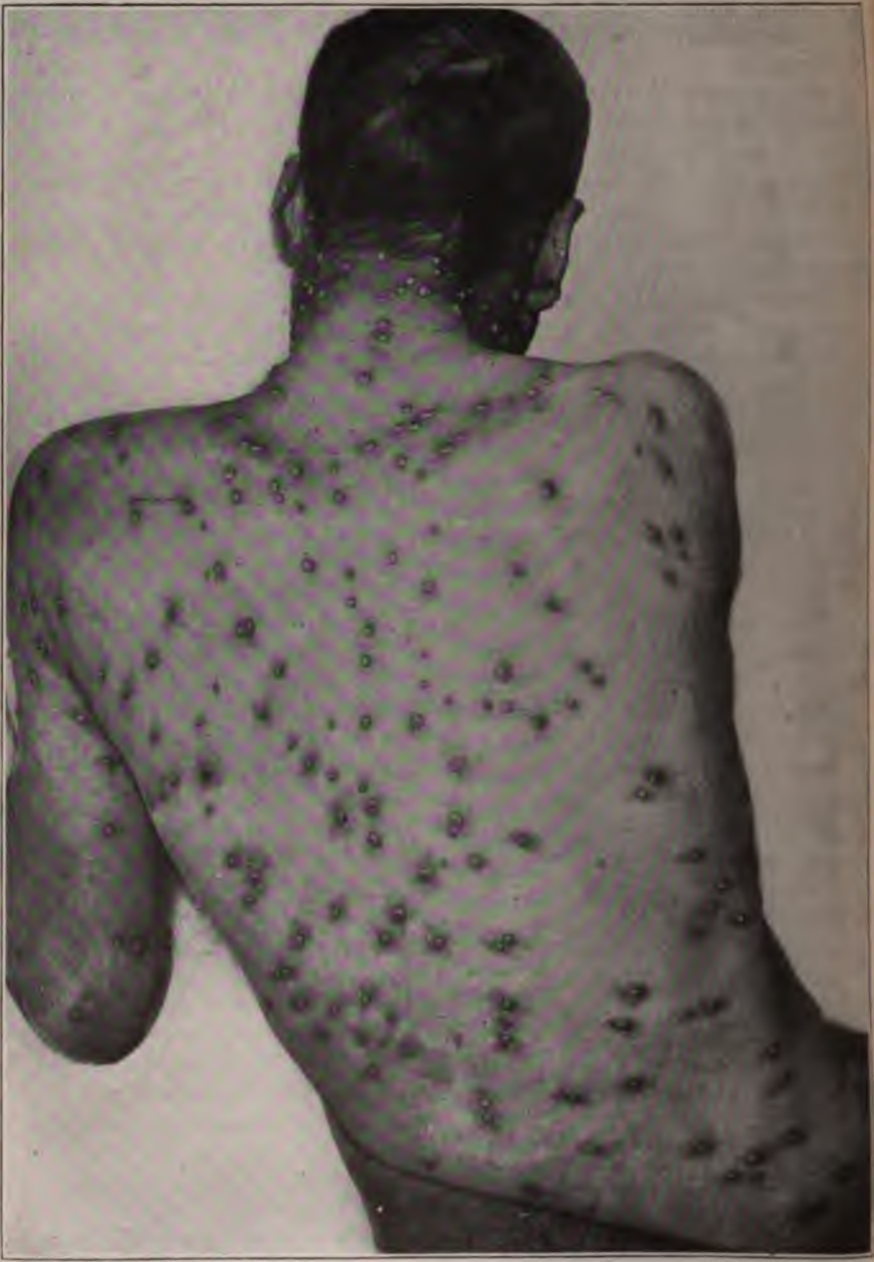
[1] Discussion on small-pox before Amer. Derm. Assoc., May, 1901, Jour. Cut. Dis., 1901, xix, p. 484.

Fig. 138



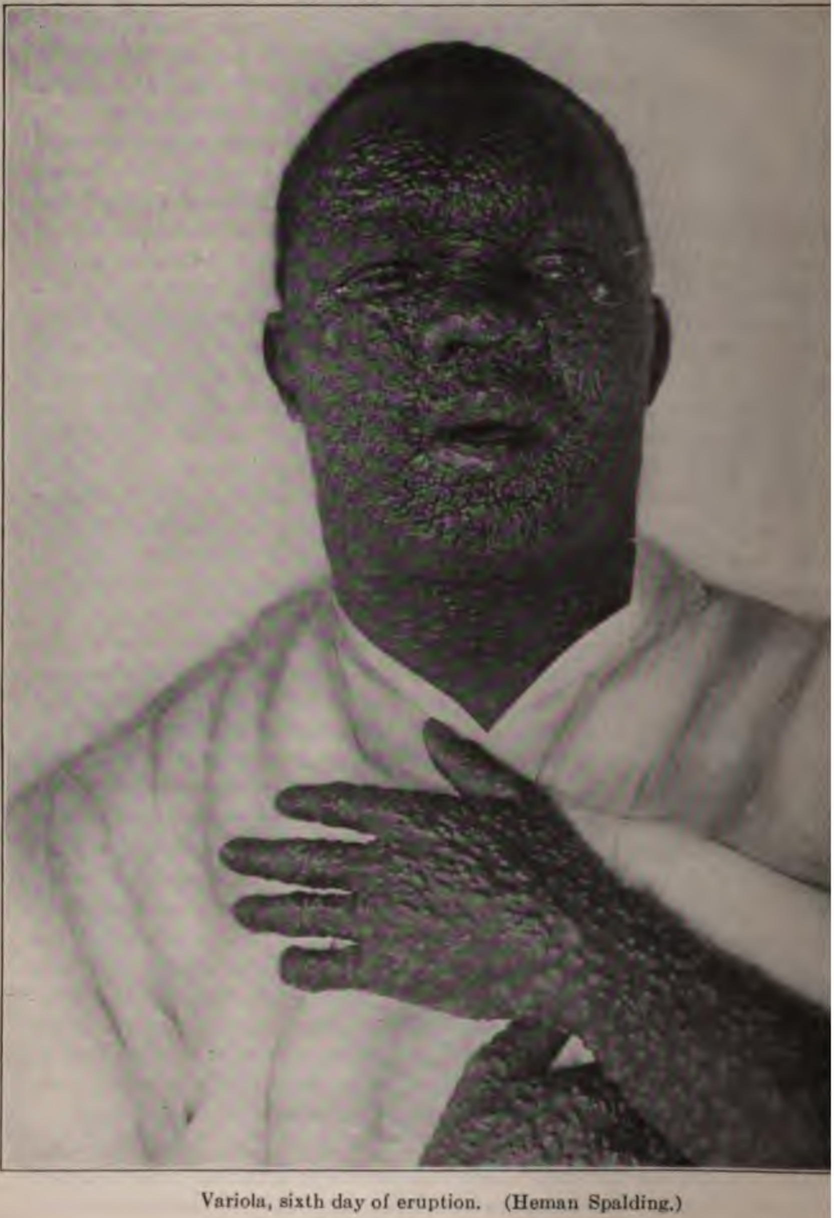
Variola, eighth day of eruption. (Heman Spalding.)

the constitutional symptoms present during the stage of invasion. The temperature often becomes normal, the pains in the back, the headache and nausea disappear, and the individual feels comparatively well. In mild cases these symptoms may not disappear for two or three days after the beginning of the eruptive stage (Welch). The earliest lesion is a small, red spot, which rapidly develops into a pinhead-sized, or larger, firm, conical papule, which presents to the touch the feeling of a shot imbedded in the skin. On the trunk they may be surrounded by a narrow, rosy areola. The lesions continue to develop for two or three days, when the earlier ones are transformed into vesicles. These are situated upon the summit of the papules and at first contain clear serum. They enlarge for two or three days, becoming pea-sized and umbilicated. The umbilication of the vesicle in variola is characteristic, though not invariably present. The epidermis around the vesicle may be irregular, puckered or fluted. About the sixth day of the eruption the contents of the vesicles become turbid and rapidly transformed into pus. The lesion enlarges and becomes globular, the umbilication disappears, the areola enlarges and assumes a more vivid hue. The skin becomes tumid and more or less swollen. The latter condition is particularly well-marked on the face which, through the swelling at this time, may be greatly disfigured, the features becoming indistinguishable. The eyelids may be so swollen as to preclude opening, and the lips, nose and ears are similarly involved. The scalp shares in the process and is frequently painful. As the lesions undergo evolution and involution in a given period, the face and scalp may present pustules while vesicles are still present upon the lower limbs. The comparative uniformity in the stage of development in the different regions is an important distinguishing feature. The pustules on the palms and soles differ in their appearance on account of the thickened, hardened epidermis. They appear deeply situated, often are abundant and may be painful. The mucous membranes of the lips, mouth, nose, pharynx, and larynx become involved, and in severe cases other mucous membranes participate in the process. In the mouth, owing to heat, moisture and friction, reddened and eroded surfaces are present in place of vesicles or pustules, though the tongue may exhibit pustules and be greatly swollen. Deglutition becomes painful and sometimes impossible. Hoarseness and aphonia may occur. The nares become obstructed with purulent material and crusts interfere with breathing. At the time of development of pustules the temperature again rises and remains high during the stage of suppuration (secondary fever). The degree of elevation of temperature and the severity of the associated symptoms depend upon the extent of the eruption. The temperature ranges from 102° to 105° F. and headache returns, with restlessness and, frequently, delirium. In severe cases the patient sinks into a typhoid state.

Between the eleventh and fourteenth days desiccation begins and all the symptoms decline. The pustules dry, forming yellowish and brownish crusts. The temperature falls, the pulse-rate becomes lower,

restlessness and delirium disappear and the general condition improve
As the tumefaction subsides and the crusts fall, purplish or violaceo
areas, as well as scars, are left. Pigmentation follows and slow
disappears. The cicatrizations are of a dull, purplish hue at fir

FIG. 139



Variola, sixth day of eruption. (Heman Spalding.)

becoming white later. They occur chiefly upon the face, and when closely set together present a ridged and corded appearance, owing to the elevation of narrow bands of normal tissue between the depressed surfaces of the scars. In time they become less conspicuous. During this period the hair may fall, but is usually restored unless the suppurative process has destroyed the hair follicle.

Confluent Variola.—This is a severe type and is characterized by an extensive and rapidly developing eruption and marked constitutional symptoms. In the early stages the temperature may reach 105° or 106° F. There is severe headache, pain in the lumbar and sacral regions, nausea and vomiting. The eruption may begin on the second day and may reach its full development in twenty-four to forty-eight hours. The evolution of the lesions from papules to pustules is rapidly accomplished, and the lesions are so closely set as to cover most of the cutaneous surface. Confluence occurs only on the face, hands and feet, the lesions remaining discrete on the trunk. The confluent pustules are usually flat and present a milky or pasty appearance (Welch). Tumefaction and edema of the face produce the deformity above described to a higher degree. The mucous membranes of the mouth, nose, pharynx and larynx participate in the process and phonation, respiration and deglutition are impeded by the tumefaction and suppuration present. The cervical glands become markedly swollen and salivation is a common symptom at this time (Sydenham).

Hemorrhagic Variola.—(*Black small-pox, Variola nigra maligna*).—Two types of hemorrhagic lesions occur: one purpuric, the other pustular with hemorrhagic contents. In the purpuric type (*purpura variolosa*) following the period of invasion, characterized by severe symptoms, an irregularly distributed scarlatiniform eruption appears on the trunk and extremities. This eruption soon becomes a deep purple-red and petechiæ, vibices and ecchymoses develop. The face is swollen and the eyes markedly affected with ecchymoses upon the conjunctiva. The temperature is not high and the mind remains clear. Hemorrhages occur from the various mucous membranes, which are coincidentally involved. This is a rapidly fatal type.

In the second type, variola pustulosa hemorrhagica, the disease begins like the ordinary forms of severe grade but the vesicles instead of containing serum are filled with blood. When pustules form they also contain blood. The hemorrhagic vesicles or pustules may be limited to certain localities, or have a generalized distribution. Petechiæ and ecchymoses are not infrequently disposed among the hemorrhagic vesicles and pustules. The early appearance of hemorrhagic lesions indicates a more severe process. Hemorrhages occur from the various mucous outlets of the body and the disorder usually proves fatal, though in this type the course is somewhat longer than in the purpuric form.

In addition to the above types, variola may occur in mild form.[1]

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[1] For a consideration of the symptoms and diagnosis of modified small-pox as it has appeared in epidemics, see Welch, Phil. Med. Jour., 1899, iv, p. 973, and paper by Hyde, Illinois State Board of Health, 1900.

Large numbers of cases of this type have occurred in epidemics in this country during the past twenty-five years. In this form the disease presents the same symptoms as those exhibited in cases modified by a previous attack of the disorder or by vaccination. In these, however, no previous modifying factor had occurred. The description given below includes this type.

Varioloid.—The prodromal symptoms are usually mild, though occasionally severe, and their duration is more variable. This stage may occupy a period of from one to five days. Upon the appearance of the eruption the general symptoms subside and there may be no secondary fever. Preliminary rashes occur more frequently than in the ordinary forms. The eruption may appear first on portions of the cutaneous surface other than the face, the course is shorter and the eruptive manifestations are less marked. The lesions are often limited to a small number although in some more severe cases they may be generalized and in rare instances tend to confluence. The disease may be terminated at the vesicular stage, the vesicles drying and becoming pustular. Occasionally these abortive vesicles form verrucous lesions, especially on the face, forming examples of the so-called horn, crystalline or wart-pox. These lesions gradually undergo resolution. The period of desiccation is, as a rule, short and after shedding of the crusts scarring rarely results. The mildness of the disorder [illegible] emphasized by the frequency with which these patients appear at [illegible] clinic feeling well and entirely unaware of the existence of a disease [illegible] any importance.

Complications and Sequelæ.—Among cutaneous complications the most common are furuncles, abscesses, erysipelas and, more rarely, gangrene. The latter, when attacking the scrotum, may terminate fatally. Schamberg[1] described an impetiginous eruption (*impetigo variolosa*) occurring during the period of desiccation, characterized by large, flat bullæ, containing a thin, dirty yellow fluid, which terminated in crusting. Other complications are otitis media, conjunctivitis, keratitis, which may be followed by ulceration and perforation of the eyeball, disorders of the respiratory tract, including laryngitis, bronchopneumonia and pleurisy. In addition, arthritis is an occasional complication.

Etiology.—The disease occurs at all ages and in either sex. It is more prevalent during the colder seasons of the year. It is transferred directly or by mediate objects, such as clothing or other articles which have come in contact with the infected individual. It may be conveyed by a third person, or a domestic animal. The contagion may be transferred in active state for some distance through the air (Schamberg). It is contagious during all the active periods, most markedly so during the stages of suppuration and desiccation. It is readily inoculable and is undoubtedly of parasitic origin, but the specific microörganism has not been isolated. Secondary invaders, such as

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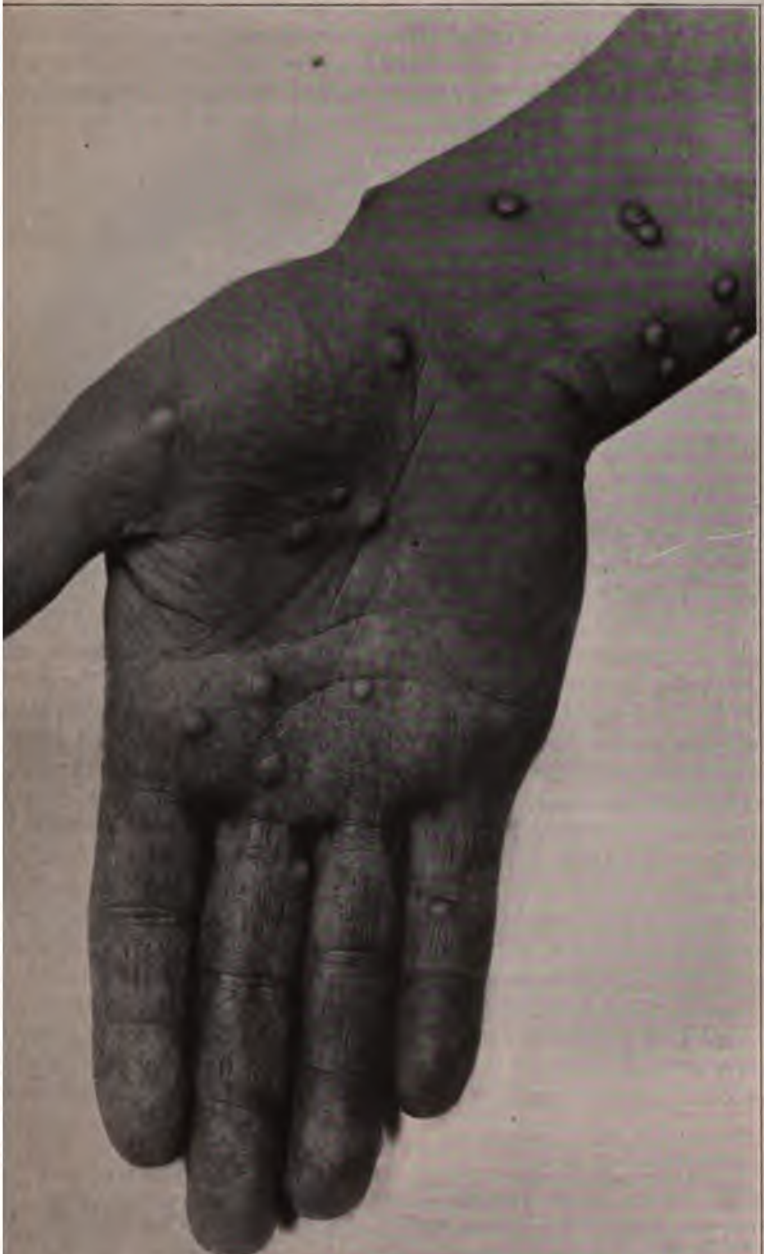
[1] Jour. Cut. Dis., 1903, xx, p. 215; "Secondary Small-pox Eruptions." See this article for more complete description of secondary eruptions occurring with variola.

FIG. 140



Variola, tenth day of eruption. (Heman Spalding.)

streptococci, are usually present and are responsible for many of the complications. The subject of protozoa has been given the most careful study and opinion is divided as to their causal relationship. The most important of the earlier contributions are those of Guarnieri,[1] who found small bodies in epithelial cells of the skin in small-pox and vaccine vesicles and in the cells of lesions produced by inoculation. These were designated *Cytorrhyctes variolæ* and *Cytorrhyctes vaccinæ*. These findings were confirmed by Clark.[2]

Exhaustive and valuable contributions on this subject were made in this country by Councilman, Magrath and Brinkerhoff[3] and Calkins.[4] While this is the most important bacteriological finding, many believe these bodies to be cell degenerations.[5]

Pathology.—The formation and structure of the vesicle in variola has been carefully studied by many, including Unna and later by Councilman, Magrath and Brinkerhoff.[6] The vesicle is situated in the rete and in the beginning changes are noted in its upper portion. Two types of degeneration occur in the epithelial cells: Reticulating colliquation, which occurs more abundantly in the upper portions and at the periphery, and ballooning degeneration in the lower portions and center of the vesicle. According to Unna, cavity formation begins in the upper portion of the prickle layer even in the papular stage by reticulating colliquation of the edematous epithelial cells. The process advances slowly, so that between and above the small cavities compressed layers of cells remain forming trabeculæ, or septa, and the roof wall. In the center the septa have a perpendicular arrangement, while at the sides they are directed outward. At the same time the epithelium in the lower layers of the rete undergo ballooning colliquation. This extends to the lowermost cells and involves the prickle layer of the hair follicle. The ballooned cells found here are smaller than those occurring in varicella, being only two or three times the size of the normal cell from which they evolve. They commonly contain two nuclei, rarely four, and very exceptionally a larger number. Epithelial giant cells, common in varicella, are rarely seen in variola. The balloon cells are disconnected from loss of the intercellular bridges or prickles and form a loose collection covering the swollen papillæ at the base of the lesion and extending upward to the middle portion of of the rete and downward along the hair follicle. The lesion is thus divided into an upper and lower portion. In the upper portion the cavity extends laterally by liquefaction of the marginal cells, while in the lower portion where ballooning degeneration is present the process extends more slowly. Thus the peripheral portion of the cavity is more superficial than the center and the upper and projecting portions form a main cavity. Below in the center and connected with this is a network of wide spaces and canals between the loose ballooned epithelial cells. The process continues by the reticulating colliquation, extending downward at the periphery, and by ballooning degeneration occurring coincidentally at the base of the new peripheral supplementary cavities. In the center there occurs a gradual transition of

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[1] Centralbl. f. Bakt., 1894, i, xvi.

[2] Brit. Med. Jour., ii, 1894.

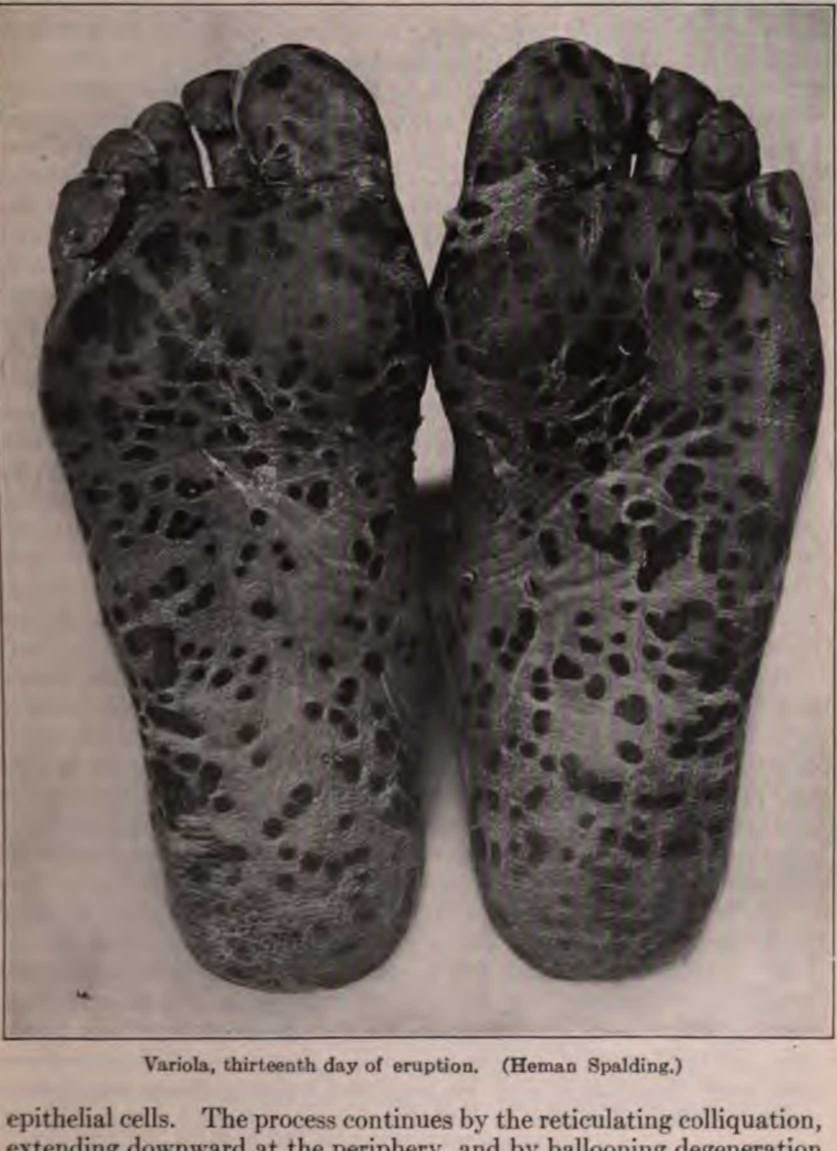
[3] Jour. Med. Research, May, 1903, ix, p. 372. See also Funk, Brit. Med. Jour., 1901, i, p. 448, abstr. in Archiv, 1903, lxv, p. 290; Stokes, Bull. Johns Hopkins Hosp., 1903, xiv, p. 214; Sanfelice and Malats, Archiv, 1902, lxii, p. 189; Thompson, Jour. Med. Research, 1903 04, x, p. 71.

[4] Jour. Med. Research, xi, 1904.

[5] Salomon, Annales Pasteur Inst., April, 1897; Ewing, Jour. Med. Research, 1905, xiii, and others.

[6] Loc. cit.

FIG. 141



Variola, thirteenth day of eruption. (Heman Spalding.)

the ballooned cells at the base into flattened cellular cords and processes of the cavity. Later, the whole of the colliquated and the greater number of the compressed epithelial cells gradually undergo fibrinoid transformation. The umbilication is accounted for by the more active reticulating colliquation going on at the periphery, while the slower ballooning degeneration continues at the center. In this way the outer margin enlarges more rapidly than the center, thus producing a depression. The papillæ at the margins are obliterated by pressure, while in the center they project into the base of the lesion. The umbilication has been explained also by the presence of the lesion around a hair follicle or sweat pore, which hold the center down. Weigert explained the umbilication by the presence of a diphtheroid degeneration in the center of the lesion which prevented its distention by the exudate, in addition to the part played by the hair follicles and sweat ducts. In a large series of sections Councilman, Magrath and Brinkerhoff usually found hair follicles or sweat ducts at the point of umbilication. However, it is well known that umbilicated lesions occur on the glans penis where no hair follicles exist. It therefore seems reasonable to attribute the umbilication to both degenerating changes and their peculiar anatomical situation. In the corium in the vesicular stage the papillary bloodvessels are dilated and many plasma cells occur in their sheaths, but emigration of leukocytes is limited. After the fifth day the vessels become markedly dilated and dense leukocytic infiltration occurs in the papillary layer. This infiltration extends upward into the epidermis, completely filling the former vesicles. The early suppuration is thus a part of the variolous process. Continued suppuration is due to added infection with pyogenic microorganisms.

Diagnosis.—The important points in ordinary cases are the initial general symptoms lasting two to four days, which largely subside upon appearance of the eruption; the selection of special areas by the eruption, such as the face, hands and feet; its course from papules to vesicles and then pustules in regular order during a period of two to four days; the appearance of secondary fever with the pustular stage, and the termination of the process with crusting. The presence or absence of the scar showing successful vaccination is of great importance.

In varicella the early general symptoms are mild or absent or begin with the eruption; the eruption elects the covered portions of the body, especially the back; the lesions are vesicular and superficial, appear in groups and may be seen at a given time in all stages of development. They run a rapid course and no secondary fever develops. In mild cases of variola with only a few lesions difficulty arises and some observation may be necessary before definite conclusions can be reached. Isolation in doubtful cases is required for protection of others.

The pustular syphiloderm may cause confusion. Constitutional symptoms sometimes accompany these lesions, which add to the difficulty. As a rule, there will be a positive history of syphilis and the concomitant symptoms may be present. The constitutional symptoms are of a low grade and of longer duration and the eruption persists for a longer period. The individual lesions may be identical in appearance. The complement-fixation test is of value. In this connection it should be remembered that latent syphilis may be present in an individual suffering with variola.

Occasionally measles may be confused with a prevariolous eruption, or with a variolous eruption in the early stages. The characteristic catarrhal symptoms of measles, and the different course pursued by the eruptions will distinguish between them. The prevariolous eruption is of short duration and the early variolous eruption soon changes its form.

Acne, drug eruptions and impetigo are readily excluded by absence of constitutional symptoms, and the character and course of the respective eruptions.

Treatment.—Isolation of the patient and careful disinfection of all excreta and all objects used by or for the patient is essential. A well ventilated and darkened apartment is advisable. The general management of the various symptoms arising during the several stages, which include the fever, nervous symptoms and complications are all to be treated as indicated by the principles of general medicine.

Local treatment for the cutaneous manifestations is used to overcome infection and relieve irritation. General baths containing mercuric chlorid in a strength of 1 : 10,000 or 1 : 20,000, or alkaline baths containing bicarbonate or biborate of sodium in the proportion of three to six ounces in thirty gallons of water, are valuable in removing pus and crusts and preventing infection. These baths also reduce the temperature. After the bath the surface may be dusted with an inert powder, or a simple ointment may be applied. Schamberg suggests lukewarm baths for the hands and feet, which often are the site of much pain. More localized suppurative foci may be cleansed with a stronger solution of mercuric chlorid, 1 : 5000. Local impetiginous areas respond to ointment containing 2 per cent. salicylic acid or an equal amount of ammoniated mercury.

No application or other treatment prevents scarring, though the prevention of infection or its limitation may reduce the cause of this condition. Tincture of iodin painted over the face daily for several days is employed and recommended by Schamberg, Rockhill[1] and others. Under this treatment the pustules shrink, crusting is accelerated and secondary infection is modified or prevented. It also lessens the odor emanating from the decomposing surfaces. It may be necessary to reduce the strength of the iodin for sensitive skins. The red light treatment of Finsen[2] appeared to be valuable in preventing suppuration, if employed with careful attention to details and instituted before the fifth day of the disease. Its use in this country has largely been a disappointment. Simple ointments, medicated or otherwise, are useful in the removal of crusts and make the skin more comfortable. Scratching should be prevented and this may be lessened by the use of antipruritic lotions which contain 1 per cent. phenol or 5 per cent. boric acid, or mercuric chlorid in a 1:1000 solution.

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[1] Jour. Amer. Med. Assoc., 1912, liii, p. 273; "Iodin in Small-pox."

[2] "Phototherapy," translated from the German by J. G. Sequeria, London, 1901; Brown, Brit. Med. Jour., 1903, ii, p. 1409; Naunyn, Unterelsässischer Arzteverein, Sitz., 26 Juni, 1903; abstr. in München. med. Wchnschr., 1903, i, p. 1360; Depray, Jour. med. de Bruxelles, 1903, viii, p. 69; Emmerson, Med. Times and Hosp. Gaz., 1903, xxxi, p. 419; Carassa, Il Morgagni, i, No. 4; abstr. in Monatshefte, 1903, xxxvi, p. 336; München. med. Wchnschr., 1903, i, p. 1810.

Prognosis.—The mortality of the disease where vaccination has been rigidly enforced has been reduced to the minimum. In recent years in this country the major portion of the victims of the disease have recovered. In the very young, in the aged and in individuals depleted by alcohol the disease is more serious. Confluent cases are grave and the hemorrhagic forms usually terminate fatally.

Varicella.—**Synonyms**: Chicken-pox. Fr., *Variolette;* Ger., *Spitzblattern, Wasserpocken;* Ital., *Moroiglione.*

Definition.—Varicella is an acute, contagious disorder, characterized by vesicles which develop in successive crops and is accompanied by mild constitutional symptoms.

Symptoms.—The incubation period is variable, according to different observers, but probably averages fourteen days. As a rule the first symptom of varicella is the eruption of the disease. Mild prodromal symptoms may occur for a day, or for only a few hours, exhibited as malaise and moderate elevation of temperature. In adults a prodromal stage of one to three days may occur (Welch and Schamberg) in which the symptoms may be more marked. Rarely, a prodromal scarlatiniform erythema is seen, which is of short duration.[1] The eruption of varicella is usually first seen on the trunk and head and later on the extremities. The lesions occur in successive crops over a period of several days, the individual lesions running a short course; as a rule, the trunk is most extensively involved and the back more than the anterior surface. The extremities are least affected, particularly about the wrists, hands, ankles and feet. The face is usually affected and in extensive cases the lesions may be numerous in this situation. Coincident with or slightly preceding the eruption moderate fever occurs, the temperature usually reaching only 99° to 100° F., and continuing from one to three days. Exceptionally a higher temperature is noted. The early lesion is a small, erythematous spot which soon develops into a vesicle with clear contents; occasionally the vesicle develops without apparent preceding erythema. The vesicles are pinhead- to pea-sized, rounded or oval, superficial in situation, having a thin roof-wall, and are well projected from the surface. Many of them are surrounded by a delicate red areola. The appearance of the early vesicle is distinctive, in that it stands out, presenting a dewdrop-like appearance, as suggested by Schamberg. Its contents later become lactescent and in some instances purulent. In the course of one to three days the lesion dries and a yellowish, thin crust is formed. The vesicle is at first tense but later becomes flaccid. The lesions are always discrete and vary in number from a few in irregular distribution to large numbers. Occasionally umbilication is simulated in older lesions by desiccation of the center, thus producing a depression. As new lesions continue to develop over a period of several days a multiform eruption is noted, which is characteristic. Thus new vesicles with clear, and older ones with turbid contents, may be seen intermingled with others which have reached the crusting stage. After the crust falls an erythematous spot remains for a short time. Occasionally scars result from infection, particularly on the face. The mucous membranes are usually attacked, a few lesions occurring on hard and soft palate, more rarely on the buccal mucous membrane, postpharyngeal wall and the tongue. In these situations the vesicles are rapidly transformed into superficial erosions surrounded by a red areola. Itching of varying degrees occurs.

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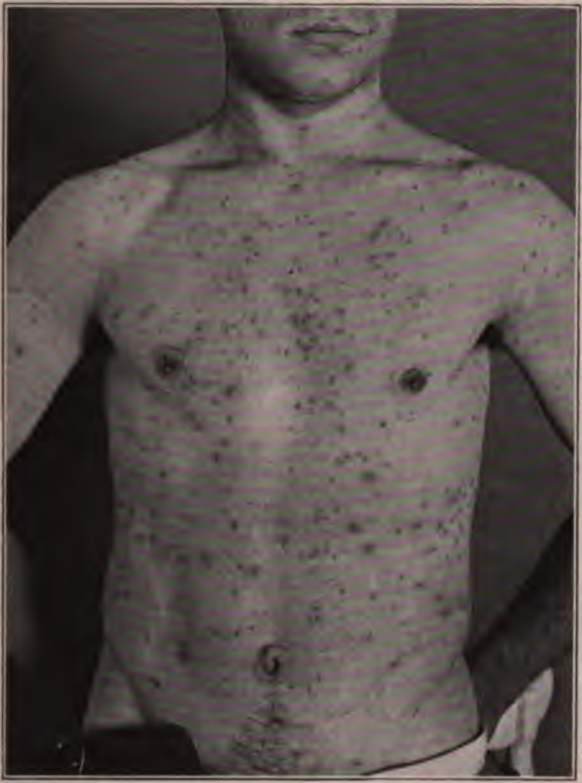
[1] Anthony, Jour. Cut. Dis., 1906, xxiv, p. 68; Welch and Schamberg, Acute Contagious Diseases, 1905; Comby, Archive de Méd. de Enfants, 1919, xxii, p. 57.

Fig. 142



Varicella. (Fordyce.)

Complications.—Welch and Schamberg describe large, flat pustules or bullæ (*impetigo varicellosa*), which may attain the size of a silver half-dollar. They occur by peripheral enlargement around the border of the crust in partially desiccated vesicles, and dry into yellowish, friable crusts. Gangrene rarely occurs and is the result of secondary infection. (Cf. section devoted to Varicella gangrenosum infantumi.)

Etiology.—The disease occurs most frequently in children between the age of one and seven years, but it has also been seen in infants under one year. It is not strictly a disease of childhood, as many adult cases are recorded. During a period of eight years Schamberg[1] saw twenty-five cases, and a number have been under the observation of the author. The occurrence of the disease is not affected by race or season. It is very contagious before the period of eruption as well as in the later stages, and is usually contracted by direct exposure to the infected individual. The duration of the contagiousness is not determined. Inoculation experiments have been unsuccessful as a rule. Le Feuvre[2] believes varicella and herpes zoster are related etiologically.

Pathology.—According to Unna, the vesicle is multilocular, its cavities being formed by the reticulating liquefaction of groups of prickle-cells in the middle and upper portions of the rete, the septa consisting of cells between the cavities which had not undergone liquefaction. Ballooning colliquation occurs in the cells at the base and lateral margins, as well as along the septa. These ballooned cells are large and contain from two to sixteen or more nuclei. The contents of the vesicles when fully developed consist of a finely granular coagulated fibrin and a few ballooned cells. Occasionally wandering cells are found at the base and in the surrounding epithelium. In the corium there is edema, marked vascular dilatation, with some connective-tissue cell multiplication, but practically no leukocytic infiltration.

Diagnosis.—Varicella is to be differentiated chiefly from variola. In typical cases no difficulty is experienced. In mild cases of variola and rather extensive cases of varicella, particularly in adults, much confusion arises. In variola the invasion period occurs within relatively fixed limits, and is characterized by fairly marked constitutional symptoms, as a rule. This, with the regular succession of papules, vesicles and pustules, occurring most profusely on the exposed surfaces, face, wrists and hands, and the disappearance of constitutional symptoms with the appearance of the eruption, are important points. In varicella the irregularity of the period of invasion, the mild, or complete absence of prodromal symptoms, the age of the patient, the early appearance of lesions on the trunk, the usual absence of umbilication in the vesicles and, most important, the multiformity of the lesions—which means that at a given time they may be seen in all stages of evolution on different parts of the body, from the beginning vesicle to the perfectly formed crust—serve as differentiating features. Occasionally impetigo contagiosa is mistaken for varicella. In the former the lesions are frequently of large size, they are more persistent, their distribution is limited to exposed surfaces, and they are rarely, if ever, accompanied by systemic disturbance.

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[1] Diseases of the Skin, and Eruptive Fevers, 1908, p. 447.

[2] Brit. Jour. Derm., 1917, xxix, p. 253; A plea for the recognition of a common origin for shingles and chicken-pox. See also report by Gunn, Brit. Med. Jour., 1918, Part II, p. 574.

Treatment.—The management of uncomplicated cases of varicella is limited to avoidance of irritative influence of all kinds. The lesion may be prevented from becoming secondarily infected by employing a mild, antiseptic ointment, such as a 1 to 2 per cent. salicylic acid, or 5 per cent. boric acid ointment. In most cases a simple dusting powder is all that is required. When itching is a prominent symptom it may be relieved by the use of zinc oxid and aqua calcis lotion containing ½ to 1 per cent. phenol.

Prognosis.—The disease in the major portion of all cases is mild and complete recovery ensues in a short time. It is so mild that isolation is considered unnecessary.

Vaccinia.—**Synonym.**—Cow-pox.

Definition.—Vaccinia is an eruptive disease occurring chiefly in the cow, which may be artificially inoculated into man (vaccination).

The material inoculated into man is obtained by inoculating calves three to six months old with the virus of cow-pox. When the vesicles are well formed the contents are removed and preserved in glycerin. After the material is obtained the animal is killed and examined for tuberculosis, a proceeding which precludes the possibility of coincidentally inoculating tuberculosis. The inoculated disease in man is characterized by a local lesion at the site of inoculation and by mild constitutional symptoms which induce immunity, more or less permanent, against small-pox. While cow-pox and small-pox have not definitely been proved to be identical, most authorities consider the former an attenuated type of the latter.

Vaccination.—On the third or fourth day after vaccination there appears at the site of inoculation a small reddened area which rapidly becomes papular. On the fifth or sixth day this is transformed into a vesicle which is slightly umbilicated and has clear contents. When the lesion arises from several points they coalesce and produce a large vesicle, or the vesicles situated upon a common inflammatory base remain discrete. In either case they puruse the same course as the single vesicle. By the eighth day the vesicle is pea- to fingernail-sized, umbilicated, has clear or lactescent contents, an indurated base and is surrounded by an inflammatory areola which increases during the next two days. The redness may extend for some distance around the lesion and be accompanied by inflammatory infiltration of the tissue. The area is sensitive and often painful and the regional glands become enlarged and tender. During this time slight constitutional involvement is indicated by moderate elevation of temperature and malaise. By the eleventh day the process reaches its height and resolution begins. The mild constitutional symptoms subside, the contents of the vesicles become cloudy or purulent, the surrounding redness and swelling subside and desiccation occurs. Desiccation is complete by the fifteenth day, the resulting crust persisting for seven to ten days longer. When the crust is shed there remains an oval or round, well-defined, slightly depressed, pitted, slightly reddened scar which later becomes white. Occasionally the vaccine vesicle does not run the typical course above outlined, but still the vaccination will be successful. For example, the process will be retarded, the vesicle not forming until the seventh or eighth day, after which it pursues an ordinary course. More rarely, it may form a day earlier than common but with a subsequently normal course. *Vaccinia hemorrhagica* is a term employed to describe the condition when the areola about the vesicle becomes hemorrhagic. Petechiæ and ecchymoses may occur coincidentally in other locations.

In rare instances an atypical crust forms which, after being shed, leaves an ulcer which may be slow in healing. In a certain proportion of cases atypical lesions occur which are not followed by protective results. A peculiar lesion, known as "raspberry sore" was early described by Morrow. This is a bright red, nodular lesion covered with a thin crust, which persists for months. Any marked irregularity in the local lesion, either in size or shape, evolution, contents of the vesicle, crust formation or ulceration should be considered an unsuccessful vaccination.

Generalized vaccinia may occur as the result of general infection, or the lesions may be induced by auto-inoculation. In the former, which is rare, the lesions develop from the fourth to the tenth day after vaccination. They may occur on any portion of the cutaneous surface and may be few or numerous. They evolve from papules into vesicles and finally pustules and occur in successive crops, thus producing at a given time lesions in all stages of development. Constitutional symptoms vary with the extent of the eruption and may be absent. The eruption undergoes complete resolution by the twenty-first day. Generalized vaccinia from inoculation is more common. The lesions may be limited to a few or the eruption may be extensive and may occur on any part of the cutaneous surface. The lesions are spread by scratching or otherwise and develop at different intervals after the primary vesicle but are rarely seen after the third week. Generalized vaccinia at times resembles variola but lacks the prodromal stage of the latter and the evolution of the lesion differs. Furthermore, it follows vaccination.

Vaccinial Eruptions.—Certain toxic eruptions are seen following vaccination in the same way that they occur in variola and varicella. The most important of these is an erythema which had been described under the title "vaccinal roseola" (*roseola vaccinosa*) and which occasionally resembles measles. It usually begins about the tenth day after vaccination, but may occur at other periods. It is first seen about the area of vaccination and spreads to other portions of the body. The macules composing this eruption are described as rosy. Their arrangement is irregular and, as a rule, they remain discrete, but occasionally by coalescence a diffuse erythema is formed. The eruption is of short duration. In addition to the above, urticaria, multiform erythema in any of its manifestations, and scarlatiniform erythema occasionally are seen. These are all conditions which are directly concerned in the vaccination process.

Several cutaneous affections follow vaccination as the result of infection occurring in the vaccination wound. The pathogenic microorganisms gain entrance occasionally at the time of vaccination, but more often subsequently. A classification of the complications of postvaccinal eruptions was made early by Morris.[1] and Frank.[2] Among local infections are impetigo, infectious dermatitis, furunculosis, cellulitis, local gangrene, erysipelas and lupus vulgaris. Only one or two of these need description. Occasionally a marked inflammatory reaction occurs about the area of vaccination characterized by swelling and redness which may spread over much of the surface of the arm. In some cases a diffuse cellulitis occurs with associated constitutional symptoms. The glands may become involved and the inflammatory process may terminate in abscess formation. In some instances necrosis occurs with sloughing and ulceration, followed by scar formation. At times infection with pyogenic microörganisms occurs at the site of the vaccination and the infection is transferred to various areas, inducing impetiginous lesions, both vesicular and bullous. A pustular dermatitis is produced in the same way. As through any infection atrium, erysipelas may find entrance through a vaccination wound. The occurrence of lupus vulgaris is rare but authentic cases are on record. (Cf. section on Lupus vulgaris.)

General infections include pyemia, septicemia, gangrene, tuberculosis and leprosy. Under present methods of vaccination pyemia and septicemia rarely occur, although in the past serious epidemics were noted. Occasionally gangrene follows, as it does after various infectious diseases. (See section on Varicella gangrenosum infantum.) While examples are recorded of the occurrence of syphilis, tuberculosis and leprosy following vaccination, it is practically impossible at the present time for such accidents to happen. Vaccine prepared as described in the beginning of this section precludes the transmission of any of these diseases with the vaccine. The only possibility, therefore, for inoculating the disorders would be through an accident at the time of vaccination, which can be avoided in all cases by observing the rules of aseptic surgery.

A group of disorders is given as sequelæ, among which are acute bullous and vesicular eruptions, which include pemphigus and dermatitis herpetiformis, eczema and psoriasis. The bullous eruptions of this type occur several weeks after vaccination and are apparently unavoidable and rare. Dermatitis herpetiformis has been described by Pusey,[3] Bowen,[4] Stelwagon[5] and others. The bullous eruptions by Grindon,

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[1] Brit. Méd. Jour., November 29, 1890; abstr., Brit. Jour. Derm., 1891, iii, p. 91.

[2] Jour. Cut. Dis., 1895, xiii, p. 142.

[3] Jour. Cut. Dis., 1897, xv, p. 158.

[4] Ibid., 1901, xix, p. 401.

[5] Jour. Amer. Med. Assoc., November 22, 1902.

Howe, Bowen and others. (Cf. section on Acute pemphigus). The occurrence of eczema or psoriasis as sequelæ is sometimes seen but the connection is difficult to explain. Psoriasis occasionally begins in the area where vaccination was performed, and it may be that the irritation of the vaccination process caused the development of a latent psoriasis. In an individual sensitized to irritation an eczema may also thus be induced or exaggerated. A more or less generalized infection has followed the vaccination of an individual suffering with moist eczema, the virus having been inoculated in various areas.

ROCKY MOUNTAIN SPOTTED FEVER.[1]

Synonyms.—Tick fever of the Rocky Mountains, Piroplasmosis Hominis, Black fever, Blue disease, Sheep camp fever.

Definition.—The above titles designate an acute infectious disease, accompanied by mild or severe constitutional symptoms, with a cutaneous exanthem having multiform characteristics.

It was first described by Wood, in 1896, and in 1899 by Maxey, since which time many observers have made reports concerning its clinical, pathological, and bacteriological aspects. Unrecorded cases have been noted in some of the regions where it is now prevalent since 1873. The disease is found chiefly in the Rocky Mountains, in the states of Montana, Idaho, Utah, and Oregon.

Symptoms.—The disease is ushered in, as are many infectious processes, with chill or chilly sensations and malaise, followed by a rapid rise of temperature and general soreness over the entire body, especially severe in the back and legs. Headache is common and epistaxis is frequently associated and may be severe during the second week. The tongue is coated and constipation is the rule. The temperature rises suddenly after the onset and remains more or less high, from 100° to 105° or 106° F., for ten to twelve days, when in mild cases it begins to decline, reaching the normal in the third week. The eruption occurs in from three to five days after the onset, beginning on the wrists, ankles, and back, and gradually spreading over the limbs and trunk, including the palms and soles in some cases. Late in the disease it may involve the mucous membrane of the mouth and throat. It appears in crops, like a purpuric toxic erythema, the first lesions being pinkish or reddish macules, which later become darker in color and finally hemorrhagic. In severe cases intense hemorrhagic areas may occupy the entire cutaneous surface. Diffused over the surface there is usually an icteric discoloration in addition to the lesions described. The eruption terminates with desquamation during convalescence. This is most marked on the hands, feet, and face.

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[1] Bibliography: Wood, Report, Surg.-Gen. Army, 1896, pp. 60–65. Maxey, Medical Sentinel, 1899, vii, pp. 433–438. Wilson and Chowning, Jour. Amer. Med. Assoc., 1902, xxxix, pp. 131–138; and Jour. Infect. Dis., 1904, i, pp. 31–57. Anderson, Bulletin 14, Hygienic Laboratory of United States Public Health and Marine Hospital Service, 1903. Stiles, ibid, No. 20, 1905. Ricketts, Jour. Amer. Med. Assoc., 1906, xlvii, pp. 33–36, p. 358, and pp. 1067–1069; Jour. Infect. Dis., 1907, iv, pp. 141–153. King, Public Reports, July 27, 1906. Idaho State Medical Association, 1908: Jour. Amer. Med. Assoc., 1908, li, p. 1809 (symposium on Rocky Mountain Spotted (Tick) Fever; papers and discussions by Stewart and Smith, Maxey, Ricketts, Tuttle, Numbers, McCalla, Taylor, Kleinman). Osler's Modern Medicine, vol. iii, pp. 535–540.

With the rise of temperature the pulse rate increases from 110 to 140 in average cases, and the respirations range around 36 per minute. A moderate bronchitis is common and pneumonia is a frequent complication. Nausea and vomiting may occur during the second week and may be intense in severe cases. The spleen is enlarged and tender and the liver shows some increase in size. Hemorrhages may occur from the nose, mouth, stomach, and bowels, and hemorrhagic effusion into the joints has been recorded. The urine is diminished in amount and high colored, and may show traces of albumin and some casts. The nervous manifestations include restlessness, irritability, pain, hyperesthesia, and in severe cases delirium and stupor, while coma usually precedes death. The blood shows a diminution of red cells and hemoglobin. The leukocytes are either normal in number or moderately increased (8000 to 14000), with an increased percentage of the large mononuclears.

Pneumonia, and gangrene of the skin of the terminal extremities, scrotum, and penis, occur as complications.

Etiology and Pathology.—The disease occurs usually during the spring months, from March to July; May and June furnish the major number of cases. It attacks both sexes and may occur at any age. The infective agent is inoculated, in most instances, by the bite of a tick (*Dermacentor occidentalis*). Several hundred cases occur annually and the virulence varies in different years. In Montana a more severe type of the disease is usually noted. It occurs commonly among people who are closely associated with sheep-herds in the mountains. Ricketts and others have reproduced the disease in animals (monkeys, rabbits, and guinea-pigs). The former also demonstrated the susceptibility to the disease of several animals indigenous to that locality (gopher, ground-hog, chipmunk, rock squirrel, and mountain rat).

A bacillus was isolated by Wohlbach[1] from infected ticks and laboratory animals which appears to be important etiologically. The organism is a minute rod occurring in pairs often lanceolate and in stained preparations surrounded by a clear zone. These findings have been confirmed by Foot.[2]

The chief pathological findings, post-mortem, have been noted in the skin, spleen, liver, pancreas, and kidneys.

Treatment.—No specific medication is known and opinion is divided concerning the value of quinin. The general care of the patient, with symptomatic treatment and good nursing, gives best results.

Prognosis.—The average mortality ranges from four to ten per cent.; at times a much higher rate has been recorded. In fatal cases, death usually occurs during the second week of the disease.

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[1] Jour. Med. Research, 1916, xxxiv, n. s. xxix, p. 121 and ibid., 1918, n. s. xxxi, p. 492.

[2] Ibid., 1919, xxxii, p. 495.

CLASS II.

HEMORRHAGES.

PURPURA.

Definition.—Purpura is a condition in which there occur spontaneous hemorrhages in and beneath the skin and mucous membranes. These may vary in size from that of a pinpoint to the palm of the hand or larger. They are smooth, reddish or purplish, non-elevated, usually fairly sharply defined, do not disappear under pressure, and undergo characteristic color-changes, finally disappearing.

Symptoms.—The hemorrhages appear suddenly as bright-red spots, sometimes singly, or as a shower of fine points, as seen in malignant endocarditis. These remain bright-red for a time, but gradually they become dull-red, then bluish, greenish-yellow, dirty-yellow, and finally they fade away; or a temporary or permanent pigmentation may remain. According to their shape, size, and arrangement, the lesions are designated as *petechiæ*, which are pinpoint- to small-coin-sized, usually well-defined macules, sometimes situated about the hair-follicles; *ecchymoses*, which are like petechiæ, except that they are larger and more irregular in shape and in distribution, sometimes covering the entire surface of a limb; and *vibices*, which are linear and band-like arrangements of ecchymoses. Occasionally, the hemorrhage takes the form of bullæ (*bullæ hemorrhagicæ*), or of nut- to egg-sized and even larger tumors (*ecchymomata*). Erythema multiforme, urticaria, and other cutaneous disorders may exist simultaneously. The location of the hemorrhages is usually the extremities, particularly the legs below the knees and the forearms, but the trunk may be involved as well, and, more rarely, the face. The lesions may show a tendency toward symmetrical arrangement. The appearance of the hemorrhage is usually unaccompanied by subjective sensations, but there may be a twinge of pain, followed by a temporary tenderness. The classification of purpura is difficult, and in the absence of more exact knowledge must be of necessity provisional. That there is an idiopathic or primary purpura seems unlikely. A most careful search in such cases would probably reveal an infectious or toxic agent.

Symptomatic Purpura.—(1) *Infectious:* Hemorrhages may occur in the course of any acute infectious disease, but in septicemia, pyemia, and septic endocarditis they are always present and may be very abundant. They are more common in variola than in any other of the exanthemata. Epidemic cerebrospinal fever derives its names, "spotted fever," "malignant purpuric fever," and "petechial fever," from the frequent occurrence of a hemorrhagic eruption. Purpura may take the place of rose-spots in typhoid. In typhus fever or in Rocky Mountain spotted fever, the eruption is always purpuric.

2. *Toxic:* A number of drugs may cause the appearance of purpura. Chloral, the salicylates, copaiba, and, in persons with an idiosyncrasy, a comparatively small dose of the iodids, may lead to an extensive purpuric rash. Chief among the toxins is snake-venom, which has been frequently made use of in experimental work. Benzol and diphtheritic toxin have been used by Duke[1] extensively in the study of the relation of blood-platelets to hemorrhage.

3. *Cachectic:* Hemorrhages about the distal portions of the extremities are very common in the cachexia of the chronic diseases, as in the anemias, the leukemias, Hodgkins' disease, tuberculosis, malignant tumors, chronic alcoholism in senility, and other disturbances of nutrition. In a series of one hundred cases of acute infectious diarrhea in children, Rolleston and Malony[2] observed eleven cases of purpura, located chiefly on the abdomen. It appeared about the thirty-fourth day of the disease and preceded death by one day.

4. *Neurotic:* Purpura may occur both in the organic and functional diseases of the nervous system. In tabes, following the lightning pains over the area where the latter has been most intense; in acute transverse myelitis; in insular sclerosis; in hemiplegia on the affected side; and in the neuralgias, hemorrhage sometimes appears. Among the causative factors functional in nature are fright, hysteria, the menstrual state, and stigmatization, in which bleeding points appear upon the unbroken skin.

5. *Mechanical:* Examples of this appear often after the paroxysms of cough in pertussis, and after the seizure in epilepsy. The application of a tight bandage may lead to a hemorrhagic rash. Stasis in dependent parts is a factor, as shown by the disappearance of the purpura during rest in bed and its reappearance on moving about.

Purpura Simplex.—This type is characterized by the appearance of petechiæ in crops, usually upon the legs. Ordinarily, the rash is the only symptom, but there may be associated with it malaise, slight elevation of temperature, mild joint-pains, and gastro-intestinal disturbance. It is seen most commonly in children, and usually lasts from a few days to a few weeks, but it may become chronic or may recur at varying intervals.

Purpura (Peliosis) Rheumatica (Schönlein's Disease).—The difference between this and purpura simplex is probably one of degree only. In this the joint-pains are more marked, and the purpuric lesions are interspersed with or develop into urticarial wheals, forming *purpura urticans.* It may be associated with erythema exudativum. In rare cases vesicles may be formed, giving origin to the term "pemphigoid purpura." The onset is marked by a sharp rise in temperature, and is usually preceded by tonsillitis, whereby the infectious organism gains entrance. Acute nephritis and pancarditis may complicate the attack. Recurrence at the same season may take place for years.

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[1] Archiv Int. Med., 1912, x, p. 445.

[2] Purpura in Infectious Diarrhea of Children, Lancet, 1911, p. 1552.

A similar complexus of symptoms, which seems to be a variant of purpura rheumatica, has been given the name of "Henoch's purpura." In this the erythema multiforme aspect of the cutaneous lesions may overshadow those of the purpuric type. It is chronic, extending over years' duration. Relapses may occur, or the disease may be constantly present. The slightest trauma, escaping even the patient's attention, may lead to subcutaneous hemorrhage. Epistaxis may occur at frequent intervals. Joint-pain and swelling may vary in intensity. Gastro-intestinal crises, associated with pain, vomiting, and diarrhea, occur and have been not infrequently mistaken for appendicitis. One patient, a young woman, was on the operating table and would have suffered a laparotomy had it not been for the timely warning given the surgeon by the writer. Nephritis, usually hemorrhagic, is of frequent occurrence, and the most common cause of death. According to Lippmann,[1] an interstitial nephritis usually results. In some cases uremia supervenes.

Under the term *purpura hemorrhagica*, also known as *morbus maculosus Werlhoffii*, or "land scurvy," are described cases which may have a gradual onset of small petechiæ, with slight constitutional disturbance, gradually developing into large subcutaneous and mucous membrane hemorrhages. Anemia is rapid, or the disease may have a sudden onset, with the formation of enormous ecchymomata, followed by death in twenty-four hours (*purpura fulminans*).

Etiology.—Purpura may appear at any age, but purpura simplex is said to be more common during the second and fourth decades. Males and females appear to be equally attacked. That it is the result of increase in fragility or permeability of the vessel-walls hardly seems plausible, in view of the fact that in chloroform poisoning, which decreases the fibrinogen of the blood, and after peptone injections, with a consequent increase in antithrombin, the hemorrhages appear soon after the alteration in the blood occurs, and disappear with a return of the blood to normal. In some cases purpura appears only after slight trauma, which in a normal skin would cause no damage. The frequency of the bleeding in the buccal mucous membrane is probably to be explained by the trauma of the food and the action of bacteria on the delicate capillaries. Normally, there is constant repair of capillaries going on, but with a blood-stream altered by toxic or infectious agents this becomes impossible, and subcutaneous and submucous hemorrhages result. That there is an altered coagulability of the blood in all cases of purpura is fairly certain. The ordinary clinical method of drawing blood through a puncture in the lobe of the ear is of little value for the determination of the coagulation time, inasmuch as the tissue juices may neutralize the excess of antithrombin, and for this reason many cases of purpura with normal coagulation time are reported. Whipple[2] has shown that in purpura and hemorrhagic diseases the delicate antithrombin-prothrombin balance in the blood is disturbed, either temporarily or permanently, by disease. Under experimental conditions, the preservation of this balance may necessitate rapid neutralization of antithrombin excess, or a rapid production of antithrombin or prothrombin. The prothrombin factor seems to be rarely involved, but when it drops to zero hemorrhages may occur. The antithrombin factor is frequently involved, and with an excess purpuric or hemorrhagic symptoms result. Experimentally, this factor can be increased by stimulation of the liver with peptone injections. Diseases of the blood-forming organs (aplastic anemia, leukemia) lead to an excess of antithrombin, possibly by the action of the products of cell-disintegration on the liver.

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[1] Ueber hemorrhagische Nephritis bei Purpura. Deutsch. med. Woch., 1912, xxx, p. 1407.

[2] II. Hemorrhagic Disease: Antithrombin and Prothrombin Factors. Archiv Int. Med., 1913, xii, p. 637.

Duke[1] has shown that the appearance of purpuric symptoms is marked by a sharp fall in the number of the blood-platelets, and by a rise with their disappearance. He was able to produce similar conditions by injections of benzol and diphtheritic toxin. No account, however, was taken of the other constituents of the blood. Inasmuch as the platelets are decreased in destruction of leukocytes, and antithrombin increased, it may be that fall in the platelet count is but incidental and not an etiological factor.

Howell[2] failed to find alterations in the constituents of the blood of three cases of purpura, and Cowell[3] was unable to produce purpura with benzol, nor was he able to demonstrate any decrease in blood-platelets.

Treatment.—Absolute rest in bed in a horizontal position is of prime importance. No extremity should be dependent. A long list of remedies has been in use, no one of which, with the exception of the calcium salts, appears to have a scientific basis. As shown by Whipple, calcium has no value except in rare cases of hemorrhage with icterus, in which the calcium has become firmly bound with the bile-pigments. A careful analysis of the blood drawn from a vein into oxalate should be made. Blood-serum, either homologous or foreign, or, better, direct transfusion, is of great value in cases with low or absent prothrombin, but is of no value, and may be harmful, in the presence of excessive antithrombin. For the latter condition there is at present no adequate remedy.

Careful and repeated blood-cultures should be made in all cases and a search instituted for a possible focus of infection, as the tonsil, teeth, or appendix, which should be removed at the earliest possible moment if the source of infecting organisms. Autogenous vaccines from such a focus should be tried.

Crocker pinned his faith to oil of turpentine. Some writers advise quinin, iron, ergot, and adrenalin. External treatment is rarely called for, except in those cases in which the hemorrhagic areas break down, forming ulcers.

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[1] Pathogenesis of Purpura, with Reference to Blood-platelets. Archiv Int. Med., x, p. 445.

[2] Ibid., January, 1914.

[3] Brit. Med. Jour., 1912, p. 1089.

CLASS III.

HYPERTROPHIES.

KERATOSIS PILARIS.

Synonyms.—Keratosis suprafollicularis (Unna), Lichen pilaris, Pityriasis pilaris.

Definition.—Keratosis pilaris is an affection of the skin characterized by the presence of minute horny plugs or papule-like lesions occupying the site of the pilo-sebaceous orifices, and occurring at any age.

Symptoms.—Keratosis pilaris may occur at any age, although it is most commonly seen during the developmental period. The lesions are found chiefly over the arms and thighs, but may occur on any portion of the cutaneous surface. The primary lesion is a papule situated at the mouth of a hair follicle, composed largely of epithelium and sebaceous material deposited in the follicle. The papules are pinhead-size or slightly larger, of a grayish hue, acuminate or convex, sometimes capped with a scale, and not infrequently pierced by a hair. More often the hair is found curled inside the papule. Occasionally it is broken open and a black dot representing the stump remains. This papule may be readily removed, after which there remains a slight depression which soon disappears. The papules are discrete and irregularly distributed in the areas above noted and are never grouped into patches. The skin between the papules may be dry and somewhat scaly, or it may be normal. The projecting papules resemble the condition termed "goose-flesh" and present a roughness to the touch suggestive of the surface of a nutmeg grater. These papules frequently occur with ichthyosis and are considered by Morris and Kaposi as a mild form of that disorder. There may be moderate itching, or the disease may produce so little inconvenience that no attention is paid to it. Brocq[1] describes a white variety, in which there occur lesions scattered chiefly over the arms, forearms, legs and thighs, usually on the extensor surfaces. In addition, three inflammatory types are described: (*a*) A mild form, in which reddish papules are disseminated among those of the white class; (*b*) a form of medium intensity, in which the papules are generally rosy-red in hue; (*c*) an intense form in which well-marked lesions occur over the surface of the chest, the lumbar and pubic regions, and the folds of the larger articulations.

Keratosis pilaris of the face, as described by French writers, is characterized by exceedingly minute, usually conical, occasionally obtuse, papules, each pierced by a fine hair, that develop over the brow, about eyebrows, over the cheeks, and in the inframaxillary region.

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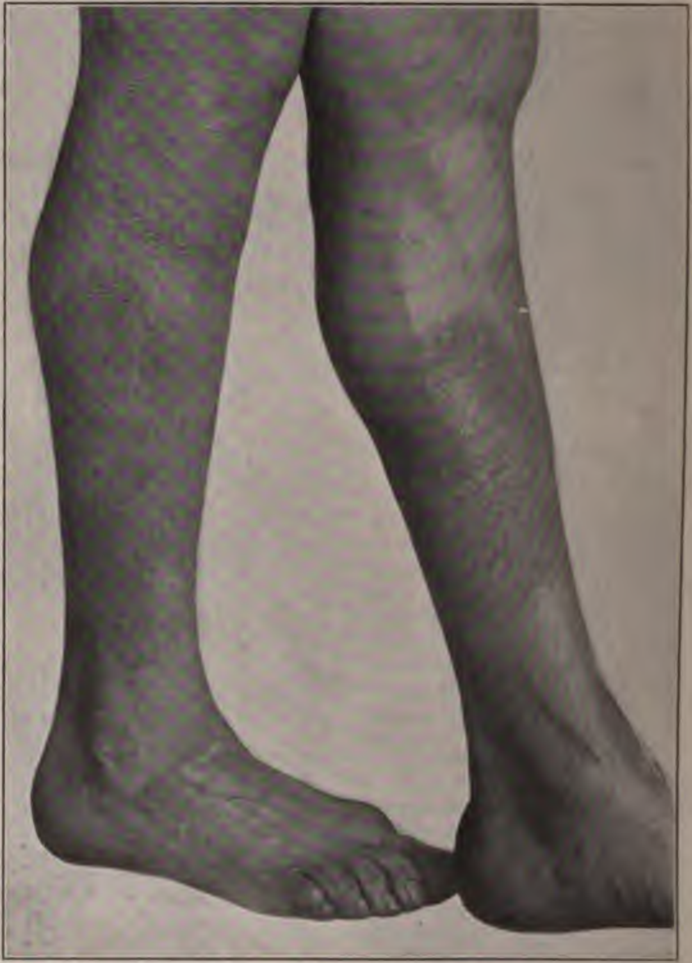
[1] Annales, 1890, s. iii, i, pp. 25, 97, and 222 (an extensive review of the subject, with bibliography).

Tilbury Fox has reported four cases in which the disease was well marked, under the title of "*Cacotrophia folliculorum*" in which the disorder was extensive and apparently was deeper and had a congenital history. It is possible in view of work done since that time that these cases belong to the ichthyotic group.

Fig. 143



Keratosis pilaris. (Fordyce.)

Etiology.—Uncleanliness is commonly given as a cause, but the disease occurs in persons who bathe as frequently as is necessary.

Cool weather has an influence on the disorder. Ichthyosis is a common cause and the disease is frequently seen in these subjects.

Pathology.—The papules are produced by a hyperkeratosis about the orifices of the pilo-sebaceous follicles. In some cases the result is an irritation, which produces a mild degree of chronic inflammation of the periglandular tissue. Giovannini[1] found in twenty-five cases that inflammation was not constant but in some cases was a marked feature. He found that the follicular orifices were much widened and deepened and filled with a horny plug, in which there were coiled often one or more fine hairs. The hyperkeratosis involved not only the follicle, but also the epidermis about it. There was more or less atrophy of the outer root-sheath of the mouth of the follicle, also of the sebaceous glands and of the erectores pilorum. In a few instances, the entire follicle, including the hair papilla, was destroyed. Unna[2] and Lemoine[3] regard the condition as inflammatory in origin and primarily involving the follicles.

Diagnosis.—The disease should readily be recognized by the peculiarities of its seat, its course and the nature of its symptoms. From ichthyosis it can be distinguished by the limitation of its lesions to the orifice of the hair follicle; from the transitory condition known as "goose-flesh," by its persistence after the surface of the skin is thoroughly warmed; and from papular eczema and the other lichenoid eruptions by the relatively insignificant character of the lesions, their evident follicular origin, and either the entire absence, or mild chronic type, of inflammatory symptoms.

The disease is to be carefully differentiated from pityriasis rubra pilaris, in which the characteristic disorder of the scalp, the appearance of plaques of disease covered with fine, pityriasic scales (often upon the tip of the nose and chin) exhibiting a peculiarly dark, smirched appearance, the affection of the nails, the characteristic papules on the dorsal surfaces of the first and second phalanges of the fingers, and the evident admixture of the disease with symptoms of seborrheic type, suffice to determine its nature.

The lesions of keratosis pilaris might possibly be mistaken for syphilis. The papular syphilodermata do not last over a long period of time, their distribution is more general, and there is commonly a history of infection and other manifestations of disease.

The greatest difficulty in the diagnosis lies in distinguishing lichen spinulosis from keratosis pilaris. In the former disorder, which is rare, the lesions occur more frequently in children and they are grouped into patches, occurring most often on the body and also on the extremities, and each papule has a horny spine in its center and the disease is not affected by seasonal changes.

Treatment.—The first consideration is proper bathing. Where this has been neglected it should be instituted and carried out with regularity. Alkaline baths are required in certain instances. Oily or fat substances should follow the bathing. Salicylic acid 1 to 10 per cent. in oil or ointment is of value in temporarily removing the lesions. Olive oil 75 per cent. and glycerin 25 per cent. is of value for use on the general surface.

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[1] Archiv, 1902, lxiii, p. 163 (with bibliography).

[2] Histopathology, 1896, p. 287.

[3] Annales, 1890, p. 224.

Ulerythema Ophryogenes.—This affection was first described by Taenzer.[1] The disorder is located, or at least usually begins, in the eyebrows, whence it may spread to adjacent parts, including the scalp, or it may appear on the extensor surfaces of the upper arms. In early stages the disease presents many similarities to keratosis pilaris, a form of which disorder it is considered to be by some (Dubreuilh and MacLeod). There usually is present a persistent erythema, with small, elevated, horny papules at the mouths of the hair-follicles. The hairs are finer than normal, and are usually broken off close to the surface. The disorder may persist for years without further change, but in the severer forms superficial inflammation and atrophy, both follicular and interfollicular, may result, so that small, depressed scars are surrounded by or included in the hyperemic areas. The resulting alopecia is permanent and may be marked, especially on the eyebrows.

The disease is said to be rebellious to treatment. Internally, a ferruginous and arsenical treatment has been adopted, with local applications of resorcin and salicylic acid, the mercurials, and stimulating shampooings with soap.

LICHEN PILARIS SEU SPINULOSUS (CROCKER).

Synonyms.—Lichen spinulosus (Devergie), Keratosis follicularis spinosa (Unna).

Definition.—This disease was first described by Crocker, and cases were shown by Crocker and Colcott Fox[2] before the London Dermatological Society, in 1883. Adamson,[3] in 1905, described a case clinically and histologically, and reviewed the literature of English and continental cases up to that date. He concludes that the cases described by Hardy and Leloir and Vidal, under the title of *Acné cornée*, were identical with this disease, and also possibly with that of Guibout. One of Hallopeau's cases, described as *Acné cornée en aires*, he also includes; the others he believes were different. In America, the disease has been seldom seen. Bowen[4] described a case, and suggested that possibly the disease here has been described under other titles.

Symptoms.—The disorder is essentially one of childhood, though a few examples are recorded in adults, and it occurs more frequently in boys than in girls. It is characterized by the development of minute papules, situated about pilo-sebaceous follicles, each having a projecting horny spine. The lesions are grouped, forming patches, and occur chiefly about the neck, the buttocks, trochanteric regions, the abdomen, thighs, popliteal spaces, and extensor aspects of the arms. The face, hands, and feet are commonly spared. The eruption appears in crops, and a patch may spring up suddenly and continue to enlarge for a week, when it remains stationary, the color gradually becoming paler (Crocker). The arrangement of the patches tends to be symmetrical. There are usually individual discrete papules in addition to those grouped and forming the patches. The horny spines, which are characteristic, protrude about one-sixteenth of an inch above the surface, and present to the hand when passed over the patch the same sensation as though passed over a nutmeg-grater. The lesions early are red in color, but soon assume the color of the normal skin. They may develop acutely or subacutely and may persist indefinitely without treatment. The horny spine may be removed, leaving a depression in the papule.

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[1] Monatshefte, 1889, viii, p. 197 (from Unna's clinic).

[2] Brit. Jour. Derm., 1905, xvii, p. 40.

[3] Ibid., 1905, xvii, pp. 39–77.

[4] Jour. Cut. Dis., 1906, xxiv, p. 416.

Filiform spines similar to the above have been described in association with lichen planus, lichen scrofulosus, the miliary papular syphiloderm, and dermatitis seborrheica.[1] A group of cases has been recently recorded in which lesions of lichen spinulosis developed on the trunk and limbs preceded in each instance for several years by folliculitis decalvans of the scalp. The nature of these cases is as yet undecided.[2]

Etiology.—The cause of the disease is unknown. It occurs chiefly in children, though occasionally adults may be attacked. Adamson[3] suggests a toxic origin.

Pathology.—Adamson[4] summarizes the microscopic findings as follows: "A horny plug distends the follicle at its upper third and extends upward for some distance beyond the level of the epidermis. It is made up of concentric lamellæ, composed of flattened, welded, horny cells. There is acanthosis of the cell-wall. There is an absence of keratohyalin granules, the horny cells of the plug being formed by a process of irregular cornification. No microörganisms were discovered. The hair-bulb is unaltered, but the sebaceous glands are atrophied or absent. There is a very slight increase in amount of cells of connective tissue type at the neck of the follicle and in the neighborhood of the papillary vessels." He concludes from these findings that the disease does not differ essentially from other hyperkeratoses of the pilo-sebaceous follicle, and believes Unna's title, "Keratosis follicularis spinulosa," better describes the findings. On the other hand, Crocker believes that vascular congestion and inflammation occur first, and that the epidermic changes are sequels. Lewandowsky[5] confirms the latter view; but by reason of the suppuration preceding the spine-formation in his case, Adamson excludes it from the category of lichen spinulosus.

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[1] MacLeod, Brit. Jour. Derm., 1908, xx, p. 85.

[2] *Cf.* Little, Brit. Jour. Derm., 1915, xxvii, p. 183. Folliculitis Decalvans et Atrophicans. Ware, ibid., p. 295; Lichen Spinulosis and Folliculitis Decalvans. Beatty and Spears, ibid., p. 331. A case of Folliculosis (Folliculitis?) Decalvans et Lichen Spinulosis. Ormsby, Archiv of Derm., 1920, n. s. vol. i, p. 471; Folliculitis Decalvans and Lichen Spinulosis. Senear, Ibid., 1920, ii, p. 198. Folliculitis Decalvans and Lichen Spinulosis. A clinical combination.

[3] Loc. cit.

[4] Loc. cit.

[5] Archiv, 1905, lxxiii, p. 343.

Diagnosis.—From keratosis pilaris it is distinguished by the slow development, the absence of redness at all stages, and the diffuseness of the eruption of the latter, and, finally, the removal of the epidermic plug in keratosis pilaris removes the entire lesion. The disease should also be distinguished from other follicular keratotic eruptions.

Treatment.—Crocker had success in using alkaline baths with friction, followed by a liniment of soft soap, spirits of wine, and oil of cade, one drachm to the ounce (4. in 30.). Bowen used oil of cade and salicylic acid in glycerite of starch also with success.

Prognosis.—Without treatment, the lesions may last indefinitely, but soon clear up with appropriate therapeutic measures.

KERATOSIS SENILIS (SENILE KERATOMA).

The condition described by Besnier under the above title might properly be called a preëpitheliomatous keratosis, as true epithelioma often begins in such a lesion. Dubreuilh[1] described this condition in a paper read before the International Congress of Dermatology, London, 1896, and Hartzell[2] confirms the clinical description and adds histological findings.

The lesions are seen in people past middle life, more often past sixty years of age, and occur chiefly over the temples and other parts of the face, ears, sides of the neck, and dorsal surfaces of the hands. They are usually flat, moderately elevated, firmly adherent crusts, yellowish-brown or black in color, and when forcibly removed show on their under surface horny prolongations, which fit into the depressions thus disclosed. The surface of the skin is uneven, bleeds readily, is moist and red, and at times superficially ulcerated. The crusts may be greasy, but are usually dry and hard. They commonly present no inflammatory symptoms, but as they are changing their type into true epitheliomata some inflammatory reaction may be noted. In many cases treated by the author several fully developed, deeply situated epitheliomata have been noted as a further stage of the above described condition.

These lesions are often found in association with other changes characteristic of the senile skin, such as atrophy, hyperpigmentation (localized or more or less diffuse), an unusual dryness, with or without scaling, and more or less persistent seborrhoic patches.

Pathology.—Hartzell[3] describes hyperkeratosis, especially about the mouths of the hair-follicles and sweat-ducts, with varying degrees of hypertrophy of the rete. In the early stages the corium is but little altered. In a transitional case cellular infiltration was noted in the corium, consisting of mononuclear leukocytes, plasma- and mast-cells, which had surrounded and punctured a down-growth of the rete. The sebaceous glands were found to be normal, but in all cases the coil-glands and ducts were implicated in the pathological process.

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[1] Annales, 1896, s. iii, vii, p. 1158.

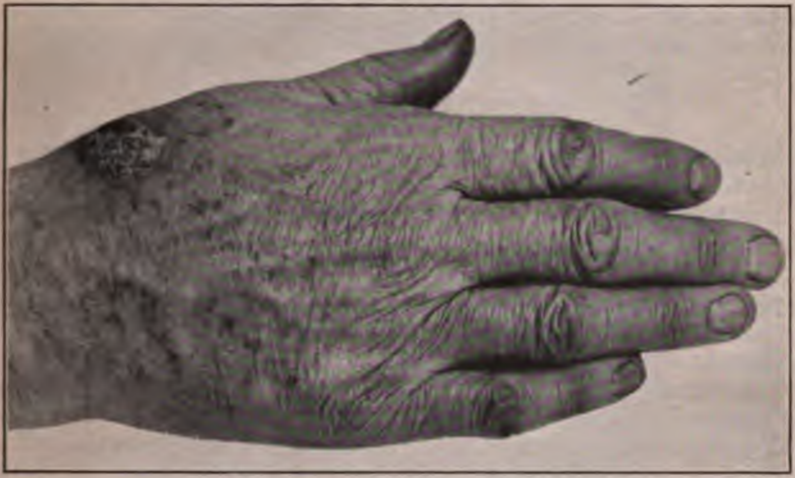
[2] Jour. Cut. Dis., 1903, xxi, p. 393 (with bibliography).

[3] Loc. cit.

Montgomery, D. W.,[1] made similar findings relative to the implication of the coil-glands and ducts.

Fordyce[2] describes the histology of a keratotic patch in a patient sixty years of age, as follows: "The stratum corneum was thickened and showed fairly well-preserved nuclei in its cells; and cornified plugs attached to its under layers distended the mouths of the pilo-sebaceous apparatus. The granular layer was absent, the rete irregular, showing areas of degeneration and vacuolation, the sharp contour of the basal layer being lost. The connective-tissue in the upper corium was degenerated, presenting a homogeneous appearance. The major portion of the vessel-walls was thickened, and there was a mild inflammatory infiltration in the upper corium." In the study of another case of senile keratoma marked degeneration of the epithelial cells was noted, and the connective tissue immediately beneath, as well as on either side of the area, was degenerated and rarefied. The latter condition is considered important as a factor in tumor-formation.

FIG. 144



Senile keratosis becoming malignant.

Sutton,[3] after a histological study, concludes that seborrhoic keratoses should be differentiated into three groups: the keratoid, nevoid, and verrucose types.

Diagnosis.—Lesions such as those described above should be recognized. Physicians, as a rule, attach little importance to them, and many cases are allowed to develop into hopeless epitheliomata before their true nature is recognized. They resemble to a mild degree the senile or sebaceous wart, but the latter usually occurs over the shoulders and back, and is soft and granular, but practically of the same color. The latter is frequently associated with quite marked itching, and but rarely undergoes malignant change.

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[1] Jour. Cut. Dis., 1914, xxxii, p. 6: The Anatomy of a Patch of Seborrhoic Keratosis.

[2] Transactions of the XVII International Congress of Medicine, London, 1913, Sec. xiii, Part 1, p. 4.

[3] Jour. Amer. Med. Assoc., 1915, lxiv, p. 403.

Treatment.—In the early stages of this disorder pomades containing from 1 to 3 per cent. of sulphur and salicylic acid or ammoniated mercury may be employed with advantage. Curettage, followed by moderate cauterization, also is effective. The salicylated plaster-mulls and radiotherapy are exceedingly useful remedial measures. The most effective treatment, however, is with solidified carbon-dioxid. This may be moulded to fit the lesion, and as a rule from one to three moderate applications are sufficient to eradicate the disorder.

Prognosis.—The prognosis is to be formulated with due caution in the case of all patients with multiple lesions of the above described type, on account of the malignant change which may take place.

KERATOSIS FOLLICULARIS.[1]

Synonyms.—Psorospermosis, Darier's disease, Keratosis vegetans (Crocker). Fr., Psorospermose folliculaire végétante.

Keratoses follicularis was first described by Morrow.[2] It was later (1889) independently studied and described by Darier in France and J. C. White, in America. On account of peculiar round bodies discovered on microscopic examination which were originally regarded as parasites the disease was made the subject of intensive study. Although rare a number of examples are recorded each year.[3]

Symptoms.—The disease is characterized by an eruption beginning as papules, which soon become crust covered, and by coalescence produce papillomatous, vegetating, and tumor-like growths. The eruptive manifestations, as a rule, begin about the head and face, and spread over the limbs, the front of the chest, the inguinal and genital regions, and the loins. In one of Bowen's cases the head and hands only were affected.

The early lesions, as described by White and Bowen, are follicular, and are essentially a keratosis of the mouths of follicles. MacLeod[4] found the major portion of the early lesions in his case to be interfollicular. They are firm, pinhead-sized papules, scarcely differing in color from the surrounding integument, which later assume a deeper hue, and, whether flattened or spherical, soon become covered with a grayish or brownish crust, greasy to the touch, and apparently prolonged into depressions beneath, much as the crust of seborrhea sicca of the face is sunk within the orifices of the sebaceous follicles. The papules, as they increase in size and become older, acquire a darker hue, until eventually they are a deep-brown and red, or even purple. Occasionally, scratch-marks and hemorrhagic crusts are interspersed.

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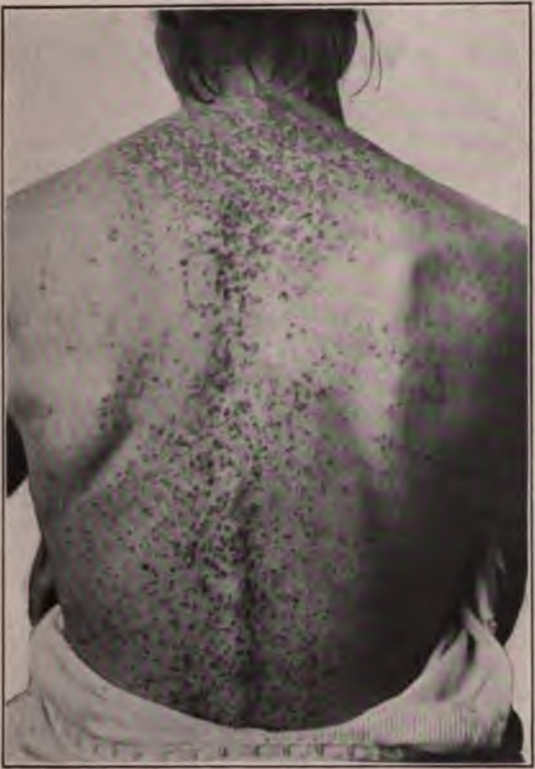
[1] Darier and Thibault. Thèse de Paris, 1889; Annales, July, 1889, x, p. 597. J. C. White, Jour. Cut. Dis., 1889, vii, p. 201, and 1890, viii, p. 13 (histological report by Bowen). Bowen, ibid., 1896, xiv, p. 209 (bibliography), and Annales, 1898, ix, p. 6. Darier, La Pratique Dermatologique, iv, p. 140.

[2] Jour. Cut. Dis., 1886, iv, p. 257.

[3] *Cf.* Little, Brit. Jour. Derm., 1916, xxviii, p. 263. Schur, Jour. Cut. Dis., 1916, xxxiv, p. 807: Report of the Disease in its Early Stage. Sibley, Brit. Jour. Derm., 1918, xxx, p. 36. Case report and histological study. Schalek, Jour. Cut. Dis., 1918, xxxvi, p. 104. Report of a case of Darier's Disease, Improved with Roentgenotherapy.

[4] Brit. Jour. Derm., 1904, xvi, p. 321.

FIG. 145



Keratosis follicularis (Darier's disease.) (Engman and Mook.)

Over the scalp, the symptoms are practically those of the crusting forms of seborrhea, save that there is no tendency to loss of hair. Over the face, the parts chiefly involved are the temples, the inside of the concha of the ears, and the folds about the nose and lips. Here, as over the parts of the trunk named above, form dark, even blackish, strata of dirty oil-crusts, spontaneously shed. Over the backs of the hands and fingers the papules and crusts are less numerous, but the papules are closely set together and do not coalesce. In the palms and soles are numerous almost imperceptible lesions of the same type.

Palmar keratosis is frequently exhibited to a greater or less degree. Trimble[1] describes keratodermia in four out of five cases.

As the disease advances to what has been described as a second stage, the papules coalesce, forming small, dark-brown tumors and papillomatous growths, which involve not only the follicles but also the interfollicular tissues. Many follicles become the sites of superficial ulcers, while the whole of the vegetating mass is bathed in a more or less abundant, fluid, mucopurulent secretion. The subjects of the malady often emit an offensive odor. The vegetating and papillomatous lesions are found chiefly in the axillary and genital regions and behind the ears.

The nails show subungual hyperkeratosis, and the nail-plate exhibits various changes. Small papular lesions have been noted on the mucous membrane in a few cases.

The disease progresses gradually until large portions of the body are covered. Occasionally, exacerbation, with rapidly spreading lesions, occurs, but, as a rule, the course of the affection is slow and the general health of the patient does not seem to suffer, except secondarily from the presence of ulcerating and suppurating lesions of the skin. Bowen[2] called attention to the fact that no cases had developed malignancy, but since that time Wende[3] reported a case terminating in superficial, deep-seated, and papillary epitheliomata.

Etiology.—Little is known definitely regarding the etiology of keratosis follicularis. In the majority of cases recorded the disease began in childhood, and in several cases in early infancy. Fernet and Scheikevitch[4] reported an example occurring in a patient aged sixty-two years. Of the cases collected, the greater number of patients were males. Heredity is to be considered. White's cases were in father and daughter, Boeck's[5] in father and two sons. Ehrmann[6] described the case of a patient whose father he had seen in Janowsky's clinic with the same disorder. Trimble[7] had five cases in three generations, as follows: mother, three children, and one grandchild.

The theory first advanced by Darier, that the disease was due to the presence of psorosperms, has been abandoned. Bowen first demonstrated the fact that these bodies are cell-transformations and not living organisms. Practically all observers, including Darier, now accept this view.

Pathology.—As originally described by Bowen, the lesions are caused by a hyperkeratosis affecting chiefly the sebaceous and hair-follicles. The process is principally confined to the neck of the follicle, but in the later stages it extends to the interfollicular tissues.

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[1] Jour. Amer. Med. Assoc., 1912, lix, p. 604. [2] Loc. cit.

[3] Jour. Cut. Dis., 1908, xxvi, p. 531.

[4] Bull. soc. franç. de dermat. et de syph., 1919, Nos. 5 and 6. Abst. Pract. Med. Series, 1919, vii, p. 100.

[5] Archiv, 1891, xxiii, p. 857.

[6] Verhandl. d. K. K. Gesellschaft d. Aerzte in Wien, October 18, 1901; abstr. Brit. Jour. Derm., 1902, xiv, p. 41.

Loc. cit.

The mouths of the pilo-sebaceous ducts are dilated into funnel-shaped openings and are packed with masses of horny cells produced by the hyperkeratosis. Brocq and several other observers believe that the process is not essentially follicular, but that it may begin outside of the ducts. The rete is usually thickened, and in the later stages of the disease the interpapillary processes are elongated. Mitoses are numerous, and in the lower layers of the rete are found fissures or lacunæ, which are believed by Bowen to be caused by fracture occurring between the soft, pliable cells of the lowest rete and the hard, precociously cornified cells immediately above. In places the pressure of the horny masses may produce thinning and atrophy of the rete. About the borders of the lesions there is an abundant pigment deposit in both the epidermis and corium. The only other change noted in the corium is a small amount of cellular infiltration. The glands of the skin are unaltered. In the tumor-like masses and vegetating lesions, marked proliferation of the rete into the corium occurs, this process being secondary, however, to the keratosis. The disease, therefore, is primarily and essentially one of the epidermis.

The round bodies formerly supposed to be psorosperms are found in the deeper and middle layers of the rete, and at the base of the horny plug filling the follicle. According to Bowen, they are swollen cells containing a nucleus which stains deeply, and which is surrounded by a clear or hyaline ring of protoplasm, outside of which is a zone containing granules of keratohyalin, the whole being surrounded by a homogeneous, glistening membrane, which may possess a double contour. Various modifications of this type are found as the result of irregular keratinization of the cells. In the upper layers, in which the process of cornification is advancing, the keratohyalin gradually disappears; but it may do so irregularly, and, losing its granular appearance, may give rise to appearances closely simulating nuclei and nucleoli. In the upper layers, also, the outer membrane may contract or disappear, leaving an empty space. At the bottom of the horny mass in the follicle the stratum granulosum is frequently absent, and there are seen irregular, shrunken, homogeneous cells, with nuclei which stain but feebly. These cells are the "grains" of Darier, and Bowen believes they are cells which have become cornified without passing through the keratohyalin stage.

Diagnosis.—The disease is to be differentiated from acanthosis nigricans by the more localized distribution of the lesions in the latter disorder, by their special sites of election (axillæ, groins, about the nipples), by the more frequent occurrence of lesions in the mouth in the latter disorder, and, finally, by the finding of a malignant growth internally in acanthosis nigricans. The disease bears a close resemblance to some forms of ichthyosis, but a careful study of the history, the character, and the location of the lesions will usually make the diagnosis clear. From Brooke's keratosis follicularis contagiosa it is difficult to distinguish. They both present keratotic lesions, the one usually, the other always, follicular, and both begin in childhood and occur in several members of a family. There is, however, no evidence of contagiousness in Darier's disease.

Treatment.—The treatment is undetermined. Marked improvement may be obtained, but no complete recovery has been reported, and with a lapse in treatment the unfavorable condition of the patient quickly returns.

The parts are to be well cleansed by shampooings and then dusted with borated, salicylated, and absorbent powders. The French, acting upon the parasitic theory of the nature of the affection, vigorously employ parasiticides, salves containing salicylic acid, sulphur, ichthyol, resorcin, pyrogallol, or iodoform, and even resort to cauterization with zinc-chlorid. The use of ointments containing salicylic acid and sulphur is followed by marked improvement. Liebarthal[1] and Mook[2] found radiotherapy of more value than other methods of treatment. The latter treated four patients by this method. Wise and Parkhurst[3] also report the successful removal of lesions in an extensive case with radiotherapy.

Prognosis.—The prognosis as to recovery from the disorder is unfavorable, but as to a serious termination of the disease is usually good.

KERATOSIS FOLLICULARIS CONTAGIOSA.

H. G. Brooke[1] described under this title a rare and apparently contagious disorder occurring in children and occasionally in adults. He saw six children in one family and three in another affected with the disease, and later saw sporadic cases. Little[5] presented two cases (sisters) before the London Dermatological Society, a third member of the family also being affected. Elliot[6] presented a single case before the New York Dermatological Society. Other similar cases are recorded. The exact nosological position of the disease is not known. It appears to be related to lichen pilaris *seu* spinulosis and Darier's disease, differing from these disorders, however, in its apparent contagiousness, and more particularly from the latter in the absence of the so-called psorosperms, the characteristic cells of Darier's disease.

The lesions in this disorder are symmetrically disposed over the neck, face, shoulders, and extensor surfaces of the arms, less commonly on other areas. They begin as blackish or yellowish-black macules, which develop into deeply pigmented papules, which have projected from their centers comedo-like plugs and small, spike-like filaments. Interspersed are some larger, fleshy papules, and in addition there occur some lesions appearing like acuminate warts. The skin in the area is dry, never greasy, and of a dirty shade. The horny excrescences are attached firmly to the tissue beneath. The eruption is widely distributed and shows no signs of grouping of the lesions.

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[1] Jour. Amer. Med. Assoc., 1904, xliii, p. 242. [2] Jour. Cut. Dis., 1912, xxx, p. 722.

[3] Archiv. of Derm. and Syph., 1920, ii, p. 430. Notes on two unusual cases of Darier's disease.

[4] Internat. Atlas, 1892, vii, Plate 22. [5] Brit. Jour. Derm., 1901, xiii, p. 417.

[6] Jour. Cut. Dis., 1894, xii, p. 362.

According to Unna, who examined sections of these cases, the pathological changes are due, first, to retention, and, second, to the formation of horny plugs at the site of the follicles. The process is essentially a follicular hyperkeratosis, the latter process extending beyond the region of the follicles.

The nature of the disorder is to progress without treatment, but it is readily relieved by local antiparasitics. Brooke recommended hydrarg. iod. in mollin. The disease has also responded readily to local applications of lard saponified with potassium hydroxid.

KERATODERMIA PALMARIS ET PLANTARIS.

Synonyms.—Symmetrical keratodermia of the extremities, Congenital keratoma of the palms and soles, Ichthyosis palmaris et plantaris, Tylosis palmarum et plantarum. Fr., *Kératodermie palmaire et plantaire.*

Definition.—Symmetrical keratosis involving the palms and soles may be congenital or acquired. It is characterized by thickening of the horny layer, and varies as to degree and extent. Krost,[1] in 1880, first described the condition, recording cases in four generations. Unna,[2] in 1883; Hyde,[3] in 1887; Dale,[4] in 1887, and many others have since recorded examples of the disorder.

Symptoms.—The lesions are situated almost exclusively on the palms and soles, though occasionally they may extend over the dorsal surfaces, especially about the joints. Symmetrical involvement of both the palms and soles is the rule, though one or the other may at times be spared. When well developed, there occurs a thick, smooth, yellowish, translucent, dry, hard, horny plate covering the areas above noted. The surface at times, instead of being smooth, may be pitted, and present a worm-eaten appearance; or, as is occasionally seen in the congenital cases, the surface is verrucous and dark-brown or blackish in color. The areas extending over the joints are not so dense and are usually pitted. The palms are uniformly covered, the plate ending abruptly at the margins, usually without areola. The part of the sole not touching the ground in walking is commonly spared.

The acquired cases are usually preceded by hyperidrosis, and often the latter condition is associated with the hyperkeratosis, in which case a sodden epidermis is present, and a pinkish or reddish areola surrounds the horny plate. When fully developed, the arsenical cases are indistinguishable from the above, but early they show individual horny or warty projections scattered over the surface, which later, by multiplication in numbers and fusion, produce a diffuse thickening.

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[1] Inaug. Diss., Heidelberg, 1880; quoted by Abraham, Allbutt and Rolleston's System, vol. ix, 1911, London.

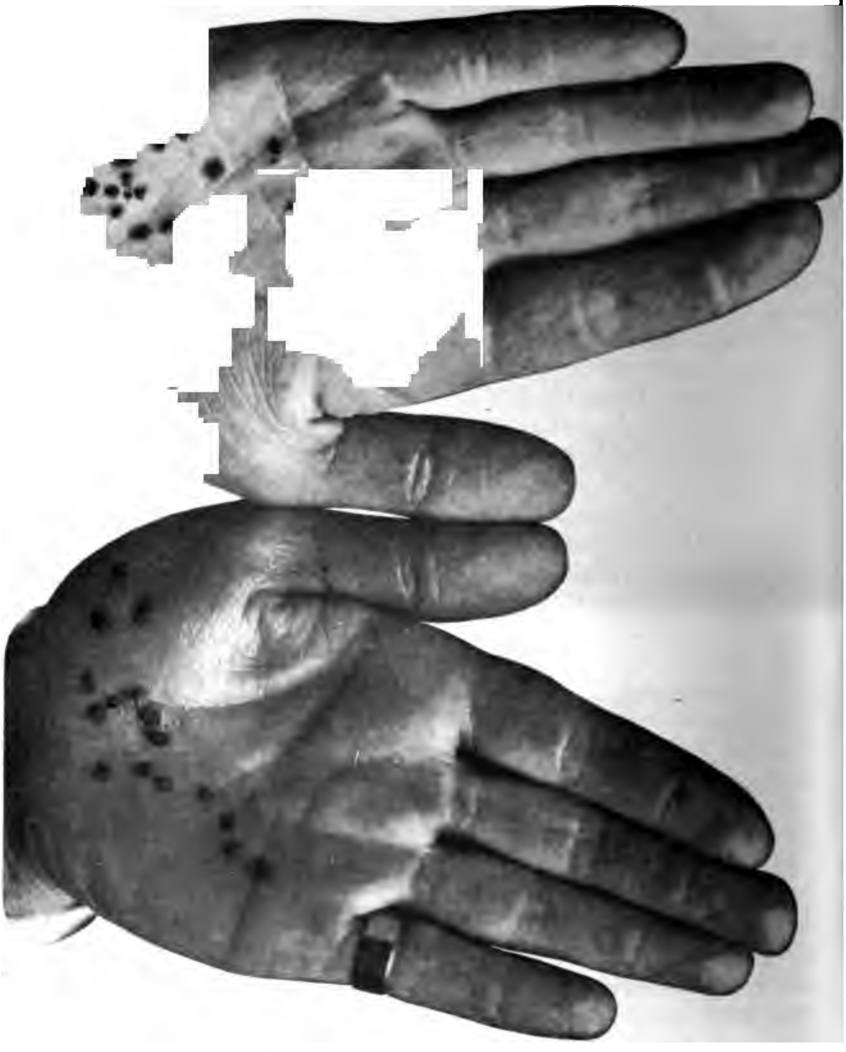
[2] Vierteljahr., 1883, x, p. 231.

[3] Med. News, New York, 1887, p. 416.

[4] Brit. Med. Jour., 1887, ii, p. 718.

The horny plate varies from one-sixteenth to one-eighth of an inch in thickness, and is rigid and inelastic, frequently becoming fissured or broken up into irregular masses. The fissures may extend deeply and involve the corium, inducing painful sensations and interfering with walking. Bassaget[1] describes a mosaic appearance produced by the breaking up of the plate.

Fig. 146



Palmar keratosis, due to arsenic.

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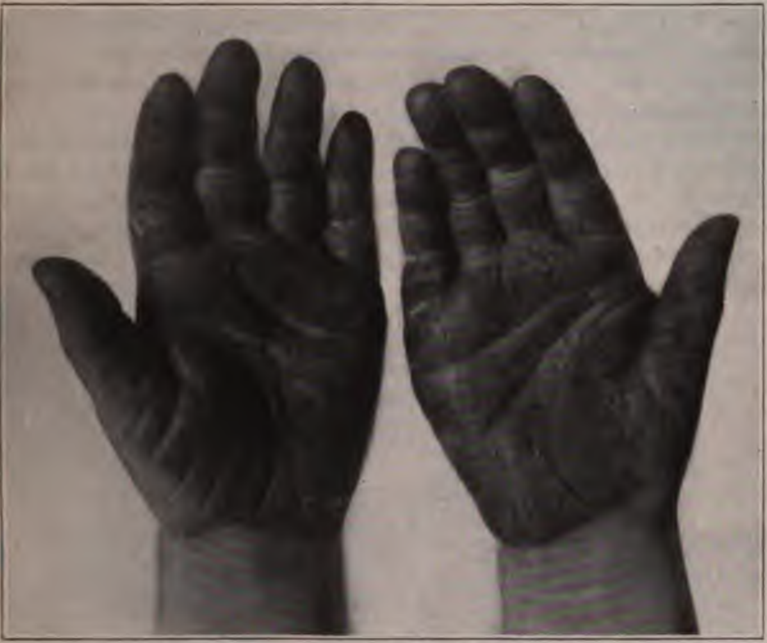
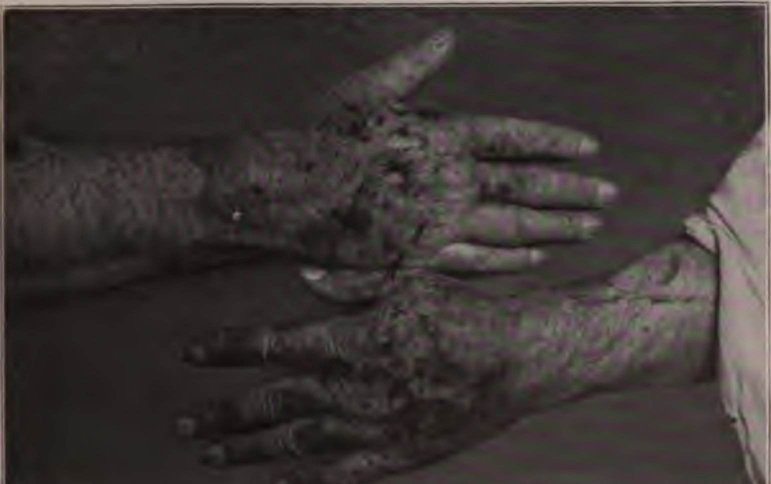
[1] Annales, 1894, v, p. 1356.

FIG. 147



Keratodermia palmaris et plantaris hereditaria.

FIG. 148

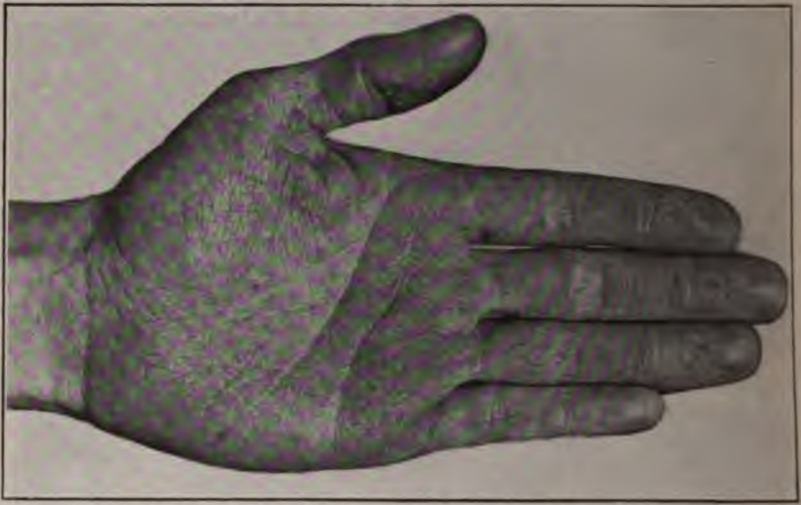


Arsenical keratosis. The illustration shows keratotic patches, similar in type to others situated over the forearms, face, scrotum, and legs, interspersed with hyperpigmentations and several true epitheliomata. In addition, there occurred a symmetrical keratodermia of the palms and soles. The disease was of 25 years' duration, and due to drinking water charged with arsenic.

Localized and diffuse forms associated with and preceded by erythema have been described by Besnier,[1] Brooke,[2] Dubreuilh,[3] and Hartzell.[4] In these cases the keratosis develops on an erythematous base, and further differs from the ordinary types by its ready response to treatment.

A punctate variety has recently been described by Galloway[5] in which the lesions were yellowish-brown flat nodules varying from minute points to the size of a millet seed. They consisted of firm, horny epithelium and in time became depressed below the surrounding surface. Many showed pits. A similar case is recorded by Matsumoto.[6]

Fig. 149



Keratoderma due to arsenic. (Fordyce and MacKee.)

Exceptionally the keratotic process extends beyond the palmar and plantar regions and involves the dorsal surfaces of the hands and feet, knees, elbows and other areas. A marked example is recorded by Vilvandre.[7]

In addition to the above types, a hyperkeratosis of the palms and soles may occur (usually in patches, though occasionally more diffuse) as the result of inflammatory processes in diseases such as eczema, psoriasis, syphilis, lichen planus, and pityriasis rubra pilaris, the special characteristics being described in connection with the diseases mentioned.

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[1] "Keratodermia erythematosa symmetrica," Internat. Atlas selt. Hautkrank., Heft. ii, 1887.

[2] "Erythema Keratodes," Brit. Jour. Derm., 1891, iii, p. 335.

[3] Ibid., 1892, iv, p. 185.

[4] Jour. Cut. Dis., 1911, xxix, p. 499.

[5] Brit. Jour. Derm., 1918, xxx, p. 123. Punctate Keratosis of the Palms of the Hands and Soles of the Feet.

[6] Jour. Cut. Dis., 1918, xxxvi, p. 280. On a New Form of Punctate Keratodermia.

[7] Brit. Jour. Derm., 1918, xxx, p. 202. An unusually extensive case of Symmetrical Keratodermia.

Localized palmar and plantar keratoses induced by intermittent pressure are described in the chapter devoted to Callosities.

Etiology.—Heredity is an important factor. Some cases are both congenital and hereditary. Vörner[1] reports the disease as occurring in four generations, sixteen out of forty members of the family being affected. Zeisler J. and E. P.[2] recorded a family tree in which nineteen members in four generations had the disease. Other instances in which the disease occurred in several members of a family in several generations are reported by Brayton, Decroo, Pasini,[3] White,[4] and many others.

In acquired cases, hyperidrosis is a most important etiological factor, and the many causes of the latter disorder are to be sought. In arsenical cases, the cause is obvious, though here, also, hyperidrosis is a factor. In other cases, none of the above factors can be found, and these are explained by some writers as being due to a neurosis.

Pathology.—Vörner states that all the layers of the skin involved are thickened uniformly, and that cornification is normal in type but excessive. He found no evidence of inflammation. Pasini reports very great increase in the number of sebaceous glands. Other observers describe marked elongation of the interpapillary processes, with dilatation of the bloodvessels and the formation of irregular horny masses over the papillæ.

Diagnosis.—The diagnosis of all forms of keratosis of the palms and soles from eczema is to be made chiefly by reason of the absence in the former of well-marked inflammatory symptoms, of vesicles, and of eczematous patches in other regions of the body. Palmar and plantar syphilides are to be distinguished with great caution. These last may be asymmetrical, especially if of "late" type; may exist where there is often a history of infection or signs of lues; and may frequently ulcerate. They have also well-defined circinate borders; and the lesions are more often multiple and isolated.

Treatment.—Internal treatment is preëminently indicated in all the hyperidrosis cases, and should be employed to meet the indications present. Abstention from tobacco, coffee, tea, and alcoholic stimulants is in general demanded. The arsenical cases may be greatly improved by cessation of the medication. Brocq advises the internal administration of sodium arsenate in large doses; but in this connection it should be remembered that cases are reported in which keratosis of the palms and soles has apparently been produced by a long course of arsenic. The local treatment is by prolonged maceration of the parts, followed by shampooings with sapo mollis, in substance or tincture, followed by salicylated pastes, plasters, or solutions of salicylic acid in collodion. Mercurial plasters and mercuric oleates may also be used with advantage. Potassium hydroxid in 10 to 20 per cent. strength has been applied as a lotion to stimulate the surface. Other formulæ recommended are salicylic acid and calomel, 1 part of each to 20 parts of glycerole of starch; and 1 part each of resorcin, tartaric acid, and salicylic acid to 20 or 30 parts of the salve-basis.

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[1] Archiv, 1901, lvi, p. 3 (bibliography).

[2] Jour. Cut. Dis., 1919, xxxvii, p. 262.

[3] Giorn. ital., 1902, xxxvii, p. 318 (bibliography).

[4] Jour. Cut. Dis., 1912, xxx, p. 276.

In three cases we have obtained very marked improvement with a few applications of the *x*-rays. In one congenital case, that of a girl five years of age, a keratosis involving the entire surface of both palms and soles, and so severe as to prevent extension of the fingers and to interfere with walking, disappeared almost entirely after sixteen treatments, during a period of six months. Eighteen months later the keratosis had not returned. Dohi and Mine[1] report marked improvement in the hands, one treated with *x*-rays, the other with radium, with little or no improvement in the condition of the soles of the same patient treated with salves and plasters.

Prognosis.—In the inherited and congenital cases complete removal of the disorder is accomplished rarely, but by continued treatment the skin can be kept soft and the patient more comfortable. It must not be forgotten that hyperkeratosis of the palms and soles, or of other parts of the body, may terminate in epithelioma.

Mal de Meleda.—Mal de Meleda is an endemic disease occurring on the Island of Meleda in southern Dalmatia. It is a congenital keratosis involving chiefly the palms and soles, but also to a degree other parts. The disease was first described in 1826 by Stulli, and more recently by Hovorka and Ehlers.[2] As described by the above authors, the disease is characterized by a yellowish, waxy, hyperkeratotic thickening of the skin of the palms and soles; the surface being covered with black dots, representing the orifices of the sweat-glands. There is also an ichthyotic thickening of the skin over the dorsal aspect of the wrists and ankles, and occasionally over the elbows and knees. The upper layers of the epidermis are moist and greasy, and exhale a disagreeable odor. In long-standing cases the sensation is diminished, and the skin assumes the appearance of the bark of a tree. Crocker believes the affection to be in part due to intermarriage in a small community.

KÉRATODERMIA BLENNORRHAGICA.[3]

This disorder was originally described by Vidal,[4] in 1893, since which time a number of cases have been recognized. Sequeira[5] was the first to observe the disorder in England, and Simpson[1] recorded the first case in America. The disease is characterized by the formation of horn-like lesions, discrete or diffuse, affecting special areas, and occurring during the course of an attack of gonorrhea.

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[1] Jap. Zeitschr. f. Derm. u. Urolog., 1913, xiii, p. 164.

[2] Archiv, 1897, xl, p. 251; abstr. Brit. Jour. Derm., 1897, ix, p. 416.

[3] Little, Practitioner London, 1916, vol. xcvii, p. 531; Keratodermia Blennorrhagica. Complete description with analysis of previously recorded cases, literature. Haase, Jour. Cut. Dis., 1916, xxxiv, p. 817; Keratosis Blennorrhagica. Full case report; Nathan. Archiv, 1916, vol. cxxi, p. 906. Brown and Hargraves, Brit. Jour. Derm., 1918, xxix, p. 107. Woodward, Lancet, London, February 23, 1918, p. 294; Gonorrheal Keratodermia. Isaac, Brit. Jour. Derm. and Syph., 1920, xxxii, p. 195. A case of Keratodermia Blennorrhagica in a woman.

[4] Soc. de Derm., January 12, 1893.

[5] Brit. Jour. Derm., 1910, xxii, p. 139; Little and Douglas, ibid., 1911, xxiii, p. 360 (case report, with nail involvement).

Symptoms.—The cutaneous manifestations of the disorder begin during the first few weeks after the beginning of the gonorrheal infection, simultaneously with the joint-complication. Debility and cachexia are commonly noted in these cases, the urethritis being a minor feature. Two types of the disease are described: first, a generalized form, in which the lesions may occur on any part of the body, the extremities, however, being the sites of predilection, the legs and forearms being involved more commonly than the thighs and arms, and the scalp and face being rarely attacked; second, a localized form, involving especially the hands and feet, this being the common type.

The lesions in most areas, except on the palms and soles, occur as isolated, but sometimes confluent, horn-like crusts, varying in size from 0.3 to 2 or 3 cc. in diameter. The smaller ones may be semitransparent, yellowish, and waxy; the larger, which are formed by agglutination or çonfluence of the smaller, are of a yellowish-brown hue. Occasionally, vesicles and vesico-pustules precede the above-described lesions. On the palms and soles diffuse thickening occurs, with nodular elevations from the surface. The lesions on the soles are apt to attack the side of the foot, where an arrangement comparable to that of a mountain range on a relief-map is noted. Thick, horny crusts frequently occur beneath the nails, associated with collections of purulent material, which later produce exfoliation of the nail-plate. In Sequeira's case the contour of each foot presented an irregular, horny mass with a nodular surface, the intervening areas being covered with a brownish-yellow, parchment-like, thickened epidermis. The areas occupied by the excrescences were sharply limited by a narrow zone of hyperemia. The nodular masses presented an appearance closely resembling a mountain range on a relief-map, as above described. In Simpson's case a circinate and gyrate configuration was noted. On removal of the excrescences, a moist, pinkish surface is presented. The lesions are superficial, and ulceration does not occur, the sequel being a transient hyperemia or pigmentation. The course of the disease depends upon that of the primary infection, usually subsiding with the subsidence of the disorder. It may recur with recurrence of the latter.

Etiology.—As indicated, the disease occurs only during an attack of gonorrhea, and then with arthritic and other complications. Thus far, the local lesions in the skin have not been found to contain the organism producing the original disorder.

Pathology.—The histological examinations made by various observers agree in the main. The epidermis shows marked thickening. Migrated leukocytes are found in the Malpighian layer, and the interpapillary processes are much elongated. The papillæ are edematous, and show infiltration with plasma-cells, polymorphonuclear leukocytes, and a few eosinophiles. Some observers believe the condition to be a parakeratosis rather than a hyperkeratosis. Gonococci have not been found in the lesions.

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[1] Jour. Amer. Med. Assoc., 1912, lix, p. 607 (case report, with histology and analysis of the literature); Roark, ibid., 1912, lix, p. 2039 (case report).

Diagnosis.—Recognition of the possibility of the occurrence of keratotic lesions during the course of gonorrheal arthritis makes the differentiation of the disorder simple. It is probable that in the past the lesions have been mistaken for those of syphilis. In certain cases psoriasis has been simulated to a marked degree. Adamson[1] has called attention to the resemblance of the disorder to arthropathic psoriasis.

Treatment.—The cutaneous lesions disappear spontaneously with the subsidence of the general infection. In local treatment resorcin and sulphur ointments have been recommended and are of value.

POROKERATOSIS (MIBELLI).[2]

Synonyms.—Hyperkeratosis excentrica (Respighi); Keratoderma excentrica; Hyperkératose figurée centrifuge atrophiante (Respighi).

This rare dermatosis was described independently in the same year (1893) by Mibelli and Respighi in Italy. Hutchins, Gilchrist, and Wende were the first to record cases in America. Since that date many patients affected with the disease have been seen in various parts of the world.

Symptoms.—Porokeratosis is an inherited, chronic, and progressive kerato-atrophoderma, which persists during life. Its elementary lesion is a definitely defined, superficial spot, which is essentially a small keratosic collar, sharply elevated above the general level of the skin like a dike or seam, enclosing a slightly atrophic integument. This dike or limiting wall is in section triangular, having a prismatic outline, with a tortuous contour, producing more or less sharply bordered figures, yellowish-gray or brownish in hue, horny in character, surmounted by a dry, firm, delicate, projecting crust, which seems to spring from a slender furrow running along the summit of the dike, constituted of a spur rising from the horny layer of the epidermis.

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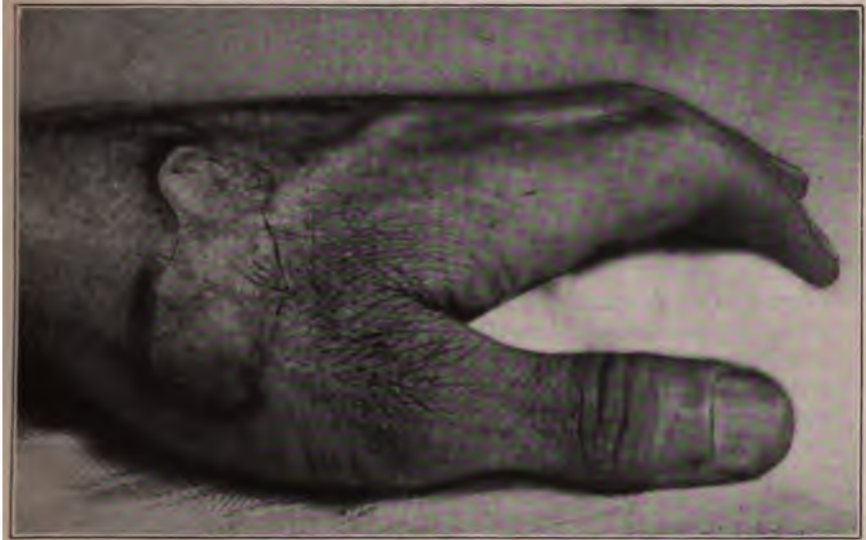
[1] Brit. Jour. Derm. and Syph., 1920, xxxii, p. 183. Keratodermia blennorrhagica: Is it a form of Psoriasis?

[2] Bibliography: Mibelli, Giorn. ital., 1893, iii, p. 313; idem., Monatshefte, xvii, p. 417; idem., Internat. Atlas of Rare Diseases of the Skin, 1893, xxvi; idem., Archiv, 1899, t. xlvii, p. 231 (bibliography to date); idem., Annales, 1905, vi, p. 503; Respighi, Giorn. ital., 1893, iii, p. 356; Hutchins, Jour. Cut. Dis., 1896, xiv, p. 373; Gilchrist, Johns Hopkins Hosp. Bull., 1897, lxxiv; idem., Jour. Cut. Dis., 1899, xvii, p. 149; Wende, ibid., 1898, xvi, p. 505; Ducrey and Respighi, Annales, 1898, iii, s. ix, pp. 609, 734; Galloway, Brit. Jour. Derm., 1901, xiii, pp. 262, 300; Heidingsfeld, Jour. Cut. Dis., 1905, xxiii, p. 29; Brocq and Pautrier, Bull. et Soc., etc., 1907, iii, s. xxiv, p. 651. MacCormac and Pellier, Brit. Jour. Derm., 1918, xxx, p. 197. A case of Porokeratosis Matsumoto, Jour. Cut. Dis., 1918, xxxvi, p. 379. The So-called Porokeratosis (Mibelli) with special reference to its Histopathology. Histological study made in 13 cases. Extensive bibliography.

The elementary lesion is this minute horny spur, firm, dry, pointed, and springing from the orifice of a cutaneous gland, about which forms the minute collar referred to above, constituted of slender, flat-topped, horny lesions, which may fuse. As gradual extension of the disk ensues, the central portion becomes progressively depressed. Where the skin is delicate and covered with lanugo hairs (buttocks, thighs, and legs), the skin of the enclosed area presents only a smooth, slightly atrophic appearance, the lanugo hairs being generally absent. Keratosis in these parts is represented chiefly by the dike or wall; there may be, however, minute projecting horny lesions in the central area, representing the orifices of the cutaneous follicles.

FIG. 150



Porokeratosis, showing the furrow running along the top of the ridge, and the atrophic appearance of the skin within the encircling ridge. (Douglass W. Montgomery.)

On the backs of the hands, where no irritation has been produced by friction, incidental to the trades and occupations of life, the hyperkeratosis is much more pronounced and the collarette larger, firmer, and more elevated than elsewhere, while the central area is remarkable for its dryness.

As the process extends centrifugally, there is persistence until atrophy of the orifices of the pilo-sebaceous pouches and the sweat-pores occurs. Situations where these features are pronounced are: the face, the scrotum, the axillæ, the pubic region, and the hairy scalp.

On the disappearance of the hyperkeratosis, the surface becomes shiny, atrophied in various degrees, and the normal furrows of the skin are somewhat more separated. The hairs are usually (not always) absent, and there is more or less peripheral pigmentation.

In such special regions as the face, for example, where the glands are numerous, the atrophic condition is less distinct, the isolated horny projections often absent, and the peripheral collarette much more slender. The appearance then is that of a delicate atrophy of the skin. In regions where there is external pressure the reverse occurs; for example, over the dorsum of the toes. Here the keratosis is more developed, the atrophy more pronounced. The impression to the eye is then suggestive of a dermato-sclerosis. In the palmar and plantar regions and on the lateral surfaces of the fingers, the appearance produced may be that of a soft corn; but the characteristic collarette suffices to establish the distinction.

Etiology.—The causes of the disease are not known. Eleven cases reported by Gilchrist occurred in four generations of one family. The record of other cases indicates clearly that the disease, or the tendency to it, is inherited, and, as Mibelli points out, often limited to members of a single family.

Inoculation experiments have, with a single exception, been negative. Wende[1] made thirty inoculations, all without result except one. In this, after ten unsuccessful attempts, a lesion was reproduced on the unaffected hand of his patient which clinically and microscopically appeared to be a true lesion of the disease.

Pathology.—The histology of the lesions shows a hyperkeratosis involving the entire area, but accentuated in the raised margin. According to Gilchrist, the hyperkeratosis occurs primarily most frequently around the sweat-pore and intradermic portion of the sweat-duct. The rete in the central area may be markedly decreased in thickness. The corium shows a cellular infiltration beneath the hyperkeratotic area, with dilatation of the blood- and lymph-vessels. In certain cases there occurs dilatation of the whole sweat-apparatus. The hair-follicles and sebaceous glands share in the hyperkeratotic process with the sweat-ducts and pores.

Treatment.—The treatment is unsatisfactory. Electrolysis and excision have been followed by comparatively satisfactory results; though recurrence often takes place, and in most instances the disease persists indefinitely.

ANGIOKERATOMA.

Synonyms.—Keratoangioma, Lymphangiectasis (Colcott Fox). Fr., *Angiokératome Télangiectasie verruqueuse* (Brocq), *Verrue telangiectasique* (Dubreuilh).

Definition.—Angiokeratoma is a rare disorder characterized by pin-head-sized and larger vascular dilatations, upon which are developed later wart-like elevations. The disease occurs usually on the extremities, particularly the fingers and toes, of individuals subject to chilblains. The affection was early described by Cottle,[2] Crocker,[3] and Colcott Fox,[1] under different titles. The first histological study was made by Mibelli,[2] who gave it the name now commonly employed, and his findings were confirmed by Pringle.[3] The latter reported two cases, with histological study and review of previous cases. Cases have been recorded in America by Zeisler,[4] Fordyce,[5] Hyde,[6] Sutton,[7] and others.[8]

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[1] Loc. cit.

[2] St. George's Hosp. Reports, 1877, ix, p. 758.

[3] Diseases of the Skin, 1st ed., p. 512.

Symptoms.—The lesions usually occur on the fingers and toes, but several instances are on record in which the scrotum and other parts were involved, even to a generalized distribution.[9]

The early lesions are pinkish points, which do not entirely disappear on pressure. The characteristic lesions are dull-reddish, purplish, leaden-hued or darker colored nodules, or small tumors, usually having a warty covering, and varying in size from pinhead to split-pea or larger. They may be discrete or closely commingled, forming larger areas. Under the diascope, much of the color may be removed, but there remains in the center a deep-red point. In a case with extensive lesions described by Strumphe[10] diascopic pressure accentuated their appearance. The vascular dilatations may be demonstrated even in those lesions which have assumed a distinctly warty character. The globoid nodules may be smooth and horny or be roughened and prickly. Scaling does not occur. Occasionally, wart-like vascular tumors of larger size have been seen in association with lesions of the usual type.

The sites of predilection are the dorsal surfaces of the fingers and toes, where the lesions usually develop following pernio. At times the varicosities of the vessels are commingled with spots, nodules, and transitional forms. The arrangement of the lesions is irregular, though at times symmetrical, and they may be grouped, or, on the scrotum, have a linear arrangement.

There are no subjective symptoms, and the affection is regarded as of so little moment by some patients that the lesions have only been recognized when examining the skin for relief of another disease.

Etiology.—The patients are commonly young, but a few cases have been reported in middle-aged subjects. Preceding chilblains is a factor in the young patients with lesions on the hands and feet. Varicocele has been noted in the scrotal cases. Sutton[11] believes that changes in the elastin, whereby vascular support is lost, is a factor.

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[1] Westminster Hosp. Reports, 1888, p. 125.

[2] Giorn. ital., fasc. iii, September, 1889; idem., Internat. Atlas of Rare Skin Dis., No. 2, 1889.

[3] Brit. Jour. Derm., 1891, iii, pp. 237, 282, and 309.

[4] Trans. Amer. Derm. Assoc., 1893, p. 54.

[5] Jour. Cut. Dis., 1896, xiv, p. 81.

[6] Dis. of the Skin, 6th ed., 1901, p. 449.

[7] Jour. Amer. Med. Assoc., July 15, 1911, lvii, p. 189 (clinical and histological study, with references to the literature).

[8] *Cf.* Senear, Jour. Cut. Dis., 1917, xxxv, p. 842; Angiokeratoma; a case report. Harris, ibid., p. 849; case report. See also Sibley, Brit. Jour. Derm., 1918, xxx, p. 109, and a discussion of this patient with histological report by Adamson, ibid., p. 85. A Note on Multiple Small Angiomata: Angiokeratoma and Multiple Telangiectases.

[9] Anderson, Brit. Jour. Derm., 1898, x, p. 113.

[10] Archiv, April, 1915.

[11] Loc. cit.

The disease may occur in more than one member of a family. Hartigan[1] records its occurrence in two sisters; Pringle[2] in four members of one family, in three of whom tuberculosis was present. The coexistence of tuberculosis is also reported by Frohwein[3] and others. Dore[4] recorded a case associated with *erythème induré* (*Bazin*), in which, however, a typical chilblain circulation was present. The connection, if any, between the disease and tuberculosis is not clear. Audry[5] believes the tuberculous hypothesis untenable, and thinks the disorder not rare; he claims to have seen sixty to eighty cases. Scheuer[6] believes that the disease is due to an hereditary weakness of the capillaries, which in time leads to acroparesis.

Pathology.—All observers agree that the early changes are vascular, and these are succeeded by epidermal hypertrophy. A uniform histological picture is described and shows oval, elongated or irregular lacunar spaces, filled with blood, occupying the papillary region, partly or completely surrounded by the hypertrophied rete, and lined with connective tissue or epidermal cells. These spaces may be divided into compartments by fibrous septa. Where the circulation has been entirely cut off, the spaces are represented by concentric layers of fibrous tissue, containing blood corpuscles and pigment. The occurrence of the intraepidermal lacunæ is explained by the supposition that the down-growing rete surrounds and cuts off some of the terminal vascular loops of the papillæ. In the subpapillary region there are dilated bloodvessels, vascular spaces, and a leukocytic infiltration. Sutton describes marked changes in the elastin. The fibers were short, fragmentary, and irregularly arranged, apparently furnishing little support to the vessel walls.

The epidermis is hypertrophied, and the stratum corneum commonly shows varying degrees of thickening, depending upon the grade of development of the verrucous element. The rete shows marked hypertrophy, especially about the margins of the nodule, where it dips down deeply into the corium. The transitional layers share in the hypertrophic process.

Diagnosis.—Angiokeratoma is to be distinguished from the superficial lymphangiomata by the early age at which the latter appear, by their location, and by the pseudo-vesiculation which they exhibit, as also by the contents of the lesions. Verrucæ vulgares occur as simple hyperkeratoses, uncomplicated with vascular dilatation, and are not preceded by or associated with chilblains.

Treatment.—The lesions may be removed by electrolysis or galvanothermo cautery. The underlying pernio, if present, should also be appropriately treated.

Prognosis.—The prognosis is favorable, as the lesions may be made to disappear under the treatment above outlined.

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[1] Brit. Jour. Derm., 1905, xvii, p. 456.

[2] Ibid., 1913, xxv, p. 40.

[3] Monatshefte, 1906, xlii, p. 345.

[4] Brit. Jour. Derm., 1903, xv, p. 323.

[5] Annales, 1910, v, s. i., p. 38.

[6] Archiv, 1909, xcviii, p. 251.

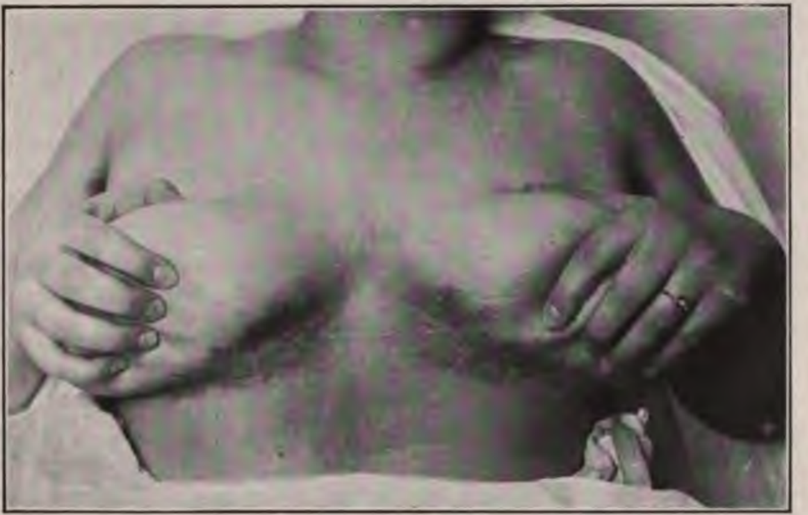
ACANTHOSIS NIGRICANS.

Synonyms.—Keratosis nigricans. Fr., *Dystrophie papillaire et pigmentaire.*

Pollitzer[1] reported the first case of this disorder, occurring in a patient from Unna's clinic. Janowsky[2] reported a similar case, at the same time accepting the title given the disease by Pollitzer. On account of its striking characteristics, a number of cases have been reported in different parts of the world. In America, Dyer,[3] Wende,[4] Klotz,[5] Schalek,[6] C. J. White,[7] and a few others have recorded cases.[8]

Definition.—This rare and striking disease is characterized by hyperpigmentation and papillary hypertrophy, and occurs in two distinct types: the juvenile, which is benign; and the adult, which is frequently malignant.

FIG. 151



Acanthosis nigricans juvenilis.

Symptoms.—The lesions in this disorder occur in certain regions of predilection, and are usually symmetrically distributed. The axillæ, the neck, the external genitals, the groins, the face, the inner aspects of the thighs, the flexor surfaces of the elbows and knees, the umbilical region, the perianal region, the backs of the hands, the breasts, the gluteal region, the hypogastrium, the forearms, the perineum, and the eyelids are the regions affected in order of their frequency, according to Pollitzer.[1] Darier states that at times the entire cutaneous surface may be involved.

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[1] Internat. Atlas for Rare Skin Dis., 1891, Plate 10. [2] Ibid., 1891, Plate 11.

[3] New Orleans Med. and Surg. Jour., 1898, li, p. 201.

[4] Quoted by Pollitzer.

[5] Jour. Cut. Dis., 1911, xxix, p. 436; ibid., 1913, xxxi, p. 306.

[6] Ibid., 1912, xxx, p. 660. [7] Ibid., 1912, xxx, p. 179.

[8] Markley, Trans. Sect. Derm., Amer. Med. Assoc., 1915, p. 165. Acanthosis Nigricans as an Indication of Internal Malignancy; report of a case. Frick, New York Med. Jour., July 31, 1915; case report. Wise, Jour. Cut. Dis., 1918, xxxvi, p. 35. Acanthosis Nigricans following Decapsulation of the Kidneys. Case report, discussion and review of literature. Dubreuilh, Annales, 1919, V Series, vii, p. 67. Pèrin, Bull. Soc. franç. de dermat. et de syph., 1918 and 1919, Nos. 5 and 6, p. 230. Case reports with histological study. Reviewed in Practical Medicine Series, 1919, vii, p. 112.

The color of the patches, when fully developed, is a deep blackish hue, but in some cases shades of yellow and brown are displayed. Often the pigment is somewhat irregularly distributed. The pigmented areas are more or less thickly covered with discrete or agglomerated nodules, papillomatous growths, or vegetating masses. In some cases these lesions are so small and thickly set as to produce a mamelonated appearance; in other cases, large, broad, sessile or pedunculated tumors spring from the pigmented patch. The natural furrows of the skin of the affected part are exaggerated. Often freckles, pigmented warts, seborrhoic warts, or pigmented nevi are scattered over the area. The neck may be surrounded by a band of lesions, dark-grayish or blackish in color, and having a warty surface. In the axillæ and about the cruro-genital folds, diffuse warty prominences frequently occur, and are especially well marked. In the mouth there may be painful sensations, and papillary lesions resembling verruca acuminata have been noted. Condylomatous growths have also been described on the surface of the tongue. Small, discrete, papillary and warty growths have been observed on other parts of the mucous membrane, including the epiglottis and pharynx. In Morris'[2] case masses of small warty growths were found on the vaginal mucous membrane, similar to those seen in the mouth.

Alopecia, complete or partial, of the hairy scalp, and changes in the nails, usually dystrophic in character, may occur.

In the malignant form of the disease the general health of the patient is affected comparatively soon, and toward the end cachexia of grave portent develops. As a rule, the patient rarely survives longer than two years after the beginning of the cutaneous lesions, the average duration being a little more than a year and a half.

In the benign or juvenile form, pigmentary changes and papillary hypertrophy are present, as above noted, but in a milder degree. In these cases the disorder may remain stationary for a long period of time and the general health be little if at all disturbed.

Etiology.—It is now generally accepted that acanthosis nigricans of the adult type signifies a cancerous involvement of one or more of the viscera. The liver, spleen, and other of the abdominal organs have been found involved. In one instance, Spietschka[3] reported the recovery of a patient after the removal of a deciduoma malignum. In three instances the disease has occurred with carcinoma of the female breast. In two of these abdominal metastases were present.

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[1] Jour. Amer. Med. Assoc., October 23, 1909, lxxx, p. 1369. (In this report the author discusses all previously recorded and some unrecorded cases and gives references to the literature.)

[2] Medico-Chirurgical Trans., 1894, lxvii, p. 305.

[3] Archiv, 1898, xliv, p. 247; abstr. Jour. Cut. Dis., 1899, xvii, p. 98.

A case was reported in which the disease was associated with pulmonary carcinoma.[1] In Pollitzer's analysis he concludes that in 80 per cent. of the cases of adult type carcinoma was probably present. This, therefore, is a factor in the etiology of the disorder. The relation of acanthosis nigricans to the malignant visceral disease is difficult to determine. According to some authors, there is an interference with the functions of the abdominal sympathetic (mechanico-nervous theory, Darier), this being a factor in the causation of the cutaneous manifestations.

In the juvenile cases the cause must be sought. In one,[2] the disease developed following a blow in the epigastrium.

More than half of the patients have been women. Infantile cases occur as early as the second year. Several have been reported as occurring before the fourth year.[3] Darier is inclined to the belief that in childhood a teratoma or benign growth may have operated in the manner of a carcinoma of the abdominal region in later life—as an irritant of the great sympathetic, provoking thus the change in the sensitive territory. The disturbance induced by decapsulation of the kidneys appeared to be causative in the case recorded by Wise.

Histology and Pathology.—The major portion of the changes are found in the epidermis, in which there are marked hyperplasia of the rete and increase in pigment deposit. In well-marked cases the hypertrophy of the rete resembles that seen in pigmented nevi. The pigment-granules which occupy the palisade-layer are iron-free. The granular layer is not implicated, except that in certain cases it may be thin. In the corium marked elongation of the papillæ is noted. A very small amount of perivascular cellular infiltration is present.

Diagnosis.—The disease is to be distinguished from seborrhea nigricans, ichthyosis, keratosis follicularis, arsenical pigmentation, Addison's disease, and xeroderma pigmentosum.

The superficial character of the lesions in seborrhea nigricans, the absence of pigmentation to any degree, and the presence of distinct scaling over the body in ichthyosis, readily rule out these disorders. From keratosis follicularis it is distinguished by the involvement particularly of the mucous membranes and the axillary and inguinal regions, areas which are not frequently attacked by the latter disorder. Intense pigmentation is another feature absent in keratosis follicularis. In arsenical pigmentation, the coloring is likely to be more diffuse and is unaccompanied by the peculiar papillomatous and vegetating lesions found in the disease under consideration. Hyperkeratosis may be present and will assist in the diagnosis. In Addison's disease a pigmentation of the mucous membranes is likely to be present, while the papillomatous and other lesions described in acanthosis nigricans do not occur. In xeroderma pigmentosum the presence of atrophic areas and groups of telangiectatic bloodvessels, hyperkeratoses, and possibly beginning epitheliomata, with the marked photophobia usually present, will readily distinguish this disorder.

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[1] Petrini de Galatz, Annales, 1914, v, p. 321.

[2] Pawlof, Monatshefte, 1902, xxxiv, p. 269 (with references).

[3] *Cf.* White, loc. cit. His case occurred in the fourth year, and he reports several others beginning at an earlier age. He has found, however, no case of malignancy under the age of nineteen years.

Treatment.—The treatment is unsatisfactory. Boeck employed extract of suprarenal capsules. White[1] noted much improvement in his juvenile case under thyroid extract medication. Local relief may be obtained by the use of salicylated plasters and ointments.

Prognosis.—In juvenile cases persistence of the disorder is the rule, but a fatal termination is not to be expected. In the adult cases associated with abdominal cancer the outlook is grave.

CALLOSITAS.

Synonyms.—Callosity, Keratoma, Tyloma, Tylosis. Ger., *Verhärtung.*

Definition.—Callosities are acquired, superficial, circumscribed, dirty-white, yellowish-white or darker, flattened, thickened, and horny patches of epidermis, dense in structure, usually insensitive, and occurring for the most part in regions of pressure and friction on the hands and feet.

Symptoms.—Callosities vary in size from that of a finger-nail to that of a section of hen's egg, being at times larger; they occur chiefly upon parts of the integument subjected to long-continued, intermittent pressure, as the hands and feet; also upon parts stretched over osseous prominences, as those over the ischia. Section of a single plaque shows it to be largest at the center and least at the periphery. The lesions are commonly encountered among mechanics, carpenters, and shoemakers; among persons wearing no coverings for the feet or ill-fitting shoes (heel, or ball of foot, or big toes), stockings, or surgical apparatus; among workers in metals, acids, or heated substances; and among musicians (harpists and banjo-players). They are so characteristic of these trades that by their location alone they point, in many cases, to the occupation of the individual who exhibits them; when they are not too large, they are essential to the prosecution of such work. Inflammation may occur in the subjacent tissues and severe dermatitis, lymphangitis, and necrosis result; they may readily serve as foci of cutaneous disease (eczema and psoriasis). They are produced by such external causes as pressure, friction, chemical agents, and heat. They can readily be distinguished from eczematous, psoriasic, and ichthyotic patches, being always limited to the sites of external contact. Plantar warts frequently resemble ordinary callosities in this situation. When the keratotic covering is removed the underlying verrucous growth is revealed. (Cf. section devoted to Verruca plantaris.)

Pathology.– The pathological features of callosities are: marked hypertrophy and compaction of the stratum corneum and thickening of the stratum granulosum; the rete mucosum, on the contrary, being thinned by the pressure. The papillæ are often flattened from the same cause. The corium may exhibit signs of inflammation when the callosity has become a source of irritation.

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[1] Loc. cit.

Treatment.—Callosities require treatment only when they are sources of pain or of discomfort. They may be removed, surgically, by the knife; chemically, by the destructive action of acids or alkalies; rationally, by disuse of the part to an extent sufficient to interfere with the operation of the cause. When painful they may be treated with hot fomentations. A nightly soaking of the callus with warm oil, kept in contact with the thickened epidermis during the night by a compress of flannel saturated with the same substance, will eventually soften the induration. Other methods of treatment advised are: the continuous application of a 10 to 25 per cent. salicylic plaster or mull (Stelwagon); the salicylated collodion-paint recommended for corns; and the scraping away of the outer layers of the epidermis with a dull knife, after soaking in a solution of lactic acid, borax, or weak potassium hydroxid solution, protecting the part afterward with zinc-oxid plaster. After softening with the salicylated plaster or with warm borax-water, the lesions may be frozen with carbon-dioxid snow, when occupying small areas.

CLAVUS.

Synonyms.—Corn. Fr., *Cor, Œil de perdrix;* Ger., *Hühnerauge, Leichdorn.*

Definition.—Corns are circumscribed, cone-shaped hypertrophies of the horny layer of the epidermis, presenting inferiorly a prolongation, which, being pressed from without inward upon the sensitive papillæ of the corium, excites pain in various degrees.

Symptoms.—Corns vary in size from that of a pea to that of a large chestnut, and commonly are described as "hard" or "soft." The former are dense and callous, occurring upon those prominent parts of the foot on which the boot, shoe, or gaiter exercises its greatest pressure. Soft corns develop upon the lateral face of a toe in apposition with another, the lesion originating from pressure through the medium of the neighboring toes. It is softer in consequence of exposure to heat and moisture. Corns are often weather-sensitive, being unusually painful before, during, or after the occurrence of storms. They should not be confounded with gouty or rheumatic deposits below the skin. They are seen occasionally upon the palms of the hands, and when occurring upon the soles of the feet are often the sources of severe distress. They occasionally become infected with pyogenic organisms, leading to suppuration.

Histology.—Corns are composed of superimposed, and often concentrically arranged, layers of epithelium, between which are found at times hemorrhagic extravasations. At the periphery of the corn the corium is unchanged, but at the point where its central cone is pressed into the deeper structures the papillæ are either atrophied or absent. A corn at the periphery exhibits, according to Unna, a thickening of the prickle- and granular-layers. There is a central horny layer, the outermost stratum of which gives evidence of "welding." But the core itself, which is composed of compressed masses of the horny layer conically pointed below, exhibits a flattened ridge-net and papillary body. Often the sweat-pores are preserved, and may be traced running, dilated and with many windings, through the epithelium deeply into the core. The granular layer here disappears, and the general flattening is so great that the margin between the horny cells and the flattened prickle-layer is lost.

Treatment.—Corns, when rationally treated by disuse of the feet, or by the adjustment of properly fitted coverings for the same, will usually fall spontaneously. They are always shed from the feet of the paralyzed. They may be softened by prolonged maceration in water, or by oil, as in the treatment of callosities. Erasion, dissection, and excision may be practised, if demanded by an exigency. Where the sufferer necessarily must continue the use of the foot, the simplest and best treatment is as follows: The part is macerated thoroughly for half an hour with water as hot as can be tolerated. Then the projecting callous portion of the corn is removed by gentle cutting or scraping until, as nearly as may be, the surface is level with the plane of the adjacent skin; after which the part is dried, and the entire surface, both of the seat of the corn and the adjacent integument, is covered completely with many narrow, short, and nicely adjusted strips of rubber-plaster. Burgundy pitch melted and painted over the part may be applied as a substitute for the plaster. When the trifling operation and dressing are complete, the patient should bear firm pressure over the corn without flinching, and walk with comfort. The plaster remains until it separates spontaneously, which is usually in the course of a few days. The corn is then macerated at night with an oil-poultice, as described above, and the dressing afterward reapplied, usually the second time by the patient. Persistence in this course is followed by complete relief, if the coverings of the feet be properly fitted. Carbon-dioxid snow may be employed, as suggested in the management of callosities. Salicylic acid plasters are of service, and the pressure may be removed by applying a perforated felt covering. Mild caustics such as glacial acetic, or trichloracetic acid may be employed and are especially valuable in the soft variety. Radiotherapy is useful in many instances.

CORNU CUTANEUM.[1]

Synonyms. Cutaneous horn, Cornu humanum. Fr., *Corne de la peau;* Ger., *Hauthorn, Hornauswuchs.*

Definition. Cutaneous horns are rare corneous excrescences, greatly varying in shape and size, often resembling the similar growths in the lower animals.

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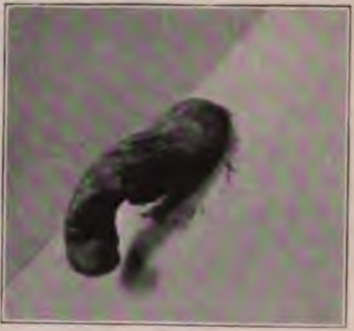
[1] For review of the subject, with bibliography, see Marcuse, Archiv, 1902, lx, p. 197; and Pasini, Giorn. ital., 1902, xxxvii, p. 175.

Symptoms.—Cylindrical, conical, straight or twisted, angular and otherwise, irregularly shaped and sized, corneous eminences, commonly single or more rarely multiple, occasionally project from the scalp, forehead, nose, lips, ears, penis, or extremities. The sites of preference are, in the following order, the scalp, forehead, temples, nose, lower extremities, male genitals, and trunk. Horns are named from their resemblance to the similar appendages in horned cattle, but they differ widely from cattle-horns, which are always implanted upon osseous tissue. Human horns are formed of dense and massed columns of epithelia, often resting upon elongated papillæ. Occasionally, on section, they exhibit the concentric arrangement of the epithelia seen in corns, but, unlike the latter, have reëntrant basal depressions into which the papillæ below penetrate. At times they are implanted in a dilated follicle, in which case the glandular elements participate in their formation. At times, also, they represent a corneus transformation of the epithelia which constitute warts. They are seen in all colors, but are often between a yellowish-brown and a brownish-black, with fissured, wrinkled, or longitudinally grooved exterior, like rough bark. They may be painless or, like other keratoses, become the seat of inflammation in various grades. They may be short or several inches in length. They may be shed spontaneously, never to return, or may shortly reappear. They occasionally develop into epitheliomata.

Brinton[1] has exhibited an anteriorly curved horn one and seven-eighths inches long and three-eighths of an inch in circumference, removed by him from the glans penis of an elderly patient. Many cases are on record of a similar growth in this situation. In the horn growing from the lower lip of an elderly man exhibited in 1886 at Dr. Hyde's clinic, the growth was longitudinally furrowed, and also at somewhat regular intervals transversely seamed, presenting thus the appearance of the joints of the sugar-cane.

Fig. 152



Cornu cutaneum. (Heidingsfeld.)

Etiology.—The cause is, without question, that of the senile wart for most cases; though, as with epithelioma, horns occur in infancy. They have been recognized as starting from a sebaceous cyst. They develop more often after the fortieth year of life, though occurring in infancy, and with slightly greater frequency in women than in men. The author has observed a cutaneous horn developing on the upper lip in a lupus vulgar s scar, which later terminated in epithelioma.

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[1] Jour. Cut. Dis., 1887, v, p. 272.

Pathology.—Pathologically, these hypertrophies are developed first either within a closed atheromatous cyst or from remarkably elongated papillæ of the corium. They are made up of cornified and hypertrophied epidermal cells. According to Unna, they are all papillary and medullated keratomata growing on a circumscribed, warty base. The first stage of their development is characterized by a simultaneous acanthosis and hyperkeratosis, dense epithelial taps reaching toward the corium. In the second stage of horn-formation, the keratosis advances and the acanthosis diminishes. Sets of horny wedges sink downward into the epithelial taps and ridges, fill the spaces between the papillæ, and are capped above by a horny cupola.

Lebert shows that horns develop into epitheliomata in about 12 per cent. of cases. As horns are really metamorphoses of epidermal cells similar in many features to warts, it is not surprising that the two often undergo the change from benign to malignant epithelial growths. In a few cases horns have developed to an appreciable degree on epitheliomata; but under the microscope this horny metamorphosis may be recognized on a smaller scale in a large number of epitheliomata situated on the back of the hands of elderly men who have been farm-laborers, sewer-builders, or workers in contact with earth.

Treatment.—Horns may be removed by extirpation, after softening with alkaline dressings, after which the surface upon which they were implanted should be cauterized thoroughly to prevent return.

Prognosis.—In formulating a prognosis, the possibility of an epitheliomatous sequel should not be forgotten.

VERRUCA.

Synonym.—Wart.

Definition.—Warts are circumscribed papillary growths varying in size and color, presenting a sessile or pedunculated base, and a flat or pointed, horny top.

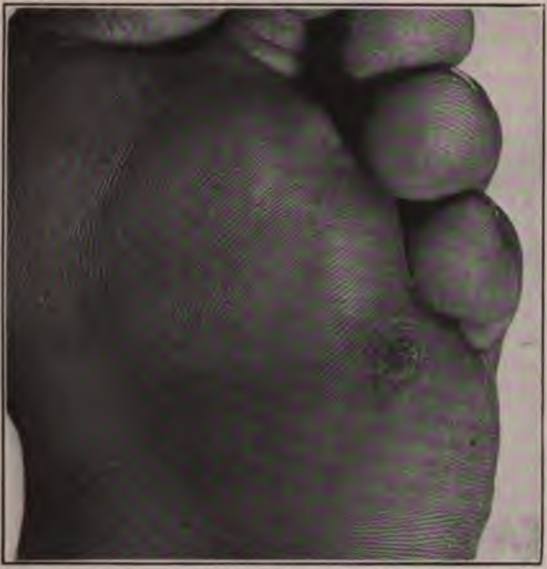
Several varieties are seen, depending on the location of the lesions and the age of the patient.

Verruca vulgaris commonly occurs on the hands of children over the dorsal surface and on the fingers. They frequently develop at the side of the nail and spread under the nail plate. They may occur on the palms, forearms, and other areas and are seen less frequently in adults. They may be few or many—be isolated or grouped—at times a single lesion mother-wart appears and after remaining for some time, is followed by the development of many others. These lesions early are smooth horny elevations and of the normal hue of the skin—older lesions become roughened and the surface presents minute horny papillary projections, and is gray, yellowish, brown or black. By greater hypertrophy of certain papillæ the irregularity of the surface may be exaggerated. They vary in size from a hemp seed to a split pea and may be oval, round or elongated. They are unaccompanied by subjective symptoms.

Verruca digitata occurs chiefly on the scalp, side of the neck and face. These warts present finger-like projections with a horny cap, whereas the other part of the lesion is comparatively soft. The hypertrophied papillæ may be separated down to the base of the lesion.

Verruca filiformis is similar to the digitate variety, but usually more circumscribed. There may be a single soft, slender, thread-like, pointed lesion or several may be grouped. This form occurs chiefly on the eyelid or face.

FIG. 153



Verruca plantaris.

Verruca plantaris (Plantar wart).[1]—These lesions often resemble a callosity and are painful and interfere with walking. They occur most often on points of pressure, but may be scattered irregularly over the entire plantar surface. They are often single but may be numerous, especially in young persons. They are pea-sized or larger, usually covered with a horny plate and surrounded by a keratotic ring. The center is composed of the usual hypertrophied papillæ and is comparatively soft. The nature of the lesion is often not seen until the horny plate is removed by treatment or the scalpel. These lesions are persistent and resistant to treatment.

Verruca plana juvenilis occurs chiefly on the face, neck and dorsa of the hands. The lesions are smooth, flat, and but slightly elevated.

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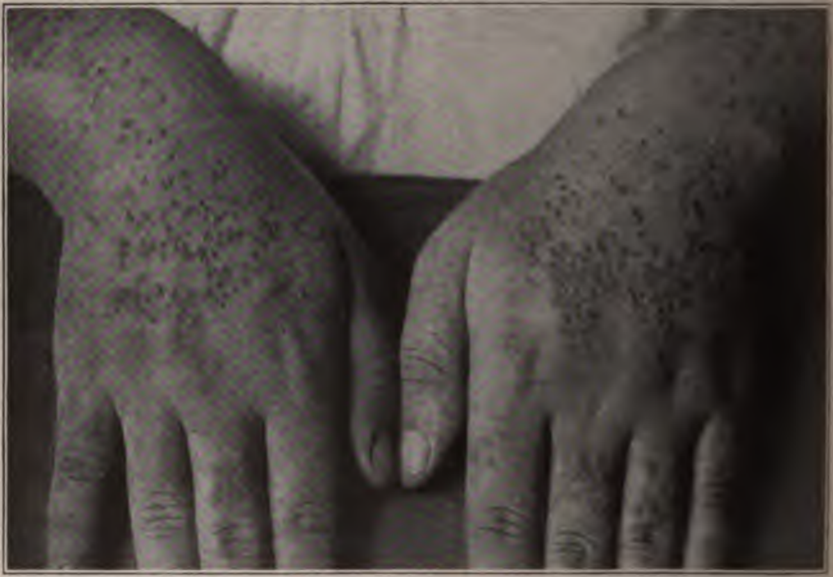
[1] Dubreuilh, Annales, 1895, vi, p. 441; Crocker, 1903, p. 575; Bowen, Boston Med. and Surg. Jour., 1907, clvii, p. 781, and Ibid., clxv, p. 937; Sutton, Jour. Cut. Dis., 1909, xxvii, p. 155, and Amer. Jour. Med. Sc., July, 1912, p. 71; Hardaway, Jour. Cut. Dis. 1906, xxiv, p. 127.

They vary in size from a pinhead to a small split pea or larger. They are irregular in outline and frequently resemble papules of lichen planus. They may be irregularly disseminated or grouped, or formed in lines. They may be of the normal color of the skin or present a white, pink or yellowish-brown hue. Although they usually occur in young subjects, examples are seen occasionally in older people.

Verruca senilis vel plana (*Verruca seborrhieca, Keratosis pigmentosa*).—These lesions occur in elderly people and are seen chiefly on the back and arms, less commonly on the chest and face. They are pea to large coin sized, flat, slightly elevated, and are yellow, brown or black, and present a granular surface. The friable, greasy crust is easily removable from the surface, revealing a red, granular or slightly papillomatous base. They are frequently accompanied by itching, which may be severe.

FIG. 154



Verruca plana juvenilis.

Etiology.—In all varieties of warts age is the predisposing factor. Trauma, in the case of plantar warts, may be considered a predisposing factor. The clinical history of warts strongly suggests an infection. They appear to be auto-inoculable and slightly contagious. Jadassohn[1] produced warty lesions in 33 instances out of 74 inoculations in 6 adult patients, the incubation period being from seven weeks to three months. Similar observations have been made by other observers. An important contribution has recently been made by Wile and Kingery,[1] who produced typical lesions of verruca vulgaris both clinically and histologically by injecting intracutaneously a sterile filtrate of wart material. These observers have since produced lesions in the second generation (personal communication). The incubation period in these cases was four to seven weeks.

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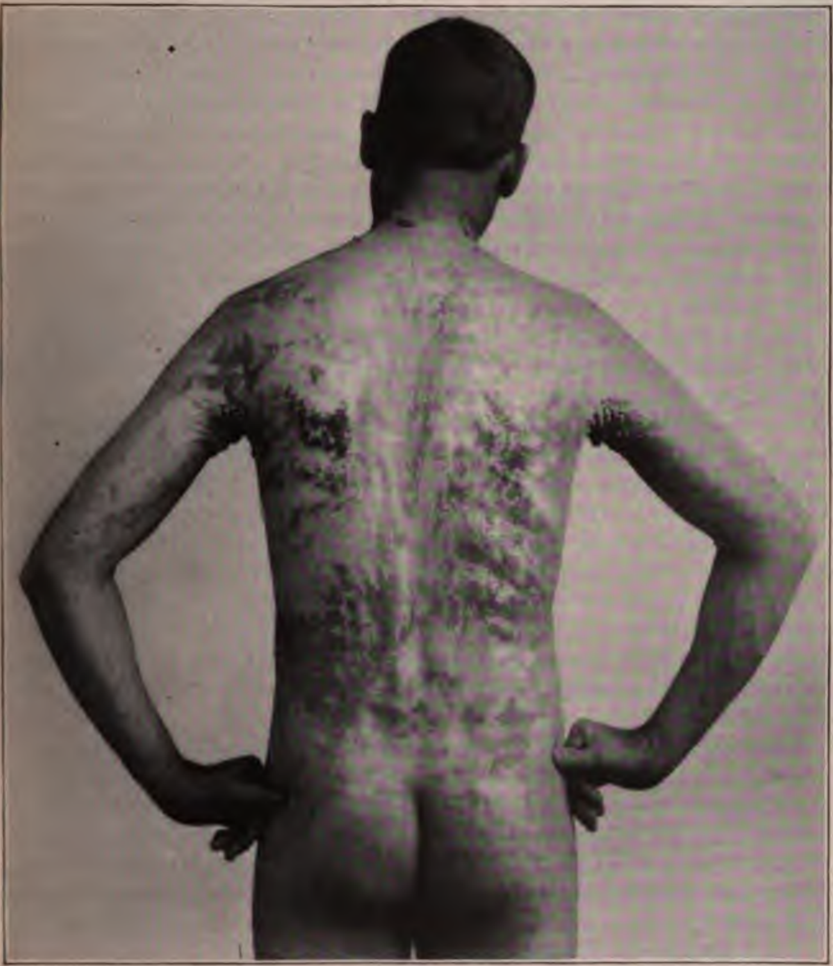
[1] Verband. der v. deutschen Cong., 1896, p. 497 (bibliography).

FIG. 155



Congenital warts.

Diagnosis.—Ordinary warts on the hands of children are readily recognized. The plane juvenile type may be mistaken for lichen planus. The rare occurrence of the latter on the face, the bluish-red or violaceous color of the papules and the accompanying itching serve to distinguish the latter. Plantar warts may be mistaken for callosities but the painful character of the former, together with the typical appearance on removal of the horny covering readily distinguish the two. The lesions of molluscum contagiosum are distinguished by their waxy white appearance together with the small central opening situated on their surface.

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[1] Jour. Amer. Med. Assoc., 1919, vol. lxxiii, p. 970; bibliography and report of results of other investigators.

Pathology.—The chief changes noted histologically occur in the epidermis. The stratum corneum is greatly thickened. There is both hyperkeratosis and parakeratosis, as many cells still contain nuclei. The rete is thickened and marked down growth of the rete pegs (acanthosis) occurs. Mitoses are noted throughout its depth particularly near the basal layer. The papillæ are elongated in proportion to down growth of the rete pegs, but otherwise are normal. Some cellular infiltration is present in the corium, but is not a prominent feature. Some difference of opinion has existed as to the starting point of the process. Wile and Kingery[1] found the earliest change to be a localized acanthosis confirming the earliest observations of Unna and Auspitz.

In the plane variety the chief change is in the rete, there being but little thickening of the stratum corneum. They present the same picture as the common variety in its early stage. Bowen[2] found peculiar epithelial cell degeneration in certain cells of the rete in plantar warts. Pollitzer[3] describes the histology of seborrheic warts as follows: The seborrheic wart is characterized histologically by slight thickening of the stratum corneum and considerable hypertrophy of the rete. In the papillary and subpapillary layer were found epithelioid cells arranged in groups and lines, together with an infiltration of fat affecting the coil-gland epithelium, the middle and papillary layers of the corium, and the epithelium of the rete Malpighii. In verruca acuminata there is little tendency to increased cornification. The rete and papillary bodies are remarkably hypertrophied, and a moderate cellular infiltration is present in the corium.

Treatment.—Internal treatment is seldom necessary, but in extensive cases it may be employed. Arsenic[4] is often of value and may be used for the ordinary or plane type. Crocker recommends magnesium sulphate in 2-grain doses three times daily. White[5] found protiodid of mercury (grs. ¼) three times daily of value in the plane variety. Dilute nitrohydrochloric acid, and thyroid extract have been successfully used.

Local Treatment.—Warts may be removed by the local application of salicylic acid, glacial acetic acid, trichloracetic acid, chromic acid and other caustics. Freezing with carbon dioxid snow is often valuable. Electrolysis is preferable in some instances. Radium and *x*-rays are also useful.

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[1] Loc. cit. [2] Loc. cit. [3] Brit. Jour. Derm., 1890, ii, p. 199.

[4] Crocker, Diseases of the Skin, 1903, 3d Ed., p. 580; Müller, Pullin, Sympson (quoted by Crocker); Stelwagon, Diseases of the Skin, 8th Ed., p. 583.

[5] Jour. Cut. Dis., 1915, xxxiii, p. 738.

In the ordinary variety on the hands when few in number, freezing the lesions with carbon dioxid snow after softening them for twenty-four hours with salicylic acid plaster is efficient. Painting with a suspension of salicylic acid 1 dram (4) in collodion, 1 ounce (30) is also valuable. When numerous, and especially about the nail, *x*-rays may be successfully employed. For the lesions in the scalp glacial acetic acid applied first and followed after a week or ten days, when the major portion of the lesion has disappeared, by carbon dioxid snow is usually successful. For multiple lesions on the face and neck *x*-rays may be used; when few, glacial acetic or trichloracetic acid, freezing, or electrolysis will be successful. For plane lesions of the juvenile type Vleminckx's solution has been successful; when extensive, *x*-rays give prompt results. The plantar wart is very resistant to treatment. It may be curetted and the base cauterized with some of the above-mentioned agents or with phenol. Here, again, *x*-rays rarely fail.[1] In the seborrheic type ointments containing sulphur and salicylic acid may be used first and followed by freezing, trichloracetic acid[2] or *x*-rays. Large lesions may be treated with high frequency spark or be excised.

Prognosis.—All varieties of warts respond to treatment. Some are more resistant than others. At times the common variety disappears spontaneously. In rare instances the seborrheic type undergoes malignant transformation.

Verruca Acuminata.—**Synonyms.**—Condyloma acuminata; Moist or Venereal wart, Fig-wart. Ger., *Spitzenwarze, Venerische Warze, Feigwarze, Spitzenkondylom.*

Definition.—Verruca acuminata are papillary growths occurring as filiform projections or vegetating masses often covered with a secretion which emits an offensive odor.

Symptoms.—The lesions in this disorder occur as a rule in the genital region and the particular sites in males are upon the glans, around the frenum and over the prepuce; in women about the clitoris, labia, ostium vaginæ, over the perineum and about the anus. Less often they occur in the axillæ, about the umbilicus and the interdigital regions of the toes.

They may be small, filiform projections and few in number, or numerous and crowded into a mass presenting a papilliform or vegetating appearance, and vary in size up to that of an egg or larger. They may be sessile or pedunculated. Their appearance has been described as cock's-comb-like or cauliflower-like and when occurring in moist situations they are covered with a puriform secretion having an offensive odor. At times this dries and forms a dry, brown crust, beneath which the offensive secretion is retained. They increase in size by the development of new lesions at the periphery. In color they are brown or pink, they often develop rapidly and tend to remain indefinitely. In dry locations they are uncolored and dry and their growth is likely to be limited. At times when luxuriantly developed the mass becomes flattened through pressure and appears like condylomata lata. In pregnant women they often grow rapidly and attain a large size, but disappear after parturition. In the colored race excessive growth is at times observed.

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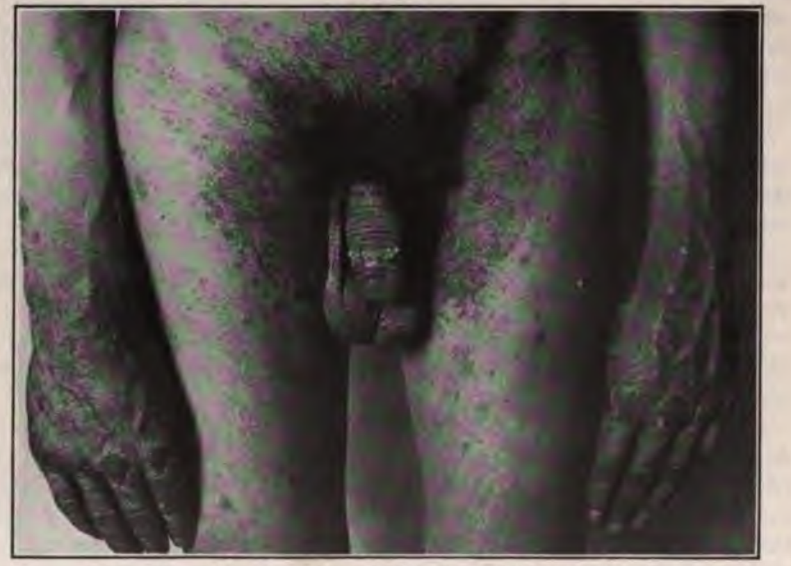
[1] Hazen and Eichenlaub, Jour. Amer. Med. Assoc., 1920, lxxiv, p. 1311. The Roentgen-ray Treatment of Verruca Plantaris.

[2] Davis, Jour. Cut. Dis., 1915, xxxiii, p. 691, References.

Etiology.—These lesions are auto-inoculable and contagious. The causative microörganism has not been identified. They occur in association with gonorrhea and other irritating discharges in the genital region.

Pathology.—The histology of verruca acuminata differs from the ordinary wart in many particulars. The stratum corneum is but slightly thickened, if at all, and in some instances it is thinner than normal. The rete is greatly hypertrophied and shows abundant mitoses. The papillæ are greatly enlarged, often branched, and contain dilated bloodvessels and lymph spaces. In the corium there is a cellular infiltration composed of connective-tissue cells, polymorphonuclear leukocytes, plasma and mast-cells.

Fig. 156



Dermatitis of hands and inguinal regions with venereal warts.

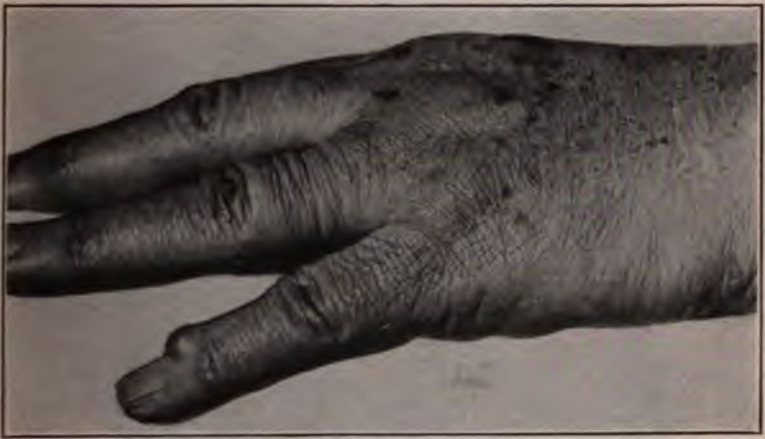
Diagnosis.—They are to be differentiated from condylomata by the indurated bases, flat top and associated symptoms of syphilis in the latter disorder.

Treatment.—Cleanliness is essential. Thorough bathing with a solution of boric acid, potassium permanganate, mercuric chlorid or phenol, followed by the application of a dusting powder of boric acid, alum, resorcin, salicylic acid (1 gram in 30 of lycopodium), or mercurous chlorid (4 grams in 30 grams of talcum) may be employed. Small lesions may be painted with tannin (4 grams in alcohol 90 grams), or a solution of liquor plumbi subacetatis, or liquor ferri chlorid, or touched with formalin. In some instances the cauterizing agents mentioned in the treatment of common warts may be employed. Radiotherapy is valuable; extensive growths require surgical removal.[1]

Prognosis.—The lesions may be removed by some method of treatment. Spontaneous disappearance seldom occurs except in pregnant women following parturition. They have no sequelæ and even when markedly developed leave but little disfigurement after removal.

Synovial Lesions of the Skin.—These cutaneous lesions possess importance from a diagnostic point of view. We have observed them in several individuals in whom the exact nature of the disorder had not been understood. They occur in the form of wart-like projections from the skin, pseudo-vesicles, and bullæ, always over the site of bursæ connected with tendons traversing the small articulations of the hand and foot. They are seen over the metatarso-phalangeal articulations; and in the hand most frequently over the dorsal face of the articulation between the distal and adjacent phalanges of the index-finger and thumb. The first form is that of a roundish, corneous, pea-sized wart, with a yellowish center, of long duration, usually insensitive unless roughly handled; or the lesion may be smooth, bluish-red in color, and present pseudo-fluctuation. Others are globoid or conical elevations covered with crusts. When punctured, a syrupy, yellowish, or grumous fluid exudes, which continues to form after repeated puncture; this being the most characteristic feature of the disorder. Subjective symptoms occur chiefly when the contents of the cystic cavity produce distention. Pain of different character then ensues, which is relieved by partial or complete evacuation of the sac.

Fig. 157



Synovial lesion of the skin.

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[1] Creadick, Condylomata acuminata, Jour. Amer. Med. Assoc., 1920, lxxv, p. 1057. Recommends surgical removal.

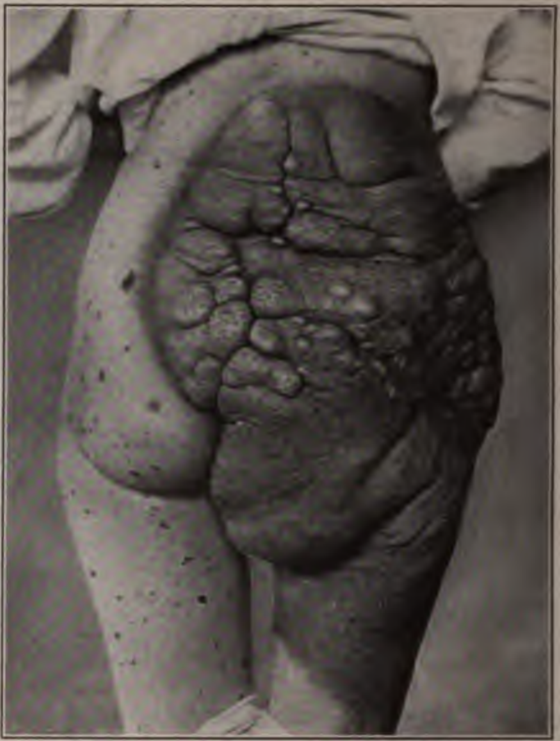
Treatment.—Surgical removal may be practised, though recurrenc
frequently happens. We have treated several patients successfull
with *x*-rays, and one with electrolysis. Lingenfelter[1] had success wit
x-rays.

NEVUS PIGMENTOSUS.[2]

Synonyms.—Pigmentary mole, Nevus spilus. Ger., *Fleckenma*
Linsenmal; Fr., *Tache pigmentaire.*

Definition.—Pigmented moles are congenital, circumscribed accumu
lations of pigment in the skin developing with or without other tegu
mentary changes.

FIG. 158



Nevus lipomatodes. (B. F. Lounsbury.)
(Patient seen in consultation and photographed by the author.)

These abnormal congenital pigmentations of the skin vary in co
from a light yellow or chocolate-brown to a blackish hue, and th
may be single, or be multiple and very numerous. They vary in s
from that of a pinhead to that of the palm, and are flat, oval, or circu
in contour; occasionally, they are so irregularly shaped as to presen

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[1] Jour. Cut. Dis., 1913, xxxi, p. 647.

[2] For studies of the different forms of nevi and full bibliography, see Möller, Arc
1902, lxii, pp. 55 and 371; and Riecke, Ibid., 1903, lxx, p. 65.

fanciful resemblance to lower animals, whence the popular belief as to their origin in maternal impressions. They usually appear during the first year of life, but may be delayed until puberty or later. During the first few years they gradually increase in size, with no elevation. At puberty they begin to project from the cutaneous surface. They occur in both sexes, and in all regions of the skin, but especially upon the face, neck, trunk, thighs, buttocks and external genitals. The so-called "white moles" are similar to the above except that the pigmentation is slight or apparently wanting. In certain cases these may become extensive and have a corrugated, cerebelliform surface (Crocker).

The term *Nevus spilus* is applied to those pigmentations which occur in a smooth and otherwise unaltered skin, and which in later life sometimes become mammillated and present a growth of poorly developed hair; *Nevus pilosus*, to those surmounted by a growth of shorter or longer, stiff or downy, dark or light-colored hair; *Nevus verrucosus*, to those which are warty, soft or hard, furrowed or smooth, accompanied by hypertrophy of the papillæ, and which often present a growth of hair; *Nevus papillomatosus*, to that variety covered with soft papillary growths, this variety having frequently an offensive secretion; *Nevus lipomatodes*, to that variety in which both fatty tumors and diffuse fatty infiltrations occur as congenital defects in association with nevi of other varieties. A remarkable case of this type was described by Hyde.[1] Other cases have been described by Jackson,[2] Howard Fox,[3] Little[4] and others.

Nevi of the above types may be symmetrically or asymmetrically developed upon the body surface and be limited or have a widespread distribution.

Giant Nevus.—Under this heading those nevi which affect extensive areas of the cutaneous surface are grouped. These frequently occupy the so-called "bathing trunk" region and are described as being of this type.[5] This affection may involve a segment of the entire trunk. In certain cases of giant nevus the anomaly in the skin closely resembles the pelt of an animal. Interspersed among the pigmented and hairy regions verrucous lesions and mammillated patches occur. In a case of this type reported by Anthony[6] there were many hundreds of pea to dollar-sized moles associated with the above type of lesions.

Linear Nevus.—Linear nevi may be presented clinically as hyperpigmentations, or as warty growths arranged in streaks, and patches, the latter being the more common variety. In some reported cases the arrangement has simulated that of herpes zoster. In the case reported

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[1] Jour. Cut. Dis., 1885, iii, p. 193, literature.

[2] Ibid., 1895, xiii, p. 66.

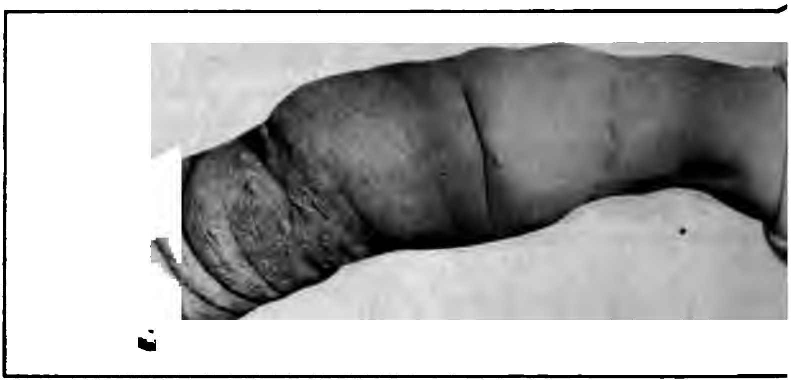
[3] Ibid., 1912, xxx, p. 484.

[4] Brit. Jour. Derm., 1919, xxii, p. 388.

[5] Howard Fox, Jour. Amer. Med. Assoc., April 20, 1912, lviii, p. 1190: A Case of Extensive Pigmented and Hairy Nevus, "Bathing Trunk" Type, with Genital Tumors (with review of 25 cases of similar type.

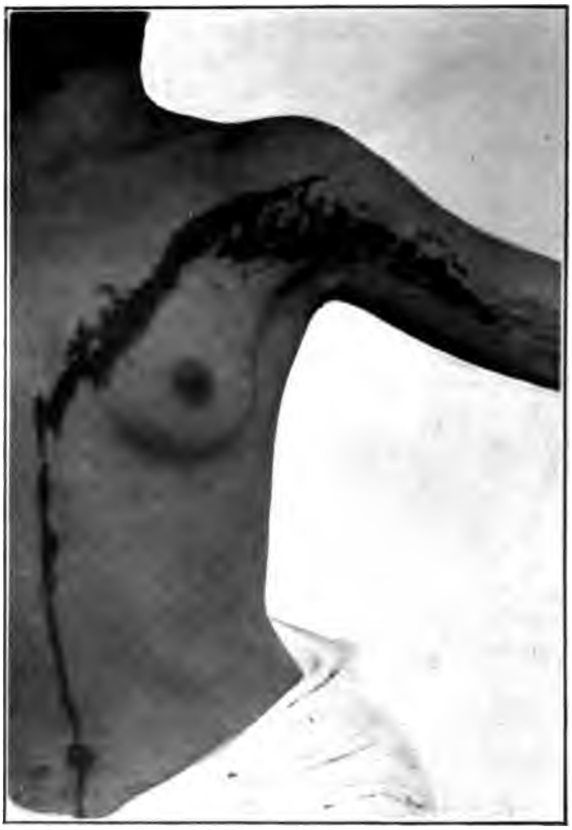
[6] Jour. Cut. Dis., 1909, xxvii, p. 471.

FIG. 159



Giant nevus in a man aged sixty years.

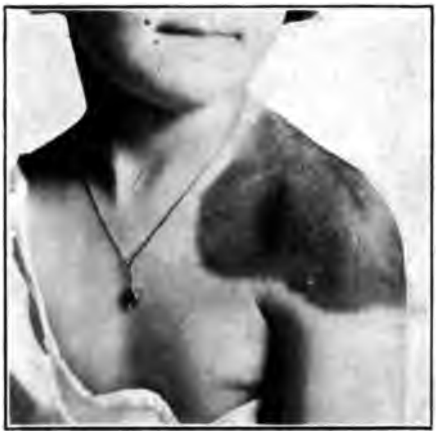
FIG. 160



Verrucous nevus.

by Hyde[1] there were multiple unilateral pigmentary nevi distributed over the left side of the trunk, along the course of the intercostal nerves, and in such a manner as strongly to suggest their correspondence to the lesions of zoster of the same region. De Amicis[2] had previously reported a somewhat similar case. In this type of nevus the warty growths extend in bands and streaks of varying widths vertically on the extremities and horizontally on the body. The warty excrescences vary in size from that of a millet-seed to a pea or larger, and are brownish or yellowish-brown or blackish in color, hard, and, when occurring in patches, are frequently fissured. Streaks and curves of pigmentation may be associated with these. In certain other cases the entire lesion consists only of hypertrophy of the pigmentary elements of the skin.

Fig. 161



Nevus pigmentosus et pilosus. (Morrow.)

Instead of being warty, the lesions may be papular, and in certain instances strongly resemble those of lichen planus. Dore[3] recorded a case in which the lesions resembled those of lichen ruber moniliformis. In a few instances, acneform lesions have occurred in association with linear nevi, presenting comedone-like lesions projecting from the mouths of follicles. In Selhorst's case[4] folliculitis and perifolliculitis, producing ulcers followed by scar-formation, occurred. Thibierge,[5] Davis,[6] Sequeira,[7] da Costa,[8] and others have reported cases of this type. In Davis' case the black dots or comedone-like lesions were discovered to be horny plugs in the mouths of spherical crypts. Okamura[9] noted comedones, acne-like lesions and scars in a nevus unius lateralis.

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[1] Chicago Med. Jour. and Exam., 1877, xxxv, p. 377.

[2] Lo Sperimentale, 1876.

[3] Brit. Jour. Derm., 1910, xxii, p. 165.

[4] Ibid., 1896, viii, p. 419.

[5] Annales, 1896, s. iii, vii, p. 1298.

[6] Brit. Jour. Derm., 1910, xxii, p. 130.

[7] Ibid., 1912, xxiv, p. 76.

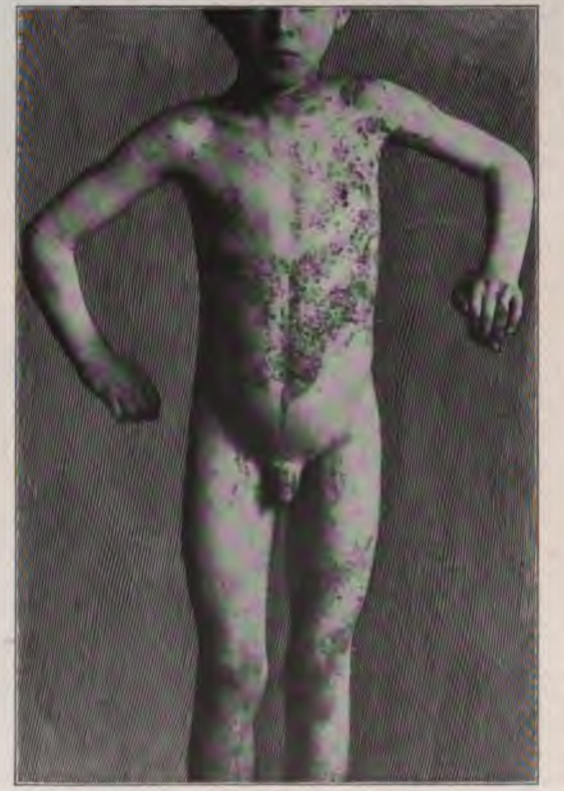
[8] Abstr. Brit. Jour. Derm., 1897, ix, p. 207.

[9] Jap. Zeitschrift für Derm. und Urologie, 1914, xxiv, p. 475; abstr., Jour. Cut. Dis., 1915, xxxiii, p. 66.

White[1] described a nevus presenting a crateriform and sieve-like appearance induced by a mass of widely dilated follicles. Scars were present from preceding infection.

The linear warty growths that extend in lines or bands, often cover the whole length of an extremity and when limited to one side of the body are now recognized by all to be nevi (nevus unius lateralis).

FIG. 162



Nevus linearis.

These formerly were described as ichthyosis hystrix, nevus nervosus, ichthyosis cornea, ichthyosis linearis neuropathica and papilloma neuropathicum unilaterale.

Mixed types occur in which vascular nevi are interspersed. Adamson[2] recorded a linear nevus with verrucous and vascular lesions, and referred to a Baretta model[3] described as *vascular nevus verrucosus.* Morris and Dore[4] described a case with follicular papules, slightly lowish in color, with dotted, depressed centers, which represents a us of the sebaceous-gland type.

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[1] Jour. Cut. Dis., 1914, xxxii, p. 187. Nevus Follicularis Keratosus.

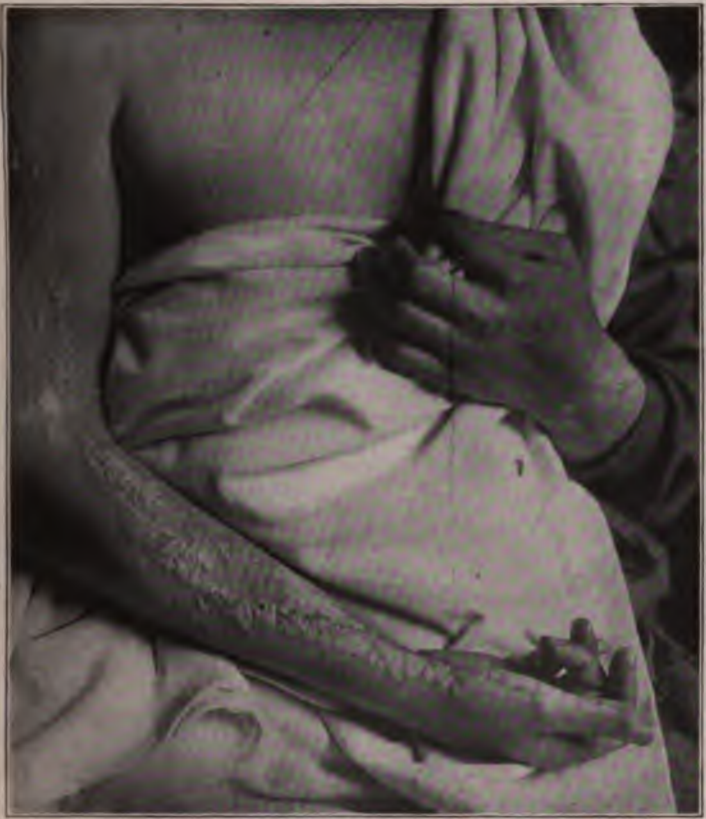
[2] Brit. Jour. Derm., 1910, xxii, p. 263.

[3] Plate XLIV, p. 259, Baretta model 1772, from the patient of Gaucher, St. Louis Atlas.

[4] Brit. Jour. Derm., 1912, xxiv, p. 27.

ccasionally, the mucous membranes are involved. Cases are rded in which the tongue and other portions of the oral mucosa, nasal mucous membrane, and the mucous membrane of the vagina e been invaded.[1]

FIG. 163



Nevus unius lateralis.

In most of the types of nevi above described, gradual progression curs up to a certain point, at which time the lesions remain stationary r the remainder of the patient's life. In a very few instances, sponneous disappearance has occurred. In certain other cases, infection th pyogenic organisms has produced destruction of the major portion the growth. In a case described by Adamson[2] a sclerosing effect curred in a pigmented vascular nevus without the intercurrence of

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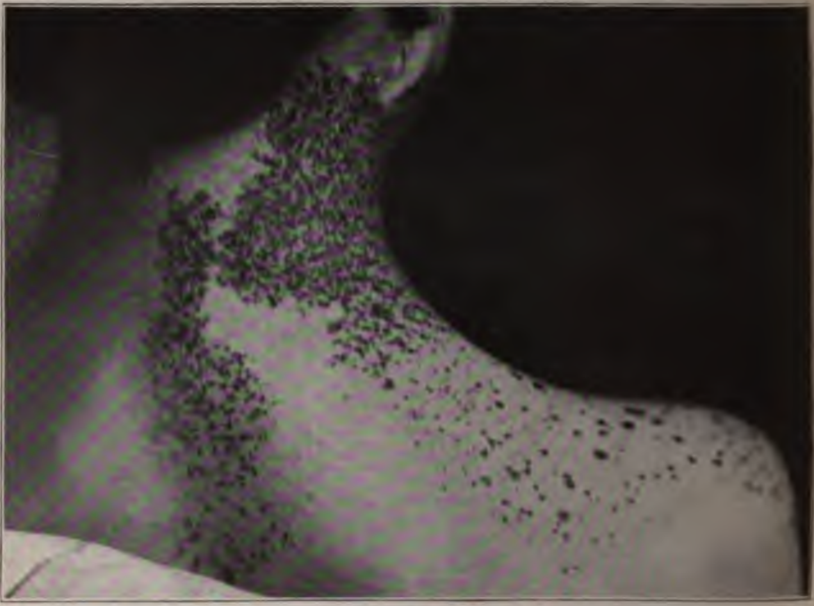
Hyde, Jour. Cut. Dis., 1909, xxvii, p. 256; Sibley, Brit. Jour. Derm., 1913, xxv, p. ; Williams, Ibid., 1912, xxx, p. 36; Sequeira, Brit. Jour. Derm., 1912, xxiv, p. 313.

Brit. Jour. Derm., 1911, xxiii, p. 77.

secondary infection. In older people there may be a malignant transformation.

Etiology.—Nevi occur in both sexes, either as congenital lesions or developing later in life. In either case they may persist without change, or undergo degenerative transformation at a later period. Many theories have been brought forward to explain the cause of the linear arrangement of the lesions. As summarized by D. W. Montgomery,[1] these are as follows: The lines follow the course of the cutaneous nerves or of the bloodvessels, or run along what are termed *Voigt's boundary lines*, or run in the metameres or segments of the body; or, lastly, that they lie along the embryonic sutures, but follow the trend of growth of the tissue.

Fig. 164



Nevus pigmentosus (unilateral distribution).

Pathology.—The histopathology of pigmented moles is characteristic, and the findings are uniform. On account of the frequent development of malignant growths on these lesions, the origin of the nevus cells has been made the subject of much study. Histologically, the epidermis, as a rule, shows but little change; occasionally, when the mole protrudes much above the surface, some hyperkeratosis may be noted, due to irritation, and there is also increased pigment in the basal layer of the rete. Characteristic findings occur in the corium. Parallel rows of oval or cubical cells, having large, oval, vesicular nuclei, pass down deeply into the corium from near the epidermis in a somewhat oblique direction. Between the rows of cells the fibrous elements are lessened, particularly the elastin. In addition to the above described cells, occasionally a few giant-cells and large cells containing pigment are found.

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[1] Jour. Cut. Dis., 1901, xix, p. 455 (with a list of 48 titles under which linear nevus has been described, and references to the literature).

As to the origin of the cells, von Recklinghausen[1] believed that they were derived from the endothelium of the lymphatics. Green,[2] Bauer,[3] Johnston,[4] and others confirm this view. Unna, on the other hand, traced their origin to the epidermis. His findings have been corroborated by Gilchrist,[5] Whitfield,[6] W. S. Fox,[7] and others. Fox states that while the major part are of epidermal origin, a few cases may possibly be mesoblastic.

In the other varieties of nevi the histological changes vary according to the structures particularly attacked. In the verrucous type, the histology resembles that of the ordinary wart. In certain linear types, Unna found evidences of inflammation and changes suggesting those seen in eczema. Elliot[8] found marked changes in connection with the sweat apparatus, consisting of cystic degeneration in the coils and ducts. Cyst-like projections of the epidermis into the corium were noted by Fisher.[9] The cysts were related to the excretory ducts of the sweat glands. Stokes[10] also described cystic involvement of the sweat apparatus in a warty nevus.

Diagnosis.—The major portion of the nevi above described are readily identified. The only difficulty in diagnosis will lie in the proper differentiation or distinction, if there be one, between the linear nevi of warty type and ichthyosis hystrix. Schalek[11] maintains that nevus unius lateralis and ichthyosis hystrix are distinct clinical entities, and that in histopathology a differentiation is readily made.

Treatment.—Pigmentary moles may be removed by excision, cauterization or electrolysis. The last-named method is applicable to the smaller and more superficial growths of this class. Fox[12] calls attention in connection with this subject, to the need of passing the needle no deeper than the epidermis, sufficiently deep merely to "blister the surface of the black spot." The electrolytic removal of hair from hairy moles often results in obliteration of the lesions. In case additional treatment is necessary, the mole may be transfixed in several directions and the current allowed to pass for a few seconds until the area becomes white. After this treatment the lesion gradually dries and forms a superficial crust, which later is exfoliated. The treatment may be repeated in case it is necessary. Carbon dioxid snow and liquid air are often highly satisfactory. In pigmented and hairy moles, a combination of freezing and radiotherapy for the removal of the more deeply situated hair constitutes a valuable means of treatment. Bunch,[1] in reporting the treatment of 300 cases of nevi (capillary, cavernous, and pigmentary), reported excellent results with carbon-dioxid snow and liquid air. Radium is advised by Wickham and Degrais[2] only in hairy and deeply colored prominent tumors. Simpson[3] reports the successful management of a pigmented and verrucous nevus with radium.

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[1] Ueber die multiplen Fibrome der Haut, Berlin, 1882.

[2] Virchow's Archives, 1893, cxxxiv, p. 331.

[3] Ibid., cxlii, p. 407.

[4] Jour. Cut. Dis., 1899, xvii, p. 132 (clinical report by Burnside Foster; histological report by J. C. Johnston.

[5] Ibid., p. 117.

[6] Brit.. Jour. Derm., 1900, xii, p. 267.

[7] Ibid., 1906, xviii, pp. 1, 47, 83 (a thorough piece of research work on this subject, with extensive references to the literature.)

[8] Jour. Cut. Dis., 1893, xi, p. 168.

[9] Derm. Wchnschr., 1915, lx, p. 65.

[10] Jour. Cut. Dis., 1917, xxxv, p. 411.

[11] Ibid , 1908, xxvi, p. 562.

[12] Electricity in Removal of Superfluous Hairs, etc., Detroit, 1886.

The technic for the employment of these agents will be found in the section on General Therapeutics.

Prognosis.—The change of a benign into a maligant neoplasm should be borne in mind. Aside from this possibility, the disfigurement occasioned by the blemishes is the only factor to be considered.

FOLLICULITIS ULERYTHEMATOSA RETICULATA.[4]

Under this title MacKee and Parounagian described a cutaneous disorder founded on the study of two cases. The eruption was limited to the face and was symmetrically distributed over the greater portion of both cheeks. The skin over the rest of the body was normal. The eruption consisted of numerous, closely crowded small areas of atrophy, separated by narrow ridges producing a reticulated honeycomb or network appearance. The individual atrophic areas were pit-like with abrupt edges about 1 mm. in depth and ranged in size from ¼ to 2 square millimeters. By coalescence areas as large as 3 sq. mm. were formed and these were square, polygonal, triangular, oval, oblong, or serpentine in outline. The skin covering the narrow partitions or ridges was on the level with that covering the unaffected portions of the face and on close inspection appeared shiny, waxy, and stretched. On palpation these ridges were found to be harder than the normal skin. There were a few small comedones both in the depressed areas and in the ridges, and in addition there were a number of milium bodies in the latter situation. The entire area was erythematous, the redness being more marked in the depressions.

The histology showed a slightly atrophic epidermis with loss of the rete pegs. There was inflammation of the corium which was manifested by vascular and lymphatic dilatation, congestion, edema, and perivascular and perifollicular infiltrations and small round cells. There were degenerative changes in the connective tissue ending in atrophy and retraction. The sebaceous glands were largely undeveloped. The most common change was noted in the hair follicles, which were greatly over-developed and otherwise changed They were large, acanthotic, greatly dilated, and filled with hardened material. Both the lanugo and ordinary hair follicles were attacked. Horny cysts derived from the hair follicles were scattered throughout the corium, and some of these were connected to the hair follicle by only a slender epithelial bridge, whereas others were completely isolated. They varied in size and could usually be demonstrated to be cross or tangential sections of tortuous hair follicles. The connective tissue showed edema and in places granular degeneration.

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[1] Brit. Med. Jour., February 4, 1919, p. 247.

[2] Radium Therapy, Paris, 1912, p. 224.

[3] Trans. Amer. Med. Assoc., Sec. on Derm., 1913, p. 30.

[4] MacKee and Parounagian, Jour. Cut. Dis., 1918, xxxvi, p. 339; A report of two cases with thorough clinical and histological study and review of similar or allied cases previously recorded.

As to the cause of the atrophy, some of it is attributed by the authors to the degeneration of collagen. It was undetermined whether this degeneration was secondary to the edema and circulatory disturbances caused by pressure, or whether it and the inflammation preceded the follicular changes. Degenerative changes were found in the ridges as well as in the depressed areas. The authors suggested that some of the depression might have been due to the presence of large double comedones. No cause for the disorder could be elicited. It could not be determined which appeared first, comedones, erythema, or pitted scars. There was no history of preceding pustules, vesicles, or papules. The disease had been steadily progressive from the start and had not been altered by treatment.

A case similar to the one above was reported by Little.[1]

ICHTHYOSIS.

Synonyms.—Fish-skin disease, Xeroderma. Ger., *Fischschuppenausschlag;* Fr., *Ichtyose.*

Definition.—Ichthyosis is a congenital cutaneous disease, characterized by a dry, harsh, and scaling condition of the skin, associated with abnormal cornification.

The disease commonly develops shortly after birth, although in certain cases adult life may be reached before it develops, and in certain others it is well developed at birth. For purposes of study, the disease is divided into several varieties; ichthyosis simplex, ichthyosis hystrix, ichthyosis congenita, and ichthyosis follicularis.

Symptoms.—Ichthyosis simplex presents symptoms of mild or severe grade. In mild form (xerosis, xeroderma) the skin is dry and harsh, and is usually the site of furfuraceous scales. The scaling occurs chiefly on the extensor surface of the arms and legs but may be generalized. The flexor surfaces and the trunk may be entirely spared in this grade. The skin over the elbows and knees is thickened and its lines are exaggerated. Small keratotic papules (keratosis pilaris) are present over the anterolateral aspects of the arms and the antero-external surface of the thighs. These present the same symptoms and are practically identical with those found in keratosis pilaris, appearing like an exaggerated goose-flesh and are rough to the touch. The entire skin has a grayish hue. As the disease progresses the scaling becomes more pronounced, and it is practically always worse in the above described areas. The scales are quadrilateral or diamond-shaped and are usually attached in their centers with the margins free. The following terms have been applied according to the type of scale present: When the scales are shiny and translucent, *ichthyosis nitida* or *ichtyose nacrée;* when brown-gray, green or black, *ichthyosis nigricans;* when appearing like a snake, *ichthyosis serpentina.* In some cases the thickening and irregularity is so great that the term *sauroderma* (crocodile skin) has been employed. In any case the more severe types are characterized by plate-like scales which are usually of a dirty grayish color. The flexors of the joints are spared in all except extreme cases; the face, scalp and nails, however, suffer. The nails become harsh and brittle and are easily broken. The hair becomes dry and the

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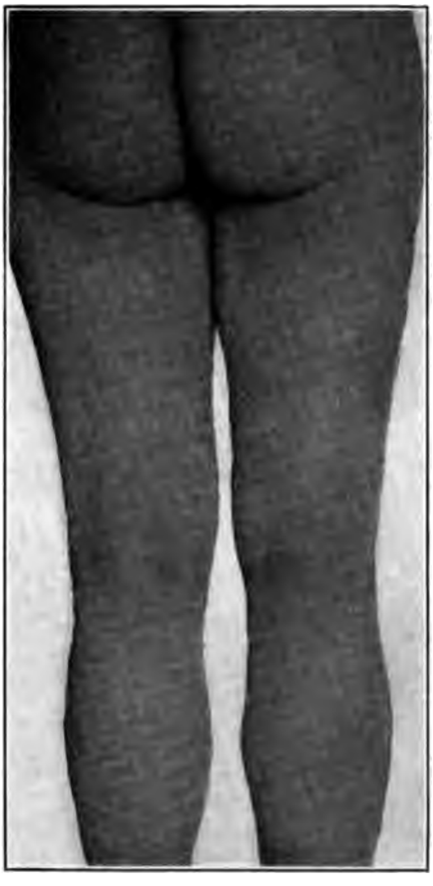
[1] Brit. Jour. Derm., 1919, xxxi, p. 201.

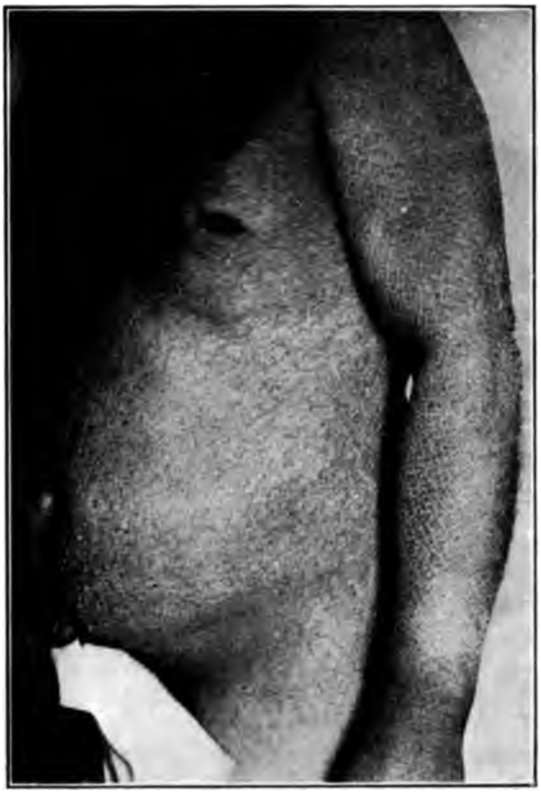
FIG. 165



Ichthyosis.

scalp covered with fine scales; the skin of the face also presents fine scaling and a slightly thickened appearance. The secretions of the entire body are diminished. The cold season has a detrimental effect on the disorder. Where the disease is marked and the horny plates have a regular setting the appearance is that of a fish skin; hence, one of its common names. The symptoms in all cases are exaggerated over the knees and elbows and in severe grades marked thickening occurs in these localities.

FIG. 166



Ichthyosis.

Ichthyosis Hystrix.—This is a term which formerly caused great confusion, owing to the fact that numbers of cases representing different types of nevi were classed under this head. Some authors believe that all cases of ichthyosis hystrix are nevi.[1] The term is restricted to those cases which present circumscribed patches of spinous excrescences, or patches in which papular or verrucous elements enter into their formation.

As a rule this disorder occurs independently, but in certain cases this type is associated with ordinary ichthyotic symptoms. Those cases in which distinct warty growths occur in lines, and others of this type, are described in connection with nevi.

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[1] Pernet, Brit. Jour. Derm., 1911, xxiii, p. 332; Sequeira, ibid., 1912, xxiv, p: 313.

Ichthyosis Congenita.—("*Harlequin fetus*," *Keratosis universalis congenita*).—This exceedingly rare disease occurs as an intra-uterine modification of the skin of the fetus, which is usually brought into the world as a non-viable monstrosity. Cases have been reported in this country by Elliott,[1] Sherwell,[2] Bowen,[3] Cabot,[4] Winfield,[5] Annie S. Daniel and Louis Cordes,[6] Moore and Warfield,[7] and others.

There appear to be two varieties of congenital ichthyosis: one practically always fatal, the other frequently terminating in recovery. By many the latter type is not accepted as being a true congenital ichthyosis. In the grave form the skin is represented by a thick, horny cuirass, deeply furrowed and resembling plates of armor. The large flakes of corneous epidermis, but partially attached to the corium, have broad, free edges. The ears, eyelids and lips are usually wanting, being replaced by corneous folds, suggesting in appearance the corresponding features of a mummy. The fingers and toes resemble talons and claws. Death usually occurs in the course of a few days from inability to secure nutrition by the act of sucking and from the imperfect development of other organs than the skin.

Sherwell[8] describes a case of congenital ichthyosis of unusual interest in that at the time of the report the infant had lived to be more than five months old, and seemed to be gaining in strength and improving in the condition of the skin. No history of heredity or of a family tendency to deformities of the skin could be obtained.

In a patient exhibited by the author before the Chicago Dermatological Society in 1908, a congenital malformation of the skin was present, exhibited at birth as a smooth envelop covering the entire cutaneous surface, which has been likened to oiled paper or collodion. After a few days this became broken up and began to exfoliate, leaving a tender but apparently normal epidermis beneath. Unfortunately, the child contracted an intercurrent disorder and died before the process was complete. Bowen[9] described a similar case and mentioned others reported elsewhere. He suggests that these cases are due to the persistence of the epitrichial layer of the embryo, which normally is entirely shed before birth, and is in doubt as to whether they should be classified as mild cases of ichthyosis congenita. In contradistinction to the true congenital cases, in those described above the general health is not impaired; and in cases that have survived for any length of time the epidermis merely presents scaling for a longer or shorter period after the shedding of the above described membrane, and later either becomes normal, or develops the symptoms of the ordinary type of ichthyosis.

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[1] Jour. Cut. Dis., 1891, ix, p. 20.

[2] Ibid., 1894, xii, p. 385.

[3] Medical Record, New York, July 6, 1895, p. 10, extensive bibliography.

[4] Ibid., 1895, xiii, p. 485.

[5] Jour. Cut. Dis., 1897, xv, p. 516.

[6] Jour. Amer. Med. Assoc., October 27, 1900, p. 1081, report of case with histology.

[7] Jour. Missouri State Med. Assoc. (commented upon in Jour. Amer. Med. Assoc., April 14, 1906, p. 1130.

[8] Loc. cit.

[9] Loc. cit.

Ichthyosis Follicularis.—Three cases of peculiar interest were described by MacLeod[1] under this title. In the 3 there were associated baldness, absence of eyebrows and eyelashes, trachoma and conjunctivitis. The hairy system was principally involved. In certain locations the hairs were replaced by pinhead-sized papules, surmounted by horny spines, the papules being uncolored and without areola. While these lesions were generally distributed, they were most marked on the extensor surfaces of the upper arms, back and sides of the neck, and the back. They were irregularly disseminated, but roughly symmetrical. The skin in general was moderately ichthyotic. There were no subjective symptoms. The lesions began on the face at or before the second year. There were 3 children thus affected in one family. MacLeod concludes that the cases belonged to the category of ichthyosis, and that, while they presented many features in common with keratosis pilaris, they differed in being non-inflammatory in origin, and therefore the title "ichthyosis follicularis" was the most appropriate one.

It will be seen from the above description that ichthyosis is a congenital affection beginning within a few weeks, or within a year, after birth as a rule. It may be comparatively local or generalized, and have mild or intense symptoms, the sites of location being the extensor surface of the arms and legs, and the parts relatively immune being the face, the axillæ, groins, the flexor aspect of the limbs, and the palms and soles. Its course is gradually to increase until a certain stage of development has occurred and then remain stationary. Exacerbations occur during cold weather; mild cases entirely clear up during the summer season. Intercurrent disease causes temporary disappearance of the symptoms. One ichthyotic patient is said to have regularly cast a slough of his integument in the autumn (Hyde). Williams[2] reported a regular shedding of the skin in an ichthyotic patient, with subsequent recurrence of the ichthyotic symptoms.

Etiology.—Ichthyosis is a congenital condition, although its first manifestations may not appear until the second year of life. Heredity is an important factor.

Thost[3] describes ichthyosis occurring in four generations. According to the ascertained genealogy, the ancestor first known to have suffered from this affection had 5 male children who inherited it, whereas 1 girl and 1 boy were spared. One of these affected subjects had 5 children, of whom 3 males showed the anomaly, while 1 boy and 1 girl remained free. Another subject, of the same generation, had 5 male and 3 female children; of these 4 boys and 2 girls became affected. One of the latter (third generation) bore 4 children, of whom 3 girls inherited the disease, while the fourth, a boy, escaped. Many other remarkable family trees could be mentioned.

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[1] Brit. Med. Jour., 1909, xxi, p. 165.

[2] Brit. Jour. Derm., 1912 xxiv, p. 233

[3] Inaug. Diss., Heidelberg, 1880; Centralbl. f. Chir., 1881, xiii, p. 154.

Crocker describes an acquired case in a septuagenarian. The affection occurs equally in both sexes and in persons of all social ranks. It is usually aggravated in cold climates and during the winter season. Kaposi states that the cause of ichthyosis appears to be a local anomaly of the nutrition of the skin "especially involving its epidermic and fatty layers." In the Molucca Islands and some other isolated regions ichthyosis, on account of its unusual prevalence, has been regarded as an endemic affection. While it is difficult to explain the occurrence of the disease in this manner, it is thought by some that heredity and intermarriage could account for it. On the other hand, these cases may be a different disorder.

Pathology.—In the mild forms, Unna describes an immediate formation of the horny layer from the rete without the intervention of keratohyalin. The result is a complete cornification, the horny cells being homogenous and containing no nuclear remnants. In this respect the process of keratinization is unusual, and contrary to the belief of many observers that cornification is impossible without the intervention of the keratohyalin of the granular layer. The rete is thinned more from an atrophic condition of the cells than from an actual diminution of their number, though this does occur sometimes, so that only one or two layers of cells cover the papillæ. The lymph spaces are also very small. The extremities of both the rete pegs and papillæ are broad and flattened and their necks narrowed, so that they suggest a dove-tailed appearance. The coil glands possess a swollen epithelium and a widened lumen, resembling their excretory ducts, which exhibit less functional activity. The follicle mouths either are dilated with a broad, horny plug, or are closed, retaining the plug in the dilated neck. In a histological study of the skin of a case of ichthyosis congenita born at term, Wassmuth found the chief changes in the epidermis. As a whole, the epidermis was greatly hypertrophied, and a clear differentiation into the usual layers was not possible, owing to the early keratinization occurring throughout its entire extent. Occasionally cells containing keratohyalin granules were noted in places, but a well-defined stratum granulosum was present only in the scalp. The stratum corneum was greatly thickened in all regions, being most marked in the scalp, the palms and soles, and least marked in the chest and abdomen. The rete Malpighii shared in the hypertrophic process, and many rete pegs were dovetailed by the enlarged and numerously increased papillæ of the corium. The papillæ were described as being enormously increased and containing networks of distended capillaries. The sweat glands were increased in number, but otherwise normal. The sebaceous glands were increased in number and at times deformed by keratinization of the follicle-mouths. The hairs grew normally except for their deformed shape, caused by the thick and dense horny layer. The corium showed only an insignificant chronic inflammation of low grade, with a decreased amount of elastic tissue and the changes in the papillæ above noted.

The histology of the lesions in ichthyosis follicularis as described by MacLeod showed the process to be primarily a hyperkeratosis involving not only the hair-follicles but also the intervening skin. There were thinning of the underlying rete and a flattening of the interpapillary processes. The lower part of the hair follicles was atrophied as a result of pressure by the hyperkeratotic plug in the funnel-shaped follicle above. The underlying corium showed some dilated vessels, with a moderate cellular infiltration. The latter, though suggesting a mildly inflammatory reaction, was considered secondary to the epithelial changes above noted.

Diagnosis.—Ichthyosis presents its characteristic symptoms and it rarely is mistaken for any other disorder. Its history begins at or shortly after birth and continues throughout life. The non-inflammatory scaling condition and absence of subjective symptoms serve to distinguish it from other scaling dermatoses.

Treatment.—Internal treatment, as a rule, is not of much value. Thyroid extract, antimony, sulphur and jaborandi have been recommended. The first mentioned is of value in certain cases. Subcutaneous injections of $\frac{1}{4}$ grain (0.016) of pilocarpin have been employed in ichthyosis to produce sweating and increase the oil of the skin.

External treatment is employed to soften, macerate or anoint the skin. The softening is accomplished by alkaline or vapor baths combined with the use of sapo mollis, following by anointing the skin with vaseline, almond, cod-liver, linseed or olive oil, benzoated lard, lanolin, or a combination of olive oil and glycerin in a proportion of 3 parts of oil to 1 of glycerin or a salicylated cocoanut oil. Stelwagon and others recommend the addition of resorcin to the above ointments in a strength of 2 to 10 per cent. Sulphur and ichthyol also have been employed in ointment forms. The French, after the removal of the superficial horny plates in a bath of soft soap and water anoint the body by friction with glycerite of starch. Van Harlingen recommends the following for application when the epidermis begins to shed after employing soft soap:

℞—Potass. iodid.,		grs. xx;	1 33
Ol. pedis bubuli,			
Adipis,	āā	℥ss;	15
Glycerini,		ʒj;	4

Anderson recommends wearing pure vulcanized India rubber garments, a method of treatment which might be exhausting for some patients.

Prognosis.—From the above description it will be seen that a patient cannot hope to make a complete recovery from his disorder. The process will stop after it has progressed to a certain stage and will remain stationary; with persistent treatment and possibly change of climate, the condition may be improved.

Erthrodermie Congénitale Icthyosiforme.—Under this title Brocq[1] groups several cases having symptoms in common which seem to represent a new dermatosis. Cases were recorded early in this country by Jackson[2] and White.[3] An important contribution to the subject has recently been made by MacKee and Rosen.[4]

Symptoms.—The disorder begins, as a rule, with a more or less generalized erythroderma, which may be of moderate or more severe grade. At a later date the erythematous skin becomes thickened and scaly, presenting the picture of ichthyosis; in adult life the redness may disappear entirely, leaving an ichthyotic skin. In some instances the scaling precedes the erythema. The general picture of the disease is that of ichthyosis with the addition of redness. Unlike ichthyosis, the flexor surfaces are involved and appear to be sites of predilection for this disorder. The changes in the skin vary from a mild xerosis to a type which might be described as ichthyosis hystrix. The palms and soles are invaded to a variable degree. The face is usually red and presents a slight scaling and ectropion may be present. The scalp usually presents scaling and the hair of the scalp and body, as well as the nails, may grow with unusual rapidity, although in certain instances the opposite condition is present, or these appendages may be normal. In a few cases bullous lesions have occurred as a complication. Bullæ are seen most frequently on the extremities, are of the flaccid type, and disappear without sequels. They reappear during the winter months and, as a rule, disappear permanently when adult life is reached. The disease is usually generalized and symmetrical although some of the cases present circumscribed lesions. Perspiration is limited except on the flexors of the extremities and on the palms and soles. A certain amount of itching occurs in some cases, but general symptoms are absent in the major portion of cases.

Pathology.—The histological picture shows a pure hyperkeratosis. The thickened stratum corneum may extend rather deeply into the hair follicle, and may also involve the orifice of the sweat ducts. The granular layer varies in thickness from slight increase to complete absence. The rete may show acanthosis, or be thin. Occasionally edema or dyskeratosis is encountered. The rete pegs may be flattened or enlarged and branched. The papillæ may be broad and flattened or narrow and elongated. There is usually a slight inflammatory reaction in the upper part of the corium, indicated by dilated capillaries surrounded by moderate lymphocytic cellular infiltration. The collagen is usually unaffected; there may be slight edema or other minor changes. The elastic tissue is reduced and fragmented. The coil glands may be dilated and usually show evidences of degeneration, or remain normal. The sebaceous glands are unaffected.

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[1] Annales, January, 1902, p. 1.

[2] Jour. Cut. Dis., 1909, xxvii, p. 36, case presentation.

[3] Ibid., 1910, xxviii, p. 533, case demonstration.

[4] Ibid., 1917, xxxv, pp. 235–343 and 511, including a report of cases and a comprehensive review of the clinical and histological features of all reported cases up to that date. The revised article in the text was largely taken from this work. Brown, Brit. Jour. Derm., 1917, xxix, p. 263, case report.

Diagnosis.—The disorder could only be mistaken for an unusual generalized keratoderma or ichthyosis. Both clinically and histologically there are very few points of distinction between it and the latter disease. The chief clinical difference lies in the presence of redness and the marked involvement of the flexor surfaces in the disease under consideration.

SCLERODERMA.[1]

Synonyms. — Hide-bound skin, Dermatosclerosis, Chorionitis, Scleriasis, Sclerema adultorum. Ger., *Hautsclerem;* Fr., *Sclérodermie.*

Definition.—Scleroderma is a disorder characterized by induration of the skin in localized patches or diffuse areas frequently associated with atrophy and pigmentation. Two fairly distinct types occur, the diffuse and the circumscribed. Occasionally both types are seen in the same patient.

Symptoms.—Diffuse scleroderma develops, as a rule, insidiously, and may begin about the extremities, the neck, face or trunk. Occasionally the disorder spreads rapidly and covers large surfaces in a short time. As a rule, no subjective sensations precede the cutaneous manifestations, though in certain cases, particularly those involving the extremities, neuralgic and arthritic pains or paresthetic sensations are the first signs. Commonly a feeling of stiffness is the earliest change noted. The early cutaneous changes vary in different types. Infiltration or a peculiar edema, the latter occurring with or without erythema, are the earliest signs in most cases. In the former the skin is indurated and feels stiff and gradually its normal lines are effaced. The disorder spreads slowly or rapidly, and the induration increases until large surfaces, or the entire cutaneous surface become involved. When more fully developed the skin becomes tense and hardened to such a degree that it cannot be picked up between the thumb and finger. The color is at first unchanged, but later a yellow or ivory-tinted hue is exhibited. As the disorder progresses, induration and rigidity increase and the skin and subcutaneous tissues become firmly bound to the underlying structures.

The face, neck, the upper parts of the trunk and the upper extremities are most frequently involved. Though it may occur on any part of the cutaneous surface, the palms and soles[2] are rarely included in the process.

When the face is involved it becomes mask-like and expressionless; mastication is difficult and opening and closing the mouth and eyes are interfered with through rigidity of the tissues of the cheeks, lips and eyelids, the last mentioned being least affected. Movement of various joints is interfered with in varying degrees up to complete fixation, this being due to the sclerosed tissue about the joints and not to articular involvement. Breathing may be difficult in cases involving large surfaces of the thorax. When the hands are involved the fingers are moved with difficulty or are fixed in a semi-flexed position and may become reduced in size. Not infrequently ulceration complicates the process.

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[1] Some valuable reports; Fox, Brit. Jour. of Derm., 1892, iv, p. 101, Early English Observations; Osler, Jour. Cut. Dis., 1898, xvi, pp. 49 and 127, Report of 8 cases and discussion of diagnosis and treatment; Lewin and Heller, Die Sklerodermie, Berlin, 1895 an extensive review; Robinson, Scleroderma, Am. Jour. Med. Sc., 1917, cliv, p. 657, a review of 68 cases.

[2] Pernet, Brit. Jour. Derm., 1916, xxviii, p. 49; case demonstration involving the soles.

According to Crocker[1] edema, in place of infiltration, may be the first change in certain cases. In these a peculiar, stiff edema occurs, likened by Wilson to "putting the finger into a bladder of lard." The edematous stage lasts for a variable time, weeks or months, when the edema becomes absorbed, the skin shrinks, assumes a waxy-white color and the atrophic stage follows.

The atrophy following some edematous cases is less extensive than the preceding edematous infiltration. In a patient under the observation of the author for many years a generalized scleroderma was present. The neck, chest, arms, forearms, hands and lower extremities were affected. In the early stages deep pitting could be produced. The usefulness of the hands and arms was much curtailed. As absorption took place the tissues became softer and distinct atrophied areas, usually linear, developed. Finally all induration disappeared and aside from the bands of atrophy and a moderate loss of strength in the hands and slight immobility of the wrists, the patient completely recovered. An interesting case of this type but of localized distribution is recorded by Sutton and Kanoky.[2]

In certain cases there is an edema which does not pit and over which there may be erythema. These cases, as a rule, begin as hard, edematous patches or diffuse areas which, after weeks or months become more hardened and tense and the color changes to a dead-white, or assumes the hue of old marble or parchment.

In the atrophic stage the skin becomes thinner, harder and more firmly bound down to the underlying structures. The normal lines are obliterated and the face presents a corpse-like appearance. It becomes smaller, the lips thin, the nose shrunken, the cheeks sunken and smooth, and the eyes expressionless, owing to the immobility of the lids. The fingers become tapered and fixed and the entire limb may become reduced in circumference by several inches, through atrophy of all the structures. Ulceration frequently occurs over bony prominences. Pigmentation usually occurs later in the disorder and may be lentiginous or diffuse. Bronzing of the entire cutaneous surface is occasionally noted, and areas of depigmentation may be interspersed in the pigmented skin. The functions of the skin are not disturbed as a rule; sensation is usually unimpaired, though some hyperesthesia and anesthesia occasionally is present. The skin feels tense and stiff but other symptoms are usually absent though itching may occur. The hair is commonly thin, or falls out completely in the affected area.

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[1] Diseases of the Skin, p. 618.

[2] Jour. Cut. Dis., 1909, xxxii, p. 556. A comparative study of acrodermatitis chronica atrophicans and diffuse scleroderma with associated morphea atrophica, with references.

In children scleroderma is characterized by the same group of symptoms that occur in the adult. The course, however, is more often acute and severe atrophic forms are less frequently seen. Cockayne[1] in an analysis of 112 cases in childhood, found a record of many that were severe and atrophic. Goodman[2] reported a generalized scleroderma beginning about the neck following tonsillitis in a child aged nine, in which the disorder entirely cleared up in five months, without sequels.

In infants indurated areas form which spread for a time, frequently coalesce with others, then undergo resolution and disappear. The skin in the areas may be normal in color, or bluish or brownish-red. The areas of involvement in these cases are the trunk, particularly the lower part posteriorly, and the extremities. Lieberthal[3] is of the opinion that this type is benign and usually clears up rapidly.

Sclerodactylia occurs as a part of the general scleroderma, or the sclerodermatous process may be limited to the hands and feet. It may be progressive, in which case the disorder begins at the fingertips and spreads upward, involving the hand, wrist, forearm, and other areas. Some of these cases begin with symptoms of Raynaud's disease and may be limited for long periods of time to the extremities. In these cases after a variable period the skin becomes firmly bound down to the underlying structures and assumes a grayish shade. The fingers become stiffened and bent and are fixed and useless. Ulceration frequently follows and portions of the fingers necrose and disappear. Telangiectasia and pigmentation may be present. Occasionally subcutaneous calcareous concretions[4] occur in this location.

The course of the disease is variable. At times its progress is arrested and this may occur at any stage. The sequels depend upon the stage of the disease when activity ceased, and the amount of destruction already accomplished. Some cases recover spontaneously or under treatment and present very little evidence of the previous affection. More often, the disorder progresses until terminated by some intercurrent affection.

Etiology.—The disease occurs at all periods of life from infancy to old age and three times more frequently in the female sex than in the male. The direct cause is not known. Many contributing factors and associated conditions are recorded. It may follow various infections, such as scarlet fever, measles, diphtheria, tonsillitis, erysipelas, pneumonia, influenza and others. It is reported to have followed exposure to cold, damp and even a prolonged exposure to the sun's rays. The association of the disease with affections of the thyroid[1] has been frequently recorded. The gland was decreased in size more frequently than enlarged and in some instances was apparently absent. Foerster[2] attributes an important part in the etiology of scleroderma to morphologic and functional changes in the glands of internal secretion, especially in the thyroid.

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[1] Brit. Jour. Children's Dis., 1916, xii, p. 225.

[2] Jour. Cut. Dis., 1918, xxxvi, p. 210.

[3] Ibid., 1916, xxxv, p. 29; report of an infantile case and discussion of scleroderma neonatorum.

[4] Sholfield and Weber, Brit. Jour. Derm., 191[illegible], xxii, p. 276; Ormsby, Jour. Cut. Dis., 1915, xxxiii, p. 392; Olson, Ibid., 1917, xxxv, p. 96, report of a case and references.

Scleroderma may also occur in association with Raynaud's disease, Addison's disease[3] and syphilis. Improvement under antisyphilitic treatment has long been recognized in some cases and a certain proportion have a positive Wassermann reaction.[4] Others present clinical signs of syphilis. Trauma may be a contributing factor etiologically.[5] Thibierge[6] concludes that traumatism is merely a contributing factor in a preëxisting pathological condition. Organic changes in the nervous system have rarely been demonstrated in scleroderma, though emotional disturbances such as shock, anxiety and grief have been noted as predisposing causes. The arrangement of the lesions in certain localized places suggests implication of the nervous system. Sequiera[7] concludes that morphea does not depend upon an affection of the peripheral nerves but that it is probably due to a central ganglionic lesion and that this acts by vasomotor or trophic influences. Cases suggesting a toxic origin have been recorded by Adamson,[8] Pernet[9] and Little.[10]

Circumscribed Scleroderma (*Morphea, Keloid of Addison*).—This variety is characterized by the occurrence of one or several discrete, well-defined, firm, smooth patches, lines or bands, that are either slightly elevated or depressed, and surrounded by a violaceous or violet-tinted halo, the involution of which may be followed by atrophy. Patches of morphea commonly begin as red or violaceous areas, fingernail to palm size or larger, and increase in size rapidly or slowly. In a variable period of time the center of each patch becomes whitish or yellowish, while the peripheral portions of the plaque are purple or violaceous. The plaques may be round, oval, or irregularly shaped, occasionally are several inches in diameter, the central portion being slightly depressed or elevated, markedly infiltrated and lardaceous. The center often has the hue of old ivory. These patches may be single or multiple. In the latter event they may be arranged along the line of distribution of the cutaneous nerves of the trunk, along the inner face of the thigh, more often on the lower than the upper extremities, and symmetrically in most cases. The skin in the affected area when picked up between the thumb and finger gives the impression of stiffness and hardness. The surface may be dry and smooth or traversed by delicate lines. In some instances the plaque is dotted regularly with depressed points, resembling the patulous orifices of sebaceous glands. The slightly discolored, minute, funnel-shaped openings contrast with the dead-white hue of the patch. The border of typical patches is characteristic. It is made up usually of a narrow zone consisting of a plexus of fine vessels having a pinkish, lilac-tinted or violaceous hue. This zone may be absent, particularly in the cases in which the temple is involved, or may be as conspicuous as the whitish central area. Purplish and even blackish hues have at times been recognized in the halo by other observers.

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[1] For discussion of this subject see Samouilson, Thèse de Paris, 1898, Roques, Annales, July, 1910, p. 383; and Sequeira, Brit. Jour. Derm., 1916, xxviii, p. 31.

[2] Jour. Cut. Dis., 1916, xxxiv, p. 1.

[3] Winfield, Ibid., 1904, xxii, p. 586; Alquier, Touchard, Archive de Méd. Experimentale, 1907, xix, p. 691.

[4] *Cf.* Whitehouse's paper on this subject presented before the American Dermatological Association, June 3, 1909, Jour. Cut. Dis., 1909, xxvii, p. 535; and Lustgarten, ibid., p. 304.

[5] Bunch, Brit. Jour. Derm., 1909, xxi, p. 24; Morphea (distribution that of the right supraorbital nerve on forehead, following an injury on the site.

[6] Annales, December, 1915, p. 645; A Review of Scleroderma and Traumatism.

[7] Brit. Jour. Derm., 1911, xxiii, p. 40; two cases of frontonaso morphea.

[8] Brit. Jour. Derm., 1908, xx, p. 230.

[9] Ibid., 1908, xx, p. 230.

[10] Ibid., 1910, xxii, p. 57.

The band form of circumscribed scleroderma usually occurs in ribbon-shaped elongations along a limb in its longitudinal axis, or on one-half of the face or forehead. Most of these cases are distinguished by the occurrence of either an elevated ridge or furrow having an elevated ridge with a furrow on one side. The median line of the forehead is a common site of this form on the face. Over the trunk it is commonly seen on the chest. Sequeira[1] demonstrated a patient with a lesion one-and-a-half inches wide extending from near the anterior-superior spine of the ilium down the entire length of the leg. In this patient other linear areas, small atrophic white patches, and several large, dark sclerosed areas were present, the patient showing both sclerotic and atrophic changes.

As a rule there are few subjective symptoms. In some instances itching, tingling, pricking and other sensations are experienced. The course of circumscribed scleroderma is chronic lasting from one to ten years or more, or subacute with evolution accomplished in a comparatively short time. As the disease clears up there is commonly left a thin, inconspicuous atrophic area.

Morphea Guttata (*White spot disease*).[2]—In this variety the eruption occurs chiefly on the anterior surface of the chest and on the neck and shoulders; occasionally on the arms, forearms, and other parts of the body. The most striking feature of the eruption is the color, or rather absence of color, the plaque being chalk-white or snow-white, The lesions vary in size; for the most part they are that of a pinhead to that of a small pea or larger, and show little tendency to coalesce. Occasionally, irregular patches are formed by incomplete coalescence of individual lesions. The lesions may have a narrow line of brownish-red pigmentation, or a small reddened areola. When patches are formed, pigmentation may be marked at their margins. In time the lesions gradually undergo a distinct atrophy, the epidermis becoming thin, parchment-like, and slightly depressed. In these cases there are no subjective symptoms.

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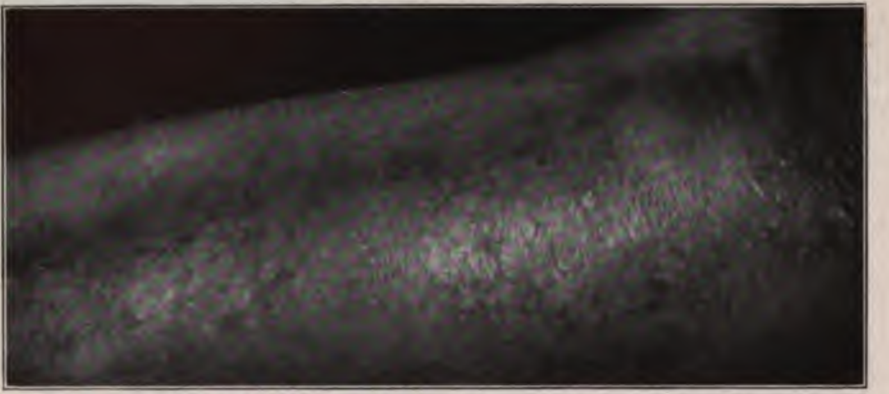
[1] Brit. Jour. Derm., 1908, xx, p. 198.

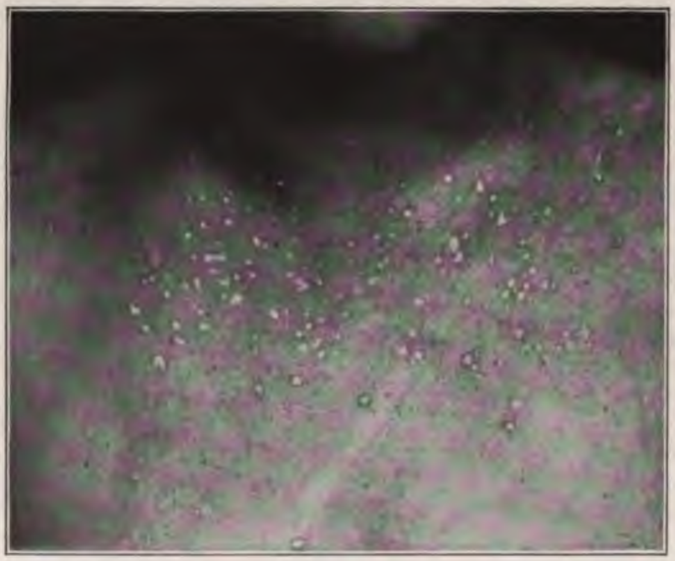
[2] Montgomery and Ormsby, Jour. Cut. Dis., 1907, xxv, p. 1: White Spot Disease (Morphea Guttata) and Lichen Planus Sclerosus et Atrophicus (a clinical and histological study of three cases, with a review of the literature). MacKee and Wise, ibid., 1914, xxxii, p. 529: White Spot Disease (a clinical and histological description, with discussion and bibliography to date.) Bunch, Brit. Jour. Derm., 1915, xxvii, p. 77; Morphea Guttata (Case report; discussion of previous cases with references). Turnbull, ibid., 1015, xxvii, p. 450: Scleroderma Guttata (Clinical and histological report with references.)

FIG. 167



Scleroderma occurring in patient with morphea guttata.

FIG. 168



Morphea guttata.

The connection between this variety and diffuse scleroderma is well shown in a patient exhibiting a band of typical scleroderma extending from the knee to the ankle (Fig. 167), and typical spots of morphea guttata on the chest and other regions (Fig. 168).

Pathology.—Most authors view scleroderma as a trophoneurosis or an angiotrophoneurosis, depending upon unknown changes in the trophic center. Osler[1] states: "The analogy of myxedema, to which scleroderma is the cutaneous antithesis, suggests that it may be caused by some alteration in an internal secretion, or some disturbance of that nice balance between the various internal secretions . . . which some believe play such an important role in nutrition. . . . The frequency with which acute forms follow an infection is paralleled by the thyroid insufficiency and atrophy caused by myxedema after a fever, such as measles and scarlet fever." The glands of internal secretion are being given much attention in regard to their etiological connection with scleroderma. In five cases Singer and Beer[2] found decrease in the size of the thyroid. Hektoen and Wells[3] found marked atrophic and cystic changes in the thyroid, and from a chemical examination demonstrated its reduced functional capabilities. Rasch,[4] describing a case of scleroderma with changes in the mucosa of the mouth and Basedow-Addison symptoms, believes there was an insufficiency of all the internal secretions. In a histological study of diffuse scleroderma, Unna found the main process to be an hypertrophy of preëxisting collagenous bundles, affecting equally all parts of the cutis, which led to simple pressure atrophy of the vessels as well as the epidermic structures. Early the bloodvessels were dilated and surrounded by swollen and increased numbers of connective-tissue cells. Later they were narrowed, cord-like and distorted by compression of the collagenous bundles, with corresponding decrease in the number and size of the connective-tissue cells. The changes in the elastin are only those due to pressure from the increased collagenous tissues. The latter extends into the hypoderm and replaces the fatty tissue. The atrophy which occurs later is similar in appearance to that produced by other causes, and is sometimes brought about by an endarteritis. The epidermic structures and vessels suffer first and the collagen later, the ultimate result being a parchment-like, thin, cutaneous plate, without suggestion of hypoderm or papillary body, and covered by an atrophic epidermic. The histology in a case of diffuse scleroderma is summarized by Hektoen as follows: "There is a great hyperplasia of the collagenous intercellular substance in the corium with flattening of the papillary body, together with a breaking up and spreading asunder of the elastic fibers, obliterative thickening of the bloodvessels, and atrophy of the interpapillary epithelium, as well as of the epidermis, the hair follicles, and the coil glands." In morphea Crocker found the epidermis only slightly altered. In the corium the superficial vessels showed thrombosis. There was a perivascular cellular infiltration, constituting irregularly branched masses of small round cells. Branching from the cell masses there was often a reticulum of fine fibrils, along which cells were attached. About the sebaceous glands and hair follicles the cell groups and reticulum were abundant. Slight deposits occurred, also, about the sweat ducts, but not about the coils. In the later stages the essential feature was an increase in the collagenous tissue. This, by pressure, obliterated to a marked degree the bloodvessels, sebaceous glands and sweat ducts. In certain areas the connective tissue was increased between the acini of the sweat glands.

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[1] Osler's Modern Medicine, 1909, p. 666.

[2] Berlin. Klin. Wchnschr., 1895, p. 46.

[3] Jour. Amer. Med. Assn., 1897, xxviii, p. 1240.

[4] Zeitschrift, 1912, xix, No. 3, p. 244.

From these findings Crocker interprets the process to be a defect of innervation, producing cell exudation around the bloodvesssls, thus narrowing the lumen, obstructing the blood flow, and leading to thrombosis and sometimes to rupture and effusion. Each atrophic spot seen near a growing patch is the base of a cone from which the blood supply is cut off, and the violet zone is due to the collateral hyperemia around an anemic area. Unna's card-like scleroderma is more superficial than the ordinary morphea above described, and in histology resembles to a certain degree the guttate form. In the latter variety the author found the following changes: The chief pathological changes were noted in the corium. The collagenous bundles were comparatively thick and hypertrophic, were straighter than normal, and showed but little interlacing. The bundles traversed the field in a horizontal direction, and only a comparatively few connective-tissue nuclei were present upon them. There were numerous dilated lymph-spaces, many of which contained some round and connective-tissue cells; only an occasional mast-cell was noted. Pigment cells were present throughout the corium and extended fairly deeply into the pars reticularis. Pigment granules were also detected apart from the cells. The elastin was normal, but its arrangement was modified by the changes in the collagenous bundles. The papillary layer was not well defined, as large bundles of collagen extended into it in places. The wavy line between the epidermis and corium was poorly defined, but not obliterated. The vessels were few and represented chiefly by narrow, longitudinal bands of cells. Their lumen was usually not demonstrable, and they were apparently obliterated by the intravascular proliferation. The glands of the skin were represented by a few deeply situated coil glands and partially obliterated sebaceous glands. All these structures were surrounded by a small, round-cell infiltration, which was also present in independent small areas. In the epidermis there was marked hyperpigmentation, the pigment granules showing throughout the basal and adjoining layers, extending in places to the third and fourth layer of cells. The hypertrophy of the collagen, comparative absence of bloodvessels, large numbers of dilated lymph spaces, hyperpigmentation both in the rete and corium, and comparative absence of the glands of the skin are all recognized features of ordinary scleroderma.

Diagnosis. In vitiligo there is an entire absence of all structural changes in the skin. The pigmented macules and atrophic patches of lepra present anesthetic or hyperesthetic symptoms and their coincidence with or sequence from other symptoms of the disease, such as nodules, bullæ or ulcers and involvement of other organs and tissues differentiates the latter from scleroderma.

A distinction between sclerema and edema neonatorum and scleroderma is given in connection with the former disease.

In ichthyosis, the congenital history, the presence of ichthyotic plates over the affected surface and the absence of induration in the skin suffice to identify the disease.

Xeroderma pigmentosum is distinguished by the presence of hyperpigmentations, warty growths, telangiectatic areas, with subsequent ulcers and tumor formation and limitation of the disease to the exposed parts.

The resemblance between scleroderma in the atrophic stage and acrodermatitis chronica atrophicans is close, and in certain cases a differentiation is impossible. (*Cf.* section on latter disorder.)

Treatment.—In the management of symmetrical or generalized scleroderma a change of climate to one which is dry and equable is of the greatest advantage. If the patient must remain under unfavorable climatic influences the clothing should be so selected as thoroughly to protect the body. In general, a nutritious diet should be ordered, and not infrequently cod-liver oil and iron tonics are of service. Roques[1] in a published record of the effects of thyroid administration, reports that success by the use of thyroid extract was noted in 63.7 per cent. The treatment should be started early and given in small dosage over long periods. Little[2] demonstrated a patient suffering with scleroderma associated with Graves's disease and later myxedema, which had been greatly benefited by the internal administration of thyroid and the implantation of human thyroid into the bone marrow. Suprarenal extract is of some value in certain cases; pituitary extract was of no value in 4 cases; testicular and ovarian extracts also have been used without success. Kolle[3] reports much improvement in scleroderma treated with coeliacin (an extract of the mesenteric gland), in dosage of 5 grains, three times daily. Schwerdt[4] reports favorable results in scleroderma by the internal administration of the mesenteric gland of the sheep in the form of tabloids, in dosage of 5 grains, once or twice daily. Philippson[5] reports relief of severe diffuse scleroderma by the internal administration of salol in doses of from 2 to 3 grams daily. Hebra[6] reports good results in 3 cases from intramuscular injections, every second day, of 10♏ of a 15 per cent. alcoholic solution of thiosinamin. Many drugs have been employed but, as a rule, these were purely symptomatic, and it naturally follows that the general treatment should be carried out according to indications in the individual patient. Local massage, galvanism, alternate hot and cold douches are of value. Daily inunctions of cod-liver oil, lanolin, lard, vaseline, or slightly scented neat's foot oil may be used. A small amount of ammoniated mercury or salicylic acid may be added. In morphea Brocq employs electrolytic puncture, as in the treatment of hypertrichosis, mercurial plasters being employed in the interval between the sittings.

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[1] Annales, July, 1910, p. 3831; abstr. Brit. Jour. Derm., 1911, xxiii, p. 92. Opotherapeutic Treatment of Scleroderma; full bibliography on this phase of the subject.

[2] Brit. Jour. Derm., 1916, xxviii, p. 124.

[3] Münch. med. Wochenschr., January 7, 1913, lx, p. 34; abstr. Jour. Cut. Dis., 1913, xxxi, p. 1057.

[4] Ibid., 1907, No. 25; abstr. Brit. Jour. Derm., 1908, xx, p. 101.

[5] Deutsch. med. Wchnschr., 1897, p. 33.

[6] Archiv, 1899, xlviii, p. 120.

SCLEREMA NEONATORUM.[1]

Synonyms.—Sclerema adiposum, Sclerema of the newborn. Fr., *Sclérème des nouveau-nés; Athrepsie.* Ger., *Fettsklerem.*

Definition.—This disease is not to be confused with edema neonatorum, from which it is distinct. It was described first by Underwood[2] in 1784, and is an affection of extreme rarity. It is a peculiar form of coagulation of the subcutaneous fat-tissue, accompanied by dryness of the skin, so that very little fluid exudes when it is incised.

Symptoms.—At birth, or between the second and the tenth day after, the lower limbs of the child assume a livid or whitish-yellow appearance, occasionally suggesting the hue of wax; and they become of a leathery consistency. This condition spreads gradually over the lumbar region, the dorsum of the body, and the chest in front and behind, and in the course of a few days may involve the entire integument excepting the palms, soles, and scrotum. When pressed upon with the finger, the skin produces the impression of half-frozen tissue; the face suggests a cold and rigid mask; the thighs in their sockets and the arms in the shoulder-joints are immobile. Usually there is somewhat less firmness of the abdominal integument. The taking of the nipple, deglutition, and even the opening of the mouth are effected only with great difficulty, and eventually become impossible. The respirations are difficult and slow (16 or less); the pulse may be 60, and in well-marked cases is imperceptible at the wrist; and the temperature is lowered (30° to 26°) though exceptions are noted. Lotta Myers[3] recorded an example in which at no time was the temperature found to be subnormal. There is often no cry.

There may be a coincident icterus; and often thrush has been observed in the mouth before the declaration of well-marked symptoms. The congenital subjects are often stillborn. The majority of victims of the disease perish before the ninth day. Diarrhea is usually present.

Etiology and Pathology.—The disease occurs in prematurely-born and feeble infants. Poor hygiene, insufficient and improper food and clothing are also factors and, according to Ballantyne,[4] most cases occur in the winter months and among foundlings. The loss of body fluids through diarrhea and internal hemorrhage are also important factors. One hypothesis concerning the origin of the disease is that it depends upon a changed condition of the fat. According to Langer and Knopfelmocher (quoted by Luithlen), the fatty acid content in the infant skin is low, while the melting point is high. From this it follows that with a marked lowering of the body temperature in the disease, solidification of the fatty tissues occurs. The absence of fat in the scrotum and a higher fatty acid content in the palms and soles accounts for their immunity. That the loss of fluidity does not necessarily depend on a subnormal temperature is proven in rare instances by the occurrence of the condition with normal or increased temperature. *Histologically*, the chief changes are noted in the corium and hypoderm, where the connective tissue is contracted and the adipose tissue contains an abundance of fatty acid crystals. Parrot considers the process to be a drying up of the skin, with consolidation of its layers, together with atrophy of the adipose tissue.

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[1] For full discussion of the subject and bibliography, see monograph by Luithlen: Die Zellgewebsverhärtungen der Neugeborenen, Vienna, 1902; also Mraček's Handbuch, Bd. iii, p. 193. Leiberthal, Jour. Cut. Dis., 1918, xxxvi, p. 29; Sclerema Neonatorum and Scleroderma; see this article for discussion of the distinctive features between Scleroderma, Sclerema adiposum and Sclerema edematosum (bibliography).

[2] Diseases of Children, 1784, p. 76.

[3] Jour. Cut. Dis., 1909, xxvii, p. 87.

[4] Brit. Med. Jour., 1890, i, p. 403.

Treatment.—The treatment of both edema and sclerema neonatorum is by elevating the body-temperature (in an incubator, wrapping the entire body in wool, and by warm water-baths), and by improving the nutrition in every possible way (sterilized milk and stimulants by the stomach-tube, through the nose or pharynx). The body may also be well rubbed with warmed oil or camphorated alcohol. Brocq suggests friction with the warm hand in the direction of the return circulation.

Prognosis.—The prognosis is grave; in rare instances, when the sclerema has been partial, recovery has ensued.

EDEMA NEONATORUM.[1]

Synonym.—Sclerema edematosum.

Definition.—Edema of the newborn is the same as that of adult life. It presents special clinical features because of the undeveloped character of the infant's skin.[2]

It is characterized by the occurrence of an indurated tumefaction of the skin, most noticeable in the lower extremities of infants affected with impaired circulation.

Edema and sclerema of the newborn have long been confused. The distinction between them was first well established in 1877, when Parrot, under the title *Athrepsie*, first described with clearness the morbid condition now recognized as edema neonatorum.

Symptoms.—The disease, which is of exceedingly rare occurrence in America, is observed in infants prematurely brought into the world or at term, and of feeble vitality. Between the first and the third day after birth the child is found to be drowsy and difficult to waken, with the posterior and other parts of the thighs and legs, the hands, and the genital organs pallid, cold, livid, and retaining the impress of the finger as do edematous tissues in general. At this point recovery may ensue, but in severe cases the edema spreads, always more markedly in the lower portions of the body, and the skin becomes violaceous red, deep yellowish, or dirty looking. As the disease advances, the integument becomes more and more difficult of indentation. Meanwhile, the infant becomes more drowsy, its respirations fewer, its cry weaker, and its temperature lower. Death may ensue from a pulmonary complication, from diarrhea, or from any intercurrent disorder. Usually, the child passes into a state of coma. When recovery ensues, the edema becomes less marked and the indurated skin more and more impressible. A few days, in satisfactorily managed cases, suffice to restore the patient to a condition of health. In some instances, the edema begins in other portions of the body than those named; and occasionally there is a marked febrile reaction.

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[1] Full bibliography for edema neonatorum and sclerema neonatorum is given by Soltmann in Eulenburg's Real-Encyclopädie, 1899.

[2] Luithlen, Mraček's Handbuch, Bd. iii, p. 201.

Etiology.—The recognized causes of the malady are prematurity of delivery, cardiac feebleness, syphilis, exposure to severe cold soon after birth, poor hygiene, atelectasis of the lungs, and malnutrition from inability to take the nipple. Blacker[1] describes a case, seemingly typical, in which there was no evident etiology. The child at five weeks was perfectly well and properly nourished, but still retained the hard edema of the buttocks, thighs, part of the arms, and chest. The mother was always well, and the pregnancy, labor and puerperium presented no unusual features.

Pathology.—There is ordinary edema present. The skin presents the microscopical characteristics of a six to eight months old fetus.

Diagnosis.—The distinction between edema and sclerema neonatorum is not made without difficulty, the disorders greatly resembling each other. In sclerema the joints, and particularly the jaws, are immobile; the disease is likely to be generalized; the induration of the integument is greater; and there is no tendency to an edema chiefly marked in dependent parts of the body, as over the lower limbs. The color of the skin in the two disorders may be nearly the same. The pitting on pressure of the swollen skin is highly characteristic of edema neonatorum.

Scleroderma in infants is distinguished by the normal general condition of the infant and by the presence of solid plaques and nodes of various sizes situated chiefly over the back and gluteal regions and by the benign course of the disorder in many cases.

Treatment.—The treatment is that of sclerema neonatorum.

Prognosis.—About 90 per cent. of the affected perish; but with proper treatment recovery may occur when edema is not generalized.

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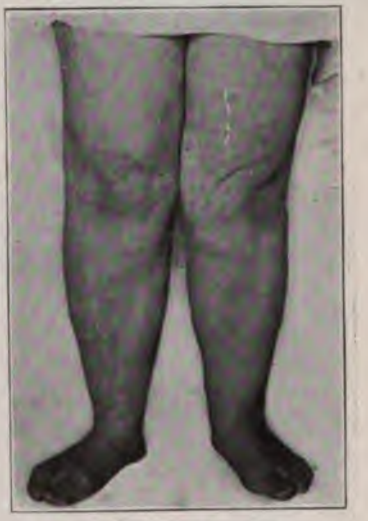
[1] Brit. Jour. Derm., 1898, x, p. 87.

HEREDITARY EDEMA OF THE LEGS (MILROY'S DISEASE).[1]

Synonyms.—Chronic hereditary trophedema, *Dystrophie edemateuse héréditaire* (Meige).

Definition.—The condition, as the title suggests, occurs as a solid, white, indolent, and persistent edema of the lower limbs, occurring, as a rule, in different members of a family for several generations. Milroy reported a series of 22 cases among 97 people in six generations. Hope and French record an instance of 13 members affected out of 42 people in five generations.

FIG. 169



Chronic hereditary trophedema.

Symptoms.—The lower limbs alone are involved, and the edema may appear shortly after birth, or the onset may be delayed until puberty or later. After becoming established, the edema remains permanently. It may involve only the ankles or the limbs to the knees (which is most frequent), or in certain cases the swelling involves the thighs as well. There is absence of pain and no evidence of local obstruction (thrombosis, embolism). In certain cases there may be acute attacks, accompanied by systemic symptoms. The cause of the disorder, aside from heredity, is unknown.

Diagnosis.—The disease is readily recognized when occurring in a family with other members affected. It is to be differentiated from other conditions producing edema, such as scleroderma, and various circulatory diseases and infectious processes.

Treatment.—Hope and French found constant bandaging of value.

ELEPHANTIASIS.[2]

Synonyms.—Elephantiasis Arabum, Pachydermia, Bucnemia tropica, Elephant leg, Barbadoes leg, Cochin leg, Spargosis fibro-areolaris. Fr., *Eléphantiasis*.

Definition.—Elephantiasis is a chronic hypertrophic condition of the skin and subcutaneous tissue, associated with lymph stasis, often resulting in marked deformity.

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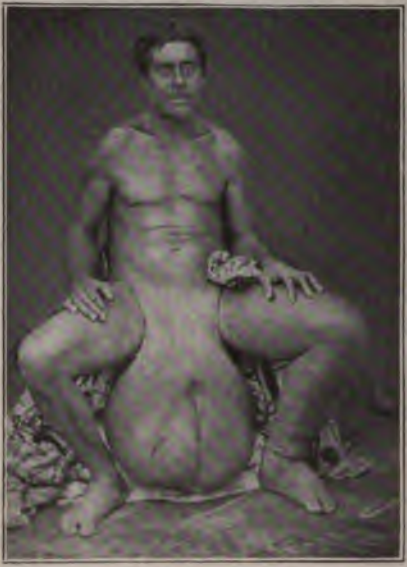
[1] Meige, Nouvelle Iconographie de la Salpêtrière, No. 6, 1899, p. 453; abstr. Brit. Jour. Derm., 1900, xii, p. 372. Milroy, New York Med. Jour., 1892, lvi. Hope and French, Quarterly Jour. of Med., 1908, i, p. 312.

[2] For bibliography, *Cf.* Hyde, Morrow's System (Dermatology), ii, p. 451; Manson, Tropical Diseases, 6th Ed., p. 706; Scheube, Diseases of Warm Countries, 1903, p. 399 (bibliography); Castellani and Chalmers, Manual of Tropical Medicine, 2d Ed., 1913, p. 1138.

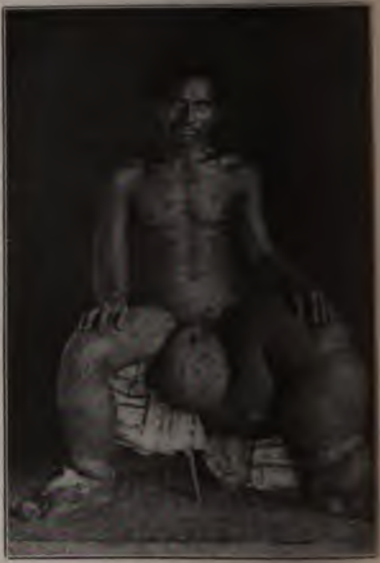
Symptoms.—The disease is endemic in tropical countries and sporadic elsewhere. The sites of election are the lower extremities, and external genitals, though the upper extremities, mammary and inguinal regions, and face may be attacked. In the endemic type the enlargement occurs following successive attacks of inflammation of the parts involved, together with constitutional disturbance. The area involved becomes red, swollen and painful, and not infrequently an accompanying lymphangitis is present, indicated by red lines extending along the course of the affected part. The nearby lymphatic glands participate in the process and are enlarged. This inflammatory process is usually described as being erysipelatous in character. Accompanying this there is elevation of temperature (elephantoid or filarial fever), chills, and in some cases delirium and prostration. With the subsidence of the acute attack the local symptoms disappear, except for a moderate edema which persists. After an interval of weeks or months there is a recurrence of the symptoms, both local and general, leaving a still more marked enlargement of the local parts. The attacks continue until the enlargement assumes huge proportions in some instances. When fully developed, upon the leg for example, the circumference may be several times greater than normal. The skin is tense and glossy, or roughened, scaling, warty or papillomatous and pigmented. Deep folds and sulci, especially about the joints, occur in which secretions, sweat and sebaceous matter are imprisoned, which emit an offensive odor. Weeping and crusted areas occur, and not infrequently

FIG. 170



Elephantiasis scroti.

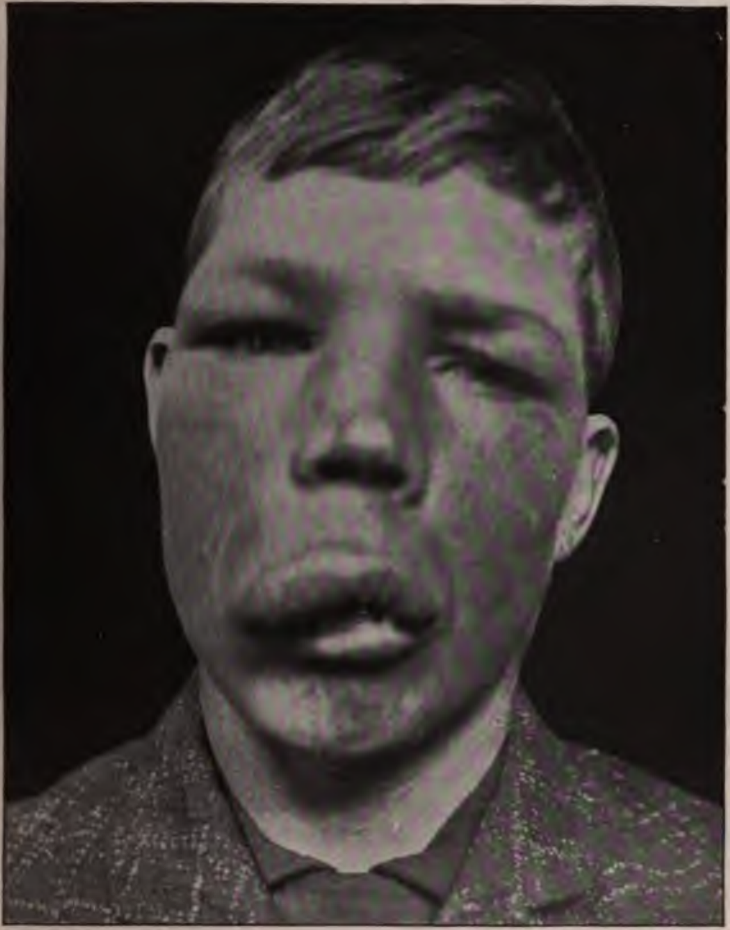
FIG. 171



Elephantiasis of the foot and leg.

ssuring and ulceration, both superficial and deep, are present. Vari-osities of the veins and lymphatics may be present, the latter giving se to vesicles which upon rupture discharge lymph. When fully eveloped upon the lower extremity the resemblance to an elephant's g is striking. Walking is impeded or impossible. When occurring the scrotum the enlargement is at times of such degree that it may ang below the knee. In these instances the penis is buried in a deep old and the urine flows through a gutter or canal from the latter organ o the surface. More pain is present in the scrotal cases during the ttacks, and in addition attacks of nausea and vomiting occur. There nay also be an associated epididymitis and inflammation of the permatic cord. The surface of the scrotum is furrowed and fissured,

Fig. 172



Elephantiasis telangiectodes of the upper lip and portions of the face.

and marked discharge of lymph may occur. Superficial ulceration occasionally is present.

In women the labia and clitoris may be similarly involved but to a less exaggerated degree. The subjective symptoms, except during the exacerbations, are mild compared with the objective manifestations. Tension from the weight of the large mass of hypertrophied tissue is the chief symptom. The disorder is usually asymmetrical, though at times both legs may be involved.

In the sporadic[1] form (*Elephantiasis nostras*) the symptoms are similar to the above, but less marked. There are recurrent attacks of inflammation of the part, erythematous, or so-called erysipelatous, with or without constitutional involvement. Usually the latter is absent or mild when present. Edema, lymphangitis and glandular involvement occur as in the endemic variety and after repeated attacks, as a rule, permanent enlargement results.

A vascular form of elephantiasis (*elephantiasis telangiectodes, telangiectatic lymphangitis, nevoid elephantiasis*) is occasionally seen. According to Virchow, this variety is congenital in origin, which develops later through overnutrition from increased vascularity. There occurs an increase in fibrous and vascular tissues in varying degree. The surface of the skin is not changed, but a lobulation may be detected by palpation (Crocker).

Lymph-scrotum (*Varix lymphaticus, Nevoid elephantiasis*).—Lymph-scrotum is characterized by enlargement of the scrotum, together with lymph varicosities and vesicles. These rupture and discharge a milky, or clear straw-colored coagulable fluid. Recurrent attacks of inflammation and elephantoid fever may occur, producing hypertrophy. In certain instances elephantiasis results.

Etiology.—Endemic elephantiasis has long been considered due to *Filaria sanguinis hominis* (*Filaria bancrofti*), introduced into the body through the bite of a mosquito (*Culex fatigans*). The disease occurs in filarial districts and, more often, among dark skinned individuals. The preponderance of cases is in the male sex. Children are rarely attacked. On account of the absence of Filaria in so many cases, recent observers attribute a minor role to this organism, though Manson[2] still strongly supports the Filarial view.

Elephantiasis nostras is believed by many observers to be due to infection with *Streptococcus pyogenes*.[3] Elliot states that *Streptococcus Fehleison* is the usual cause of this variety. It has followed lymphatic obstruction from many causes, such as by pressure of neoplasms and cicatrices, and extensive operative removal of lymphatic glands.[1] It may be associated with late syphilis.[2] Shattuck[3] states that sporadic lymphatic elephantiasis and endemic elephantiasis are not essentially different. Congenital cases are recorded by Moncorvo[4] and others.

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[1] Cases of non-filarial elephantiasis occurring in temperate latitudes have been reported by Brenstein and Price (following peritonitis), Brit. Med. Jour., March 16, 1907, p. 617. MacGregor, Ibid., 1898, p. 1597; Fowler, Brooklyn Med. Jour., February, 1897; Lase Chicago Med. Record, December, 1905; Southam, Brit. Med. Jour., May 3, 1902 (gigantic enlargement of the right lower extremity, with illustrations); Rogers, Med. Record, July 28, 1900; Elliott, Jour. Cut. Dis., 1917, xxxv, p. 17. (Elephantiasis Nostras. Reviews of the subject with report of a case. (Good bibliography.)

[2] Tropical Diseases, 6th Ed., 1918, p. 706. (Gives reasons for considering Elephantiasis a Filarial Disease.

[3] Unna, Histopathology, p. 493; Sabouraud; Whitfield, Allbutt and Rolleston's System, 1911, p. 10; and many others.

Pathology.—The skin is greatly thickened and all the elements show hypertrophy. The cut surface is white or yellowish-white, and exudes lymph on pressure, (Crocker). The lymph channels and veins often remain open. Microscopically, the fibrous tissue is greatly increased and extends deeply into the hypoderm. There is a marked increase in connective-tissue cells and nuclei and they are large and may present long processes. Elastin has been found increased by most observers. The vessels, arteries and veins are enlarged, their walls thickened and frequent thromboses are present. The lymphatics are enlarged and their walls thickened and the lymph spaces dilated. Elliot found the elastin decreased, confirming Unna's findings, and believes the primary changes occur in the bloodvessels rather than in the lymphatics. There occurs a perivascular cellular infiltration of round, plasma and mast cells, with some giant cells. In Elliot's case mast-cells were very abundant and plasma cells were practically absent. The sebaceous glands and hair follicles are atrophied from pressure late in the process. The epidermis shows varying changes, according to the stage or type of the process. Hypertrophy is at times noted while atrophy from pressure occurs in some instances. Hyperkeratosis and parakeratosis are both noted. Hyperpigmentation in the lower rete cells occurs in the pigmented cases and the epidermal changes are secondary. The microscopical findings show the process to be inflammatory and not merely a stasis.

Diagnosis.—Fully developed cases are readily recognized. The sporadic cases usually present but little difficulty. The recurrent attacks of inflammation with or without constitutional disturbances with gradual enlargement of the affected part are characteristic.

Treatment.—In endemic cases removal from the region where Filaria exist is important. Protection from the mosquito carrier is important. Proper hygiene and diet are essential Rest in bed during attacks of fever and quinin are important. Elevation of the limb or part involved, with local antiseptic dressings indicated in the case, are recommended. Elastic bandaging is valuable if skilfully employed. Surgical procedure is often necessary. Ligation of arteries and nerve stretchings have been employed with varying results. Fibrolysin has been used with benefit by Castellani.[5] Elliot suggests antistreptococcic serum in addition to local treatment.

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[1] *Cf.* Heidingsfeld, Amer. Jour. of Urology, May, 1912, viii, p. 263.

[2] *Cf.* Adamson, Brit. Jour. Derm., 1919, xxii, p. 161: Elephantiasis associated with Tertiary Syphilis. McDonagh, Ibid., 1912, xxiv, p. 24; Syphilitic Elephantiasis of the Scrotum (lymphangitis).

[3] Boston Med. and Surg. Jour., cxliii, No. 19, p. 718.

[4] Sur la Patholog. de l'Elephant. congenit., Paris, 1895.

[5] Jour. Cut. Dis., 1908, xxvi, p. 225.

ACROMEGALY.[1]

Acromegaly is a disorder involving several organs of the body and incidentally the skin, due to pathological changes (neoplasms) in the pituitary body.

Symptoms.—Transitory swellings due to vasomotor changes affecting the face and hands often precede for some time the classical manifestations of the disorder, which include cephalalgia, rachialgia, and paresthetic symptoms suggesting hysteria. These are followed by characteristic thickenings of the bones of the hands and the feet, spreading at times to the foot and the leg, and involving also the face, especially the under jaw. In well-marked cases the under incisors project beyond the line of the other teeth; the maxillary, malar, and occipital bones are thickened; the nose becomes long and broad; and the under lip, ears, tongue, and larynx are deformed by thickening. The fingers are large, blunt-pointed ("drum-stick deformity"), and tipped with nails that appear smaller than normal in comparison with the bulbous digits. The so-called "hexagonal face" is thus produced. In connection with these symptoms there may be interference with articulation, due to thickening of the tongue, a rough sound to the voice (from laryngeal changes), motor disturbances, and exophthalmos.

The skin and mucous membranes are often the seat of changes. In the skin there may be pigmentation, sclerosis, hyperidrosis (often coincident with polyuria), hypertrichosis, and the formation of keloid at points of trivial traumatisms. The nails are thickened, flattened, and grooved. The subcutaneous fat often is increased. At times there is an almost characteristic engorgement of the skin of the cheeks, which, taken together with the altered contour of the face described above, furnishes a classical picture.

MYXEDEMA.

Synonyms. Cretinoid edema, Cachexia strumipriva, Cachexia thyroidea. Fr., *Cachexie pachydermique.*

This disorder was first described by Sir William Gull,[2] in 1873, and it has since been studied, both abroad and in this country, by many observers.

A complete description of the disease and a *résumé* of the literature are found in the report of the Clinical Society of London for 1888, and in Murray's elaborate contribution to the same subject, in the *Twentieth Century Practice of Medicine*, vol. iv, 1895. The report embodies the results of the researches of a committee—including Ord, Horsley, and others—specially appointed by the Society to investigate the subject.

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[1] For bibliography, see Marie, Rev. de Méd., 1886, vi, p. 297; Marie and Marinesco, Trans. Derm. Cong., Berlin, 1890; Souza-Leite, De l'Acromégalie, Paris, 1890 (abst. of 49 cases); Collins, Jour. Nervous and Mental Dis., 1893, xx, p. 48 (bibliography). Arnold, Virchow's Archiv, 1894, cxxxv, p. 1 (with list of cases published since 1890); Shallcross, Phila. Med. Jour., 1901, vii, p. 771; and Kuh, Jour. Amer. Med. Assoc., 1902, xxxviii, p. 295 (full bibliography).

[2] Trans. Clin. Soc., London, 1874, vii, p. 180. Also see Hun, Amer. Jour. Med. Sc., 1888, p. 196 (notes on 150 cases in literature). For late reports, see Adami, Trans. Fourth Cong. Amer. Phys. and Surg., 1897 (review of subject and bibliography); Murray, Lancet, 1893, i, pp. 667 and 747; and Howard, Jour. Amer. Med. Assoc., 1907, xlviii, pp. 1226, 1325, and 1403 (historical, clinical, and pathological description, (with case reports).

Symptoms.—The disease occurs in both acute and chronic manifestations, usually after the fortieth year, and in women more often than in men. It may, however, first be noticed in childhood.

At the outset there is observed a gradually occurring, persistent and remediless anemia, succeeded in turn by hebetude, sluggishness of body-movements, and a characteristic change in the integument. The skin becomes dry, rough, yellowish, waxy, translucent, and firm, and does not pit on moderate pressure. The surface involved is commonly the seat of a fine furfuraceous desquamation, the mucous membranes often participating in the morbid process. In the cheeks there is usually perceptible a brawny redness, defined at times as a sharply circumscribed, pinkish flush, due to distention of the minute capillaries, extending quite to the lower eyelids, which may, as in Ball's cases, be wrinkled, boggy, and swollen. The eyes, for this reason, seem smaller and more widely separated. In consequence of the swelling and immobility of the features, the facies is characteristic: the broad, thick nose; swollen, pendulous, or even everted lips; expressionless eyes, and leathery cheeks producing upon the observer the impression of a mask. The skin of the other regions of the body participates in these changes, the backs of the hands, for example, becoming wrinkled or distended; the palms dry and fissured; the feet participating in the same morbid process. The hair becomes harsh and falls in nearly 90 per cent. of cases, even to the production of extreme baldness. The nails become discolored, grooved, and cracked, and the teeth often carious, fragile, or wholly lost. The mucous membrane of the mouth (gums, palate, pharynx) becomes tumid and fungous.

In the triangles at the side of the neck, and also posteriorly, are "bolsters" of fat. Pigment-alterations readily occur; moles increase in size; and the general tint of the skin may vary from that of dry parchment to the hue in Addison's disease. The gait is waddling and uncertain. The thyroid gland atrophies. Anesthesia is of common occurrence. The tongue, uvula, and fauces are often so thickened and immobile as to make speech slow and indistinct. The temperature is usually subnormal, the mental faculties seriously impaired, the sight and hearing altered, digestion vitiated, and the muscular strength greatly reduced.

The course of the disease is chronic, lasting for years, and terminating usually in a state of marasmus, with fatal issue.

Etiology.—The cause of myxedema is imperfectly understood, though its association with the abolition of the thyroid gland (by pathological change or ablation) is generally admitted. Stokes reports ten cases of acute myxedema following thyroidectomy. In these cases, beside the rapid occurrence of the symptoms enumerated above, there were convulsive seizures of an epileptiform character. Of four hundred and eight complete thyroidectomies analyzed in the Clinical Society's report, in sixty-nine myxedema developed. The result did not occur when part of a gland was left. The influence of heredity is distinctly shown in cases reported by Ball, Ord, Saville, and Taylor. The disease affects women more often than men, in the proportion of seven to one. Children are attacked, but the malady is more common in individuals between thirty-five and fifty years of age.

Pathology.—In nearly all cases examined the thyroid gland is found to be markedly reduced in size and its glandular structure seriously impaired by substitution of fibrous connective tissue for the epithelial cells lining its secreting acini. At first there is a small round-cell proliferation, which gives place to changes resulting eventually in a firm thickening of both the gland and its capsule. The lumen of the arteries becomes obstructed; and, in certain cases, new-formed lymphatic tissue is found surrounding the atrophied lobules.

Examination of affected regions of the skin discloses slight epidermal atrophy, replacement of connective-tissue trabeculæ with fine nucleated fibrillæ, a small-cell infiltration in the upper part of the corium, and an endarteritis obliterans similar to that recognized in the thyroid gland. The epithelium of the coil and sebaceous glands is the seat of swelling and proliferation, which eventually produce occlusion of the lumen of these emunctories and explain largely the cutaneous symptoms of the malady. The hair-follicles and the nerves (fibrosis of hair-pouch, perineuritis) may or may not be invaded by a similar process.

Diagnosis.—Cases of myxedema are readily distinguished from those of elephantiasis by the generalization of the symptoms, the nervous state of the patient, the fat-deposits, and the condition of the thyroid gland. Acromegaly involves the bones; in lepra there are commonly anesthetic symptoms or characteristic nodules.

Treatment.—The treatment of myxedema has hitherto aimed at amelioration of the symptoms by the employment of roborant and tonic measures; alkaline and sulphur baths; electricity and massage. The later method of treatment, however, is by thyroid-grafting, by administration of thyroids, and by hypodermatic injection of from 5 to 15 minims of liquid glycerin extract.[1] Whether there be employed the thyroid gland itself, the liquid extract, or the desiccated extract in tablet form, the results are satisfactory in so large a proportion of cases that the prognosis of this group of disorders presents no longer an element of gravity. The headache, faintness, loss of weight, neuralgias, and even albuminuria, with other symptoms immediately following the employment of the thyroids named above, do not seem to have an adverse influence upon the beneficial effects of the treatment.

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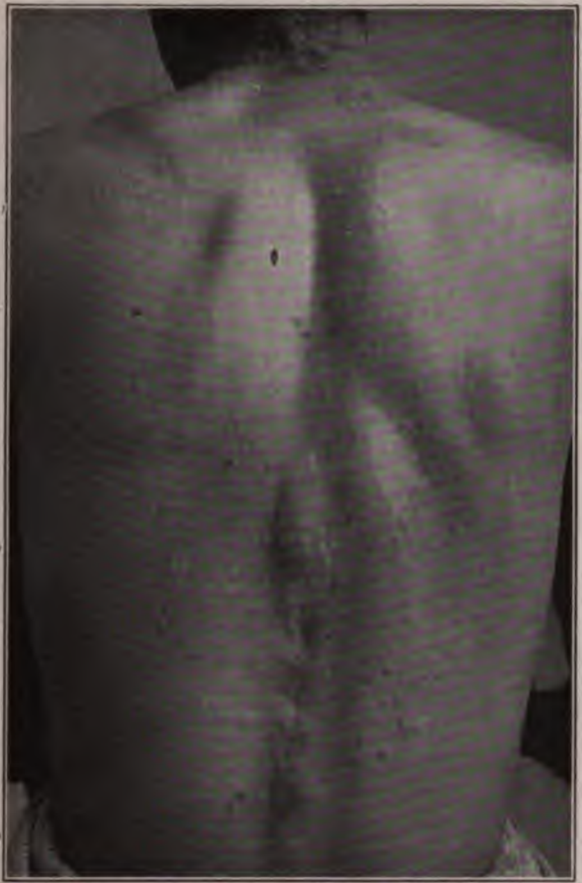
[1] *Cf.* "Feeding Thyroids in Myxedema," by J. J. Putnam, Amer. Jour. Med. Sc., August, 1893.

DERMATOLYSIS.

Synonyms.—Chalazoderma, Pachydermatocele, Cutis pendula, Lax skin.

There appear to be two well-differentiated disorders described under the above title, one form corresponding closely with the fibromata, and being so classed by Crocker and others; while the other presents chiefly a congenital abnormal laxation of the skin. In the fibroma variety, the masses of pendulous skin may depend from the face, neck, arms, and other situations, and may be associated with other molluscum tumors or occur independently (*Cf.* Fibroma Pendulum). Of interest in this connection is the report of a case by Wise,[1]

Fig. 173



Diffuse and disseminate dermatolysis. (Fred Wise and E. J. Snyder.)

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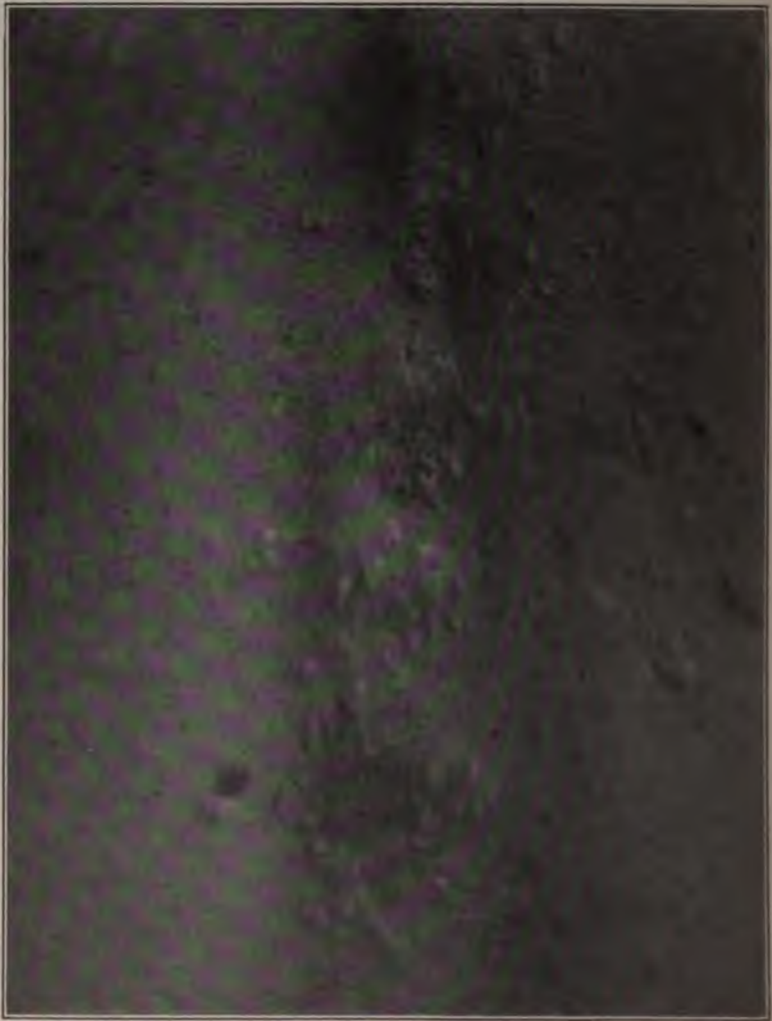
[1] Jour. Cut. Dis., 1914, xxxii, p. 139.

under the title, "Diffuse and Disseminate Dermatolysis;" a condition closely related to molluscum fibrosum, according to this writer. The case presented an eruption of slightly raised, pinhead- to pea-sized, soft, wrinkled, irregularly rounded, papular elements, discrete and grouped, and also areas of pigmentation. The chief sites were the back, the sides and back of the neck, and the posterior surface of the arms. Histologically, there was found a myxomatous connective-tissue new-growth.

Fig. 174



Diffuse and disseminate dermatolysis. (Fred Wise and E. J. Snyder.)

In the other type of cases considered under this heading (*elastic* *skin, cutis hyperelastica* (*Unna*)), the skin of patients affected is in a condition resembling that of the young of several among the lower animals (pups of large hounds, etc.), where enormous flaps of skin may be gathered up between the fingers and extended a foot or more from the underlying tissues. On releasing such folds, the skin retracts to its former position. The skin is usually thickened, but it may be stretched for a considerable distance; as, for example, the skin from the surface of the chest may be made to cover the entire face. Its color may be normal or hyperpigmented. In other respects no changes are noted. Usually, all the functions of the integument are preserved. The anomaly is congenital, but may be exaggerated, as in the case of freaks. Remarkable examples of this condition have been recorded by Duhring,[1] Crocker,[2] and others.

In a histological study, Unna (p. 984) and Williams found the elastin altered only in that it presented a more wavy course of the fibers, and they were longer, as the result of constant stretching. The collagen showed absence of the broad bundles in the deeper layers, and their place was taken by smaller and longer fibers. Both the larger bloodvessels and fine capillaries, as well as the nerves, showed an abnormally winding course. The capillaries, the lymphvessels, and lymph spaces were dilated, and the vessels frequently showed a leukocytic perivascular infiltration. The sebaceous glands were more voluminous than normal; the muscles were also increased in number. They conclude that the condition may be explained by the increase of the muscles, the winding condition of the vessels, and the absence of the broad bands of connective tissue usually found deeply situated.

CUTIS VERTICIS GYRATA (UNNA).

This condition was originally described by Jadassohn,[3] and later by Unna,[4] who gave it the above title. Subsequent cases have been recorded by Pospelow,[5] Veress,[6] Audry,[7] Bogrow,[8] Vorner,[9] Galant,[10] Lenormont,[11] and others. The disorder is characterized by furrows occurring in the scalp, over the crown and back of the head, presenting a condition which roughly suggests the convolutions of the cerebrum. There may be a few up to twelve or fifteen or more furrows in an individual case. In some of the recorded cases inflammatory processes have preceded the condition, while in others no such history was obtained. Wise and Levin[12] recorded the first American case in which there occurred five gyri separated by four furrows. Three gyri were well marked and visible while two were less distinct and demonstrable only by palpation. They ran transversely across the occipital region of the scalp, were parallel and most marked in their central portions, becoming less distinct toward the hair margins. Alderson[1] has also made a clinical and histological study of the condition.

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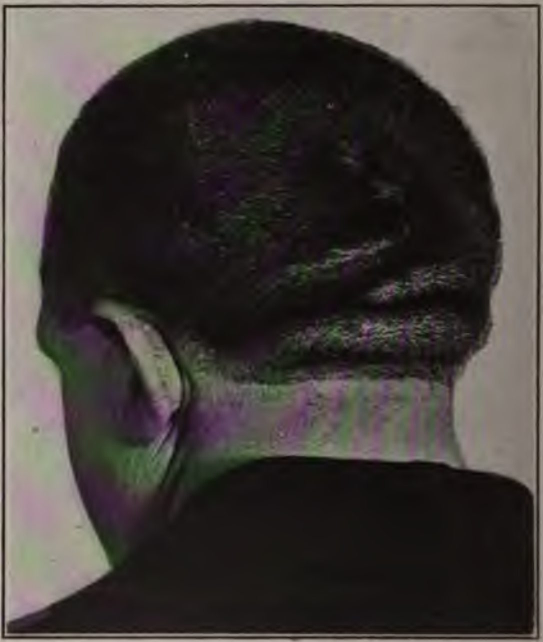
[1] Dis. of the Skin, 3d ed., p. 420.
[2] Dis. of the Skin, 3d ed., p. 949.
[3] Verhandl. d. Deuts. Derm., Qs. ix, Kong. zu Bern, s. 452.
[4] Monatshefte, xlv, p. 227.
[5] Russ. Zeit. f. Haut., 1909, xviii, p. 7.
[6] Zeitschrift, xv, p. 675.
[7] Annales, 1909, p. 257.
[8] Monatshefte, 1910, l, p. 16.
[9] Derm. Wochenschr., 1912, liv, p. 309.
[10] Corr.-Bl. f. Schweiz. aerzte, June, 1918.
[11] Annals, 1920, v, p. 225. La pachydermie du cuir chevelu. Review of literature.
[12] Interstate Med. Jour., May, 1918.

Fig. 175



Cutis verticis gyrata. (Wise and Levin.)

The etiology is not known. Some observers view the disorder a congenital affection developing later in life; others as a disorder d to inflammatory processes induced usually by microörganisms. Bogr believes the condition to be a congenital tendency to furrowing, inter fied by subsequent infection. Galant believes the condition to b congenital malformation. Lenormont describes the condition occurring in two types—one inflammatory the other congenital m formation.

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[1] Personal communication.

CLASS IV.

ATROPHIES.

ATROPHIA SENILIS.[1]

Synonyms.—Senile atrophy of the skin, Atrophoderma senile.

Definition.—This is the frequently recognized cutaneous degeneration peculiar to old age. The skin becomes colored in various shades of brown, either uniformly or in tolerably distinct pea- to bean-sized maculations over the face, the dorsum of the hands, the genitalia and the anus, and the lower extremities.

Symptoms.—The skin assumes a dull-yellowish hue, is seamed with furrows and wrinkles, is dry and inelastic, may desquamate slightly, and, losing the cushion of fat upon which it rested in earlier life, is either readily raised from the subcutaneous structures or depends from them in loose folds. The hairs on the affected areas may fall or may undergo regressive changes to the lanugo type. Pea- to finger-nail-sized, verruciform, dirty-yellowish accumulations of sebum and epidermis become visible, often in numbers, on the face and elsewhere, soft and readily scraped from the surface, or firmly adherent and scaly; or there may be small, pendulous, shrivelled pouches, representing fibromata that have disappeared. These epithelial growths, especially when irritated, are not infrequently the beginning of malignant epithelioma. Occasionally, they are commingled with whitish and grayish maculations or pinhead-sized and larger telangiectases.

In quantitative senile atrophy the pathological changes include: a general thinning of both corium and epidermis, as a result of which their characteristic interdigitations largely disappear; an increased pigmentation in the rete; a shortening of the hair-follicles; a dilatation of the sebaceous and coil-glands, the mouths of which often become blocked with epithelial detritus; the obliteration of some vessels and the dilatation of others; and the disappearance of the fat-cells from the meshes of the connective tissue.

In degenerative atrophy there may be fatty, amyloid, vitreous, and other changes of one or of several elements of the skin. Neumann described a senile atrophy with a granular degeneration and a vitreous swelling of the connective-tissue fibers. Schmidt, Reizenstein, and Unna think these changes due to a peculiar arrangement of the elastic fibers and their partial degeneration into *elacin*, or, in combination with the collagen, into *collastin* and *collascin* (Unna). These changes in the elastic fibers are manifested through the peculiar staining qualities of the latter, and in the light of modern technique are exceedingly interesting, as they occur not only in atrophy, but also in other cutaneous disorders.

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[1] For bibliography, see Himmel, Archiv, 1903, lxiv, p. 47.

Treatment.—Senile atrophy cannot be remedied, but it may often be prevented or postponed by securing for the skin and for all the tissues of the body the best possible nutrition and hygiene, and by protecting the skin from exposure to cold and other harmful influences. The nutrition of the skin may often be improved by the proper use of bran- or salt-baths, massage, electricity, or inunctions of oil. Cod-liver oil or other fats may usually be added to the diet with advantage. Care must be taken to protect all warty and other epithelial growths from irritation, with a view to the prevention of malignant changes. (See also Keratosis Senilis.)

DIFFUSE IDIOPATHIC ATROPHY OF THE SKIN.[1]

This disorder, described under many titles, occurs, as a rule, on the limbs, chiefly the lower, extending from the dorsal surface of the feet to the knees, and at times on the hands, forearms and arms, including the shoulders. It rarely involves the face. The extensor surfaces of the limbs are most markedly involved. The lesions have a tendency to be symmetrical, but several instances are recorded of their appearance on one side only.[2] In fully developed cases the skin presents a red, bluish-red, dark-red, or brownish-red, thin, atrophic, wrinkled appearance, in many cases likened to crumpled cigarette paper, or it may have a parchment-like, translucent look. Through it the superficial veins and tendons are visible. In some cases branny or flaky scaling is present, the skin being loose, easily movable, with the wrinkling most marked over the knees, elbows, and dorsal surfaces of the hands. In the earlier stages, red or bluish-red areas of varying size appear, which enlarge, new patches gradually developing, and by coalescence forming diffuse areas. In many cases the dorsum of the hand, the elbows, and the knees have been the initial site. In rare instances, inflammation and edema may occur. In other instances, the skin, instead of being lax, is tense, its surface smooth or rough, and yellowish-white in appearance, with a zone of reddish-brown discoloration. In these cases resemblance to scleroderma is marked. Not infrequently the two disorders coexist. In the affected areas the hairs are lost, and the physiological processes, such as sweating, are greatly diminished or absent. The subjective symp-

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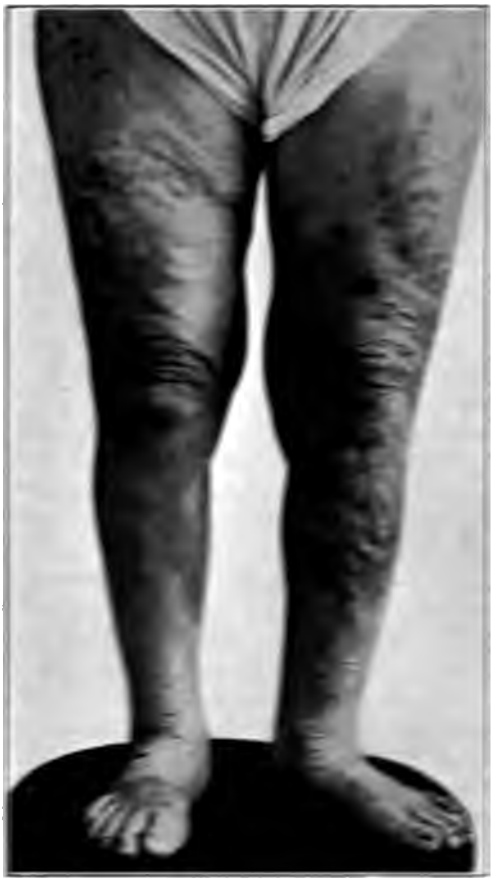
[1] For case reports, discussion of findings, classification, and full bibliography, see Buchwald (Archiv, 1883, p. 553); Herxheimer and Hartmann (Archiv, 1902, lxi, p. 57); Herxheimer (Jour. Cut. Dis., 1905, xxiii, p. 241, with particular discussion on differentiation between acrodermatitis and scleroderma); Herxheimer and Schmidt (ibid., 1911, xxix, p. 257. Further discussion on differentiation of scleroderma); Finger and Oppenheim (Die Hautatrophien, Wien and Berlin, 1910); Kanoky and Sutton (Jour. Cut. Dis., 1909, xxvii, p. 556. A comparative study of acrodermatitis chronica atrophicans and diffuse scleroderma with associated morphea atrophica, with bibliography); Irvine (Trans. Sec. on Derm., Amer. Med. Assoc., 1913, p. 133. A case report with histology, illustrated with 4 clinical and 8 histological cuts; with thorough discussion of idiopathic atrophy of the skin relative to classification. Many references); Wise (Jour. Cut Dis., 1914, xxxii, p. 295. Thorough report of a case, clinical and histological, with discussion and extensive bibliography). Wise, Archiv. of Diagnosis, 1915, vii, No. 1 The Differential Diagnosis between Acrodermatitis Chronica Atrophicans and Diffuse Idiopathic Atrophy of the Skin. Scholtz, Archiv. of Derm. and Syph., 1920, i, p. 565. Acrodermatitis chronica, with report of a case.

[2] *Cf.* Pollitzer, Jour. Cut. Dis., 1913, xxxi, p. 424. (Case demonstration. Lesions limited to one limb from ankle to thigh, preceded by a stage of infiltration.)

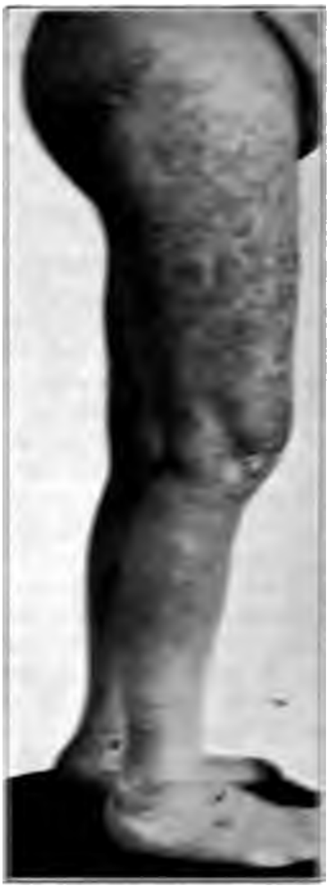
[illegible]



[illegible]

Front View.

[illegible]



[illegible] View.

toms are not marked, except there may be some itching or burning. Some of the cases described in the literature are examples of atrophic scleroderma, while others present atrophic changes due to [illegible].

[illegible] in the same patient. This patient [illegible]

[illegible]

In 1910 Herxheimer and Hartmann reported a group of twelve cases, with others taken from the literature, characterized by local atrophy, which was always preceded by inflammatory reaction. To this group they gave the name *Acrodermatitis chronica atrophicans*. In these cases the process began, as a rule, on the dorsal surfaces of the fingers or hand and spread upwards over the forearms and neck, rarely involving the face. The development does not necessarily include a continuous extension, as in many instances new lesions occur at a distance. The earliest change is a soft infiltration and thickening, of purplish-red color, or, as described later (1905) by Herxheimer, as tumor-like formations, characterized by nodules, deeply situated, of light- or dark-red color, and only moderately elevated. The infiltrations are raised, but not sharply outlined, and they gradually fuse into the normal surrounding skin. The sites of election are the hands, forearms, and elbows. After months or years, the affected areas (both infiltrations and tumor-like formations) undergo atrophic changes, becoming pale or yellow, thin, wrinkled, and presenting all the signs of the idiopathic atrophy above described. The clinical characteristics of this form which distinguish it are, therefore, the early, soft infiltrations and nodules, which are later succeeded by atrophy, both processes at times being coincident.

Erythromelie[1] (Pick) and *erythema paralyticum* (Neumann) are now recognized as idiopathic atrophy of the skin in the earlier stages.

Etiology.—The disease has been noted more frequently in females than in males, and usually occurs about the fortieth year of life. It is recorded as occurring from the fifteenth to the sixty-eighth year. The essential cause of the disease is unknown, although a process practically identical is not infrequently produced by syphilis, and also by scleroderma. Trauma, temperature-changes, exposure to heat and cold, and sudden chilling of the surface have all been noted as factors. Irvine's extensive case occurred in a blacksmith.

Pathology.—In the terminal stages all varieties show a similar structure, which includes marked atrophy of the epidermis and of all the structures of the corium, particularly the elastin. In the variety described as acrodermatitis chronica atrophicans, in the early stage the epidermis shows some hyperkeratosis. In the corium the reticular layer presents a cellular infiltration of connective-tissue and mast-cells, the papillary layer being similarly involved but to a less degree. The bloodvessels show inflammatory changes, with a perivascular cellular infiltration in localized areas. In the areas of infiltration the elastin is reduced and shows degenerative changes (granular and fragmentation). In the atrophic stage the rete is greatly thinned and its basal layer hyperpigmented. In the corium the papillæ are usually effaced, and there is present a more or less diffuse cellular infiltration.

Diagnosis.—The chief difficulty is to distinguish acrodermatitis chronica atrophicans from scleroderma. In typical cases, in the early stages, the soft infiltrations of reddish or purplish-red color of the first-named disorder are readily distinguished from the harder, whitish or yellowish infiltrations of scleroderma. In the final stages, the wrinkled, loose, somewhat scaly, atrophic skin in the one differs from the smooth, scar-like atrophy, which is hard and bound down, in the other. Atypical cases are practically indistinguishable both clinically and histologically.

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[1] Über Erythromelie (Pick), Klingmüller, Archiv (Festchr. Kaposi), 1900, p. 629.

Prognosis and Treatment.—The disease progresses to a certain point and then remains stationary. Except in rare instances, the general health is unaffected. The normal skin cannot be restored, hence the treatment resolves itself into that indicated by the individual case, both as to general and local management.

ATROPHIA MACULOSA ET STRIATA.

Synonyms.—Atrophic spots, Atrophoderma Striatum et maculatum Fr., *Vergetures.*

Definition.—This disorder is characterized by linear or small circumscribed areas of cutaneous atrophy.

Symptoms.—The disorder is seen most frequently in areas of the body where stretching or marked distention of the skin occurs, whether from physiological or pathological processes. It also occurs following certain diseases and one form, the macular, independent of either of these conditions. In the striate form the atrophy occurs in lines, often running parallel, frequently in curves or in an irregular manner. The affected skin is thinned, slightly depressed, presents a glistening surface and is white or of a delicate purplish hue. Occasionally small, macular lesions occur coincidentally with the striæ. They are unaccompanied by subjective symptoms. In the affected areas there is a partial or complete alopecia. These striæ are seen most frequently over the abdomen from distention in pregnancy (*linea albicantes, linea gravidarum*), from abdominal tumors and in obese individuals. They also occur over the hips, buttocks and upper portion of the thighs, and occasionally over the breasts. Their direction is transverse to the direction of tension.

Striate atrophy occasionally follows typhoid fever, scarlet fever, pulmonary tuberculosis and other wasting diseases. In these it may be symmetrically or asymmetrically disposed. Weber[1] reported a case of unilateral striate atrophy on the thorax associated with pulmonary tuberculosis. Adamson[2] described a marked case of macular and striate atrophy occurring in a patient suffering with tuberculosis of the lungs and lichen scrofulosorum, and another[3] extensive macular case, in which the trunk, neck, arms, elbows, and thighs presented numerous oval, fingernail-sized, whitish, bluish, atrophic macules. The patient was suffering with nephritis. Sections of the skin showed a well-marked cellular infiltration around the bloodvessels and a total loss of elastic tissue.

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[1] Brit. Jour. Derm., 1917, xxix, p. 247, with references.

[2] Ibid., 1910, xxii, p. 350.

[3] Ibid., p. 351.

Shepherd[1] described a group of cases of macular and striate atrophy following typhoid fever. The characteristic lesions were described as occurring about the knees, in which situation, both above the knee and over the patella, the striæ were well-marked, purplish in color, elliptical in shape, tapering to a fine point at each end, and in direction transverse to the axes of the limb and parallel to one another. The largest stripe was about six inches in length and one inch in breadth at its central portion. Shorter parallel stripes and bands occurred in the vicinity. One one side there were five stripes of various lengths and widths, some being merely lines. In addition to the colored stripes, there were several depressed spots of shiny skin, from pea- to bean-sized. On standing, the purplish color became deepened and the lesions resembled scars following burns. The skin was smooth and glistening, puckering transversely, and devoid of hairs. There was no change in subjective sensations. The skin felt dry, and a sense of depression was indicated to the finger. On flexion of the knees, the striæ presented a depressed appearance. According to Shepherd's further observation, these lesions did not present a stage of hypertrophy. Their first appearance was indicated as shiny, depressed, cicatricial-looking spots, which gradually enlarged. Several appeared to coalesce and by uniting formed a stripe. The color early was reddish, later purplish-blue, still later of a lighter color.

Bunch[2] under the title *Striæ patellares*, described two cases presenting symptoms similar to the above, each case following typhoid fever. Cockayne[3] reported three cases in which linear striate atrophy occurred following influenzal pneumonia.

Idiopathic macular atrophy has been recorded by a number of observers. Duhring[4] describes this condition as beginning in the form of erythematous spots or lines of rose or violaceous tint, made up of hyperemic capillaries, which sooner or later are succeeded by the atrophic changes described above. He quotes Wilson, Liveing and Taylor as having reported similar instances and calls attention to the similarity of some of these cases to morphea. Jadassohn[5] described an extensive macular case, with lesions varying in size from that of a lentil to a shilling, occurring chiefly over the extensor surfaces of the limbs; and Heuss[6] describes a similar case. Grindon[7] describes a case with dime-sized atrophic white spots on the back, chest, abdomen, and hip, associated with hyperpigmentation and vitiliginous spots.

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[1] Jour. Cut. Dis., 1901, ix, p. 59; describes his case and discusses others of similar type in the literature.

[2] Brit. Jour. Derm., 1905, xvii, p. 1; with references pertaining to these cases.

[3] Brit. Jour. Derm., 1919, xxxi, p. 93.

[4] Diseases of the Skin, 3d Ed., p. 442.

[5] Verhandl. der deutsch. derm. Gesellsch, Congress, 1891.

[6] Monatshefte, 1901, xxxii, Nos. 1 and 2; abstr. Brit. Jour. Derm., 1901, xiii, p. 198. a comparative study, with classification.

[7] Jour. Cut. Dis., 1913, xxxi, p. 270.

Weber[1] demonstrated a case of macular atrophy associated with vitiligo. The lesions were in various stages of evolution, from the erythematous macular to the atrophic stage, were of the size of a three-penny piece, and were situated on the trunk and extremities.

Macular atrophy is described in connection with many disorders. Its connection with lupus erythematosus has been the subject of much discussion. That some cases reported are the results of lupus erythematosus[2] there seems to be no doubt, and also that some of the reported cases are examples of guttate morphea. In the early stages of the latter disorder no difficulty is experienced, clinically or histologically, in recognizing the disorder. In the stage of atrophy, the conditions are practically indistinguishable. In addition, small atrophic spots occur as sequels in syphilis, lupus, atrophic lichen planus, and other diseases.[3]

Congenital atrophy may be diffused or localized, the latter being the more frequent. The disorder appears to be a congenital defect rather than an atrophy. When occurring in the scalp, the lesions are present at birth, and consist of slightly depressed, smooth, bald areas, coin-sized and larger, having well-defined borders. In the case described by Rihel and Vörner,[4] they found on microscopical examination that the epidermis was greatly thinned, that the corium also presented atrophic changes, the papillary layer and muscles being poorly developed, while the sebaceous and coil glands and hair follicles were absent.

Etiology and Pathology.—In what has been stated above, the causes of the various atrophies, so far as known, have been indicated. The most constant histological change occurs in connection with the elastin. In the distention cases the elastin appears to be ruptured. In other cases it shows great degenerative changes and is reduced in amount or completely obliterated. The remainder of the structures of the skin also show atrophic changes in varying degrees.

Prognosis and Treatment.—The scars are irremediable and no specific treatment can be outlined.

ATROPHY OF THE FATTY LAYER OF THE SKIN.

Gilchrist and Ketron[5] describe this unique condition in a girl eight years of age, in whom the disease had been present for six months. The earliest lesion was noted on the calf of the right leg. Others soon developed on the opposite leg, and later on the thighs. When examined the lesions were found to be limited to the lower extremities, extending from the inguinal region to the ankles. The inner and posterior aspects of the thighs were involved as well as the greater portion of the legs. The lesions ranged from pea- to palm-size and larger. The smallest area was pea-sized, irregular or round, bluish tinted, slightly depressed and macular, with a poor definition. From these lesions there developed larger, sunken, morphea-like patches which were round, irregular or band-like and reached the size of 10 cm. The depth of the atrophy varied from that which was scarcely perceptible in the smallest lesions to 2 mm. in the larger ones. The skin over these areas appeared normal, but was of a dusky bluish color. On palpation the affected areas were found to be hard, sclerotic, freely movable, irregular masses, which varied in size from those which were barely palpable to those the size of the finger. These masses were situated in the subcutaneous tissue and were beneath the atrophic macules and the apparently normal skin. In the larger areas where atrophy was complete these masses had disappeared. The skin over these areas was freely movable, could be picked up between the fingers and had the normal elastic feel. The skin over the smaller macules could not be picked up but was bound down to the deep infiltrations and presented a feeling similar to that of a scirrhus cancer of the breast. On the legs, where the atrophic process had developed to a greater degree there were palm-sized atrophic areas presenting a smooth or serpiginous border. These had become confluent with other patches, leaving here and there islands or dents of normal looking tissue which had a hard or edematous feel. The lesions on the legs differed from those first described in that the major portion of them were elastic and not bound down; the color of this portion was dusky-bluish, and veins beneath were visible. The edges, on the other hand, were indurated, pinkish-yellow or reddish, and covered with thin, white scales.

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[1] Brit. Jour. Derm., 1917, xxix, p. 273; case demonstration and report of two additional cases.

[2] Little, Brit. Jour. Derm., 1911, xxiii, p. 250; case demonstration of macular atrophy associated with lupus erythematosus. For discussion of the connection of lupus erythematosus with macular cutaneous atrophy *Cf.* Vignolo-Lutati, Monatshefte, 1907, xlv, pp. 329 and 404. A brief abstract is given in the Jour. Cut. Dis., 1908, xxvi, p. 192.

[3] *Cf.* Fabry. Derm. Wchnschr., 1917, Bd. lxiv, p. 1.

[4] Archiv, 1903, lxvi, p. 407.

[5] Jour. Cut. Dis., 1916, xxxiv, p. 728; "A unique case of atrophy of the fatty layer of the skin, preceded by the ingestion of the fat by large phagocytic cells (macrophages)."

The histological findings showed that the lesions in their development and evolution followed a definite course leading to the ultimate loss of the fatty layer without other permanent changes in the skin. The disease begins with the formation in the fatty layer of small nodules detected only by deep palpation. These nodules are made up of cells of a chronic inflammatory type. The inflammatory process spreads only in the fatty layer, the fat itself being taken up in the cytoplasm of large phagocytic cells (macrophages). After the fat is removed the infiltration is replaced by fibrous tissue, the contraction of which produces the visible atrophic macules which are bound down to the underlying masses. Later the fibrous tissue itself becomes absorbed, leaving a soft, elastic skin which, owing to the loss of the fatty layer, has sunken down directly onto the muscular fascia. The macrophages strongly resemble xanthoma cells, but the latter are produced by a cholesterol-fatty-acid-ester, while the macrophages in the present case are formed *in situ* from the normal fat.

The authors concluded that in this condition there is a gradually progressive change in the fat of the subcutaneous tissue due to some unknown cause. This metamorphosed fat acts as a foreign body in the tissues and induces foreign body inflammatory reaction. Macrophages derived from the fixed connective-tissue cells and the endothelium of the capillaries phagacytose this changed fat which they probably liberate in some soluble, inert form which can be excreted through the blood and lymph channels.

GLOSSY SKIN.

Synonym.—Atrophodermia neuritica.

This condition was first described by Paget,[1] and subsequently by Mitchell, Morehouse and Keen, in their experience with injuries occurring in the Civil War. It consists in peculiar atrophic changes occurring in the fingers. Paget's original description, quoted by Mitchell,[2] is as follows: "Glossy fingers appear to be a sign of a peculiarly impaired nutrition and circulation, due to injury of the nerves. In well-marked cases, the fingers which are affected are usually smooth, hairless, almost void of wrinkles, glossy, pink, ruddy, or blotched as if with permanent chilblains. They are commonly, also, very painful, especially on motion, and pain often extends from them up the arm. In most cases this condition of the fingers is attended with very distinct neuralgia, both in them and in the whole arm." In addition to the symptoms above described, vesicles and bullæ are observed in the skin, together with ulceration. The skin is usually dry, but in certain cases quite a marked hyperidrosis may occur. The nails are changed in a characteristic manner, being curved both longitudinally and transversely, and in some cases considerable thickening occurs beneath the free end. The skin may retract sufficiently to expose the nail-matrix. About the toe-nails ulceration occurs, and there is also a certain amount of deformity.

Etiology and Pathology.—The disorder occurs following injury to the nerves, when they are either partially severed, as in a gunshot wound, or a neuritis is subsequently set up. It has also occurred in maculo-anesthetic leprosy, in gout, and in rheumatism. The disorder is properly classed among the trophoneuroses, and is dependent upon the inflammatory condition of the nerves supplying the affected part. Turney[3] states that the vasodilators are specially involved, which is shown by the hyperemic condition of the skin and the profuse sweating occasionally noted in the affected areas.

Treatment and Prognosis.—The condition tends to spontaneous recovery, and in the interim protection from external injury and from extremes of heat and cold is necessary. The pain is usually relieved by cold, sometimes hot, applications. In certain cases, where intense pain is present, complete severance of the nerve has been found necessary.

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[1] Med. Times and Gazette, 1864, i, p. 58.

[2] Turney, Allbutt and Rolleston's System, 1910, vii, p. 82.

[3] Loc. cit.

PERFORATING ULCER OF THE FOOT.

Synonyms.—Malum perforans pedis. Fr., *Mal perforant du pied.*

Definition.—A trophoneurotic ulcer usually occurring on the foot, accompanied by a localized keratosis and sinus formation.

This disorder was first named by Vesigné, and has been thoroughly studied by Savory and Butlin,[1] Gasquel[2] and others.

Symptoms.—The disorder occurs on the feet, over points of pressure, usually the first or fifth metatarsophalangeal joint. The first objective symptom is a proliferating thickening of the epidermis, like a corn, usually single, occasionally multiple. Inflammation and suppuration occur beneath this thickening, spreading downward through the soft parts, occasionally to the bone. Gradually a sinus forms, reaching from the side of the corn to the deeper parts involved. When fully developed. the lesion consists of an ulcer or sinus. the surface of which is covered with a growth of granulation tissue, surrounded by a thick, horny collar. There is little discharge from the sinus, and it is unaccompanied in most cases by subjective symptoms; anesthesia of the surrounding parts is the rule. Occasionally, there is hyperesthesia, and at times a marked hyperidrosis of the part. Pigmentations, callosities, and nail changes are also noted. In rare instances the palm may be similarly involved.

Etiology and Pathology.—The exciting cause of the disorder is local injury by pressure in an area which presents a lessened innervation, either from disease of the spinal cord, as in locomotor ataxia, or the peripheral nerve, as in syphilis. leprosy, or other causes of neuritis. Gasquel[3] found that in 61 out of 91 cases there was a central nervous lesion, in 8 a peripheral nerve lesion, and 14 were diabetic. Of the 69 central nerve lesion cases, 32 had tabes, 17 general paresis, 8 symptoms of alcoholism, 4 traumatic disease of the cord. 8 various cord lesions, and one Friedreich's disease. Of the 91 cases, 84 were males. Of 79 cases, 3 were under twenty years of age, 4 between twenty and thirty, 22 between thirty and forty, 31 between forty and fifty. and 19 over fifty. Thomanczewski[4] recorded 10 cases associated with tabes, leprosy, diabetes, or cerebral and spinal disease, the location, character and course of the ulcers being practically the same in all cases. He believes the ulcers are due to trophic changes in the tissues, resulting from systemic disease, usually that of the central nervous system, though the local anesthesia and pressure are undoubtedly etiological factors.

Diagnosis.—The disorder is to be differentiated from simple callosities which have become secondarily infected, by the absence in the latter of anesthesia and other evidence of nerve derangement. It may also have to be distinguished in its early stages from plantar warts. In the later, ulcerating stages, tuberculosis and syphilis have to be excluded.

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[1] Med. Chir. Trans., 1879, lxii, p. 373.

[2] Thèse de Paris, July, 1890; a résumé of ninety-one collected cases.

[3] Loc. cit.

[4] München. med. Wchnschr., 1902, xlix, pp. 779 and 840.

Treatment.—The treatment is unsatisfactory. Rest of the part with curettage of all the diseased tissue will frequently cause the ulcer to heal, but it recurs after the patient begins to use the foot. Amputation is unsatisfactory, as recurrence in the stump has been not infrequently noted.[1] Mechanical devices to prevent pressure being exerted on the foot have been of service. The systemic disease should receive proper treatment.

Prognosis.—The outcome is doubtful.

MULTIPLE BENIGN TUMOR-LIKE NEW GROWTHS OF THE SKIN.

Under this title Schweninger and Buzzi[2] described a condition, examples of which have since been seen by Morris, Colcott Fox, Van Hoorn,[3] Stelwagon[4] and recently by Pusey,[5] and Pringle and McCormick.[6] The lesions described in these cases are bean- to small coin-size, bluish-white, slate-tinted, bladder-like formations, with delicate telangiectases over the surface of some. By pressure most of them can be forced into a hollow point in the underlying tissue, the tumor returning like a ventral hernia on removal of pressure. The small lesions are rounded and elastic, but as they become older they flatten and become darker in color, harder, and less resilient. They undergo spontaneous involution, leaving flaccid, pitted scars.

The lesions are found chiefly over the back, shoulders and arms, and occasionally on other areas. The course of the disease is slow, new lesions gradually developing as older ones undergo involution. They are unaccompanied by subjective symptoms, and have no effect on the general health. In sections excised from the skin, Buzzi found an absence of elastic tissue in the affected areas, with slight increase of its fibers at the margins, and round-cell infiltration was noted about the superficial bloodvessels and glands of the skin. Crocker states that he has seen similar lesions associated with cutaneous fibromata after their absorption. The microscopic picture is that seen in cutaneous atrophy. The treatment so far employed has had no effect on the growths.

MORVAN'S DISEASE.

Synonyms.—Syringomyelia, Analgesic paralysis with whitlow. Fr., *Panaris analgésique.*

Definition.—Morvan's disease is an affection of the spinal cord inducing cutaneous and other symptoms involving chiefly the upper extremities.

Symptoms.—The disorder may begin with pain in the arm followed by loss of muscular power, or analgesia may be the first symptom.

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[1] *Cf.* Lapowski, Jour. Cut. Dis., 1909, xxvii, p. 183.

[2] Internat. Atlas of Rare Skin Diseases, 1891, v, Plate XV.

[3] Quoted by Crocker, 3d Ed., p. 702

[4] 7th Ed., p. 667.

[5] Jour. Cut. Dis., 1917, xxxv, p. 582; clinical and histological report.

[6] Proc. Royal Soc. of Med., 1919, xii, p. 21; A case of the condition described as multiple benign, tumor-like new-growths of the skin.

Whitlows occur and are characteristic. They develop at irregular intervals; sometimes none appear for years and several may be present at once. In addition to whitlows, fissures, vesicles and bullæ occur. The skin is bluish or purplish and may be thinned or thickened. In time atrophy of the interosseous muscles, of the muscles of the flexors of the wrists and of the tissues forming the thenar and hypothenar eminences results. Contractures and ulceration occur and the terminal phalanges may be lost by necrosis. Marked deformity occasionally follows. An important feature is the loss of sensation to pain, heat and cold and the retention of that of touch. Other symptoms are hyperidrosis, diminution of, variability in or absence of the reflexes, visual changes, scoliosis and arthritic complications. The disease occurs usually upon the upper extremities and the lower are rarely involved. Only one extremity may be attacked, but usually it is one followed by the other. The disease develops insidiously, runs a slow course and may last for many years. Morvan's disease closely resembles anesthetic lepra and Zambaco[1] considers it a modified form of the latter.

Etiology and Pathology.[2]—The disease occurs chiefly in males and commonly between the ages of twenty and fifty. Traumatism, malaria, rheumatism and syphilis have been cited as possible causes. Neuritis and thickening of the neurolemma have been demonstrated in the nerves distributed to the affected parts, as well as sclerosis of the posterior cornua and columns of the cord. Cavities varying in contour and size are found behind the canal in the spinal cord which are produced by the absorption of gliomata.

Diagnosis.—The recognition of a typical case is not difficult. The paretic symptoms together with whitlows and the dissociation of sensation are characteristic. The resemblance to anesthetic lepra is close.[3] Scleroderma may have to be differentiated.

Treatment.—The treatment is symptomatic; general tonic treatment, including proper feeding, assists. Whitlows and cutaneous ulcers require antiseptic lotions and ointments. Surgical procedure may be necessary. Many patients live for years.

KRAUROSIS VULVÆ.[4]

This disorder was first described in 1885 by Breisky, though Weir of New York earlier described the disorder as ichthyosis, and it was later described by Tait. More recently Thibierge, after a large experience, classified the disease into several types, four of which are as follows: (1) a white form, leukoplasic; (2) a red, or inflammatory form; (3) a senile form; and (4) a postoperative form.

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[1] Trans. First Internat. Leprosy Congress, Berlin, 1889.

[2] Monod and Reboul, Archiv. gen de Méd., 1888, ii, p. 28; Prouff and Gombault, Gaz. hebdom, 1889, pp. 308-318; Joffroy and Achard, Archiv. de Méd. Exper., 1890, ii, p. 540. Sachs and Armstrong, New York Med. Jour., 1892, lx, p. 482.

[3] *Cf.* Dyer, New Orleans Medical and Surgical Jour., 1893-4, xxi, p. 81.

[4] Weir, New York Med. Jour., March, 1875; Tait: Serpiginous vascular degeneration of the nymphæ, 1877; Breisky, Zeitschr. f. Heilkund. Prag., March 15, 1885; Heitzman, Trans. Amer. Derm. Assoc., 1888, p. 64; Martin, Centralb. für. Gyn., 1894, xiii, pp. 310, 323 and 394; Reed, Amer. Gyn. and Obstet. Jour., New York, 1894, v, p. 556; ibid., Treatise on Diseases of Women, 1913, p. 702; Veit, Handbuch der Gyn., 1898, Bd. iii. p. 145; Jayle, Rev. de Gyn. et de Chir. abdom., July-August, 1906, p. 633; Thiberge, Annales, 1908, ix, p. 1; Baldy and Williams, Amer. Jour. Med. Sc., cxxviii, p. 528.

Symptoms.—Most of the patients are women either of advanced years or at the menopause, or younger women in whom this condition has been prematurely induced by the surgical removal of the uterus or ovaries. It also occurs as a sequel to other diseases, such as lichen planus, local pruritus and inflammatory changes suggesting eczema. In the simple form Brocq[1] states that the disorder is characterized by complete, or an almost complete disappearance of the labia minora, considerable diminution in the volume of the labia majora and the hood of the clitoris by retraction of the latter, of the vestibule and of the fourchette, and by rigidity of all the tissues of the vulva which becomes smooth and of a yellowish color. There is atresia and loss of sensibility. According to Reed, the earliest change is the occurrence of small, reddish areas around the ostium vaginæ. These are depressed, painful to the touch, and soon the vaginal orifice is narrowed, the elasticity of the tissues diminished, and the skin becomes pigmented, thin, translucent, tense and glossy, with the natural folds obliterated. In well developed cases many changes are noted. The parts may be symmetrically or irregularly involved, the labia majora appearing to have been obliterated when the disease is at its height, the labia minora absent or fused indistinguishably with the adjacent tissues. The nymphæ, the clitoris and its hood, and the vestibulum are involved in the atrophic process. In some cases the parts are whitish and dry. In others the skin is furrowed, wrinkled, pitted, or scale covered. At times the bloodvessels become visible beneath the thickened and glazed epidermis. The color of the retracted tissues may be whitish, reddish, pinkish, yellowish or bluish. A characteristic glistening appearance, shining as if varnished, may be present in all forms. In certain instances lesions resembling leukoplakia have been described, Jayle, discussing the relation of leukoplakia to kraurosis, calls attention to the absence of any tendency to retraction in the pure types of leukoplasia, yet believes that the two affections may coexist. Kraurosis is frequently preceded and accompanied by intense itching, yet this symptom has been absent in a number of reported cases, and in some there is a loss of cutaneous sensibility. A catarrhal discharge is recorded in most instances.

Etiology.—The cause of kraurosis vulvæ is not known. A preceding inflammatory condition and specific chronic catarrh of the mucous tract, gonorrhea and extirpation of the uterus,[2] are all given as etiological factors. Montgomery and Culver[3] stated that lichen planus of the sclerotic and atrophic type frequently induces the condition described as kraurosis vulvæ. In cases associated with itching, the scratching and resulting traumatism are cited as effective in its production. Reed states that the peripheral trophic nerves or their ganglia are to be considered the origin of the disease.

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[1] Annales, 1914-15, No. 11, v, October, 1915, p. 578.

[2] Olshausen, cited by Reed.

[3] Jour. Cut. Dis., 1915, xxxiii, p. 572; "Lichen sclerosis vulvæ."

Pathology.—Veit, Martin and Baldy and Williams agree in believing that kraurosis vulvæ is a purely inflammatory disease, produced by local causes. Jayle, recognizing in many cases the atrophic nature of the process, finds nothing in the local histological changes sufficient to explain the special phenomena exhibited. Thibierge believes that the process is a local manifestation of a senile, atrophic involution of the general integumentary system. The histology shows early hyperemia and exudation, with round-cell infiltration, and later atrophy of all the structures, including the appendages of the skin, followed by sclerosis.

Diagnosis.—The peculiar symptom-complex above described, occurring in a woman after the menopause, is readily recognized. An ordinary leukoplakia is not accompanied by the intense atrophy seen in this disorder.

Treatment.—The treatment is largely surgical. Medicinal treatment is indicated in the leukoplasic cases, in which cauterization has been practised with good results. Heitzman reported the entire relief of three patients by the local use of salicylic and pyrogallic acids, combined with curettage with a sharp spoon. The removal by surgical measures of the sclerosed mass, wholly or in part, followed by plastic operations, has been successfully practised.

Prognosis.—The prognosis is unfavorable for most cases. The course of the disorder is toward persistence and slow extension. The possibility of malignant degeneration is always to be remembered.

AINHUM.

Synonyms.—Dactylolysis spontanea, Banko-kereude, Sukha pokla, Quijila. Ger., *Absägen*.

Ainhum (from a Nagos term meaning "to saw") is an affection of the colored races chiefly, especially of the negroes of the West African coast, as also of the natives of the Soudan, of Algiers, Egypt, the Transvaal, and, next in frequency to Africa, of the inhabitants of Brazil, though it has been reported in Buenos Ayres, the Antilles, and British Guiana. The disease was first described and named by Dr. Da Silva Lima.[1] It is possible that Clarke[2] may have observed the same or a similar condition, described by him as a dry gangrene of the little toe.

American cases[3] have been reported by Herrick, Shepherd, Matas,

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[1] Gazeta Med. de Bahia, 1867, Nos. 13 and 15.

[2] Trans. Epidem. Soc. of London, p. 105.

[3] *Cf.* Da Silva Lima, Arch. of Derm., October, 1880, p. 367, and several other communications by the same author; Shepherd, Amer. Jour. Med. Sci., January, 1887 (with cut); Ref. Handbook of Med. Sci., Art. Ainhum, with four cuts; Hornaday and Pittman, No. Car. Med. Jour., September, 1881; Brayton, Jour. Amer. Med. Assoc., July 8, 1905 (with cut); Wellman, ibid., March 3, 1906, p. 636 (with analysis of etiologic theories); Wheatland, ibid., August 26, 1905, p. 631 (with cut); Moreira, Monatshefte. xxx, No. 8, p. 361, abst. Brit. Jour. Derm., 1900, xii, p. 334 (a histological contribution); and Herrick, Phila. Med. Jour., 1898, i, p. 246.

Hornaday and Pittman, Wheatland, and Brayton. Though most of the patients have been negroes, the disease has been recognized in a few cases in white subjects.

Symptoms.—Ainhum affects the smaller digits, chiefly the little toe, but also other toes and fingers, sometimes one or more of the digits of the same foot or of both feet being involved simultaneously or successively. The onset is by the development of a furrow or shallow groove on the plantar face of the toe or palmar aspect of the finger near the digito-plantar or digito-palmar web. This furrow gradually deepens and spreads in a circumlinear direction until the digit is girdled by a constricting and indurated ring in the form of a superficial, depressed gutter. The segmented portion of the digit becomes swollen, in consequence of the constriction, to twice or several times its normal size; and in time, usually in the course of two to ten years, the segmented part, at first resembling a small potato attached to a slender pedicle, drops from its original attachments. In this way, a species of spontaneous, bloodless amputation is effected. The nail of the member that is about to be detached by this process usually turns outward, the digit being commonly laterally everted. The changes in the segmented part, both in the nail and the tissues of the phalanges, are those naturally arising from strangulation of the member. The disarticulation may be effected at the first, second, or third joint; or even in the continuity of the phalanx. There is little pain, save such as is produced mechanically by the use of the foot or hand from which the digit depends. Occasionally, ill-conditioned and foul-smelling ulcers develop. In rare cases ulceration persists in the site of the wound left after separation of the digit.

In some instances trophic, vasomotor, and sensory changes, particularly of the limb where ainhum is progressing, are striking features of the case. The skin of the part may be pigmented, scaling, wrinkled, puckered, with wasted muscles, or covered with an unusual pilary growth, the tendon-reflexes obliterated, and sensibility decreased. Thickening and shortening of the foot, flattening of the plantar arch, and palmar and plantar keratoses may be conspicuous.

The disorder rarely attacks the white race. Dr. Hyde saw three cases of this type (one in France), where there was a coincident palmar and plantar keratosis, which he believed was of the same character as that recognized in the dense sclerotic ring which was working the amputation of the digit. Stelwagon[1] describes and pictures a case occurring in a patient the subject of pityriasis rubra pilaris. A typical case in a negress, involving the little toes of both feet, was seen by the author.

Etiology.—The disease occurs most often in male subjects of the African race, and in adults; but is recognized also in children, and quite rarely in the white race. Wellman reviews the several arguments urged in explanation of the disorder, and disposes of the theories that ainhum is a leprous lesion; that it is a trophoneurosis; that it is the result of self-mutilation; and that it is a circumscribed scleroderma. He believes with Manson that continued irritation produced by wounds of the foot in sharp grasses in the dark-skinned races, prone to the production of keloid, is responsible for the disorder. He also suggests that the chigger may be at times a factor in its production. In some instances the disease would seem to be hereditary, as there are reports of families every member of which has suffered. In other instances several members of two generations of a single family have developed ainhum.

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[1] Diseases of the Skin, 7th ed., 1914, p. 626.

Pathology.—The pathogenesis of the disorder is undetermined. Histologically, the constricting ring is seen to be composed invariably of fibrous tissue, surmounted by a thickened epidermis. The blood-vessels of the corium are thickened and enlarged, but their caliber is lessened by an endarteritis. Degenerative processes follow, both of a fibrous and fatty nature. The bones apparently suffer secondarily from the constriction.

Treatment.—In early cases the constricting band should be severed, while in advanced cases amputation is required. Prophylaxis is by protection of the feet.

Prognosis.—The disease progresses slowly; relapses are rare. The process in general ends with removal of the constricted member.

CLASS V.

PIGMENT ANOMALIES.

LENTIGO.

Synonyms.—Freckle, Ephelis. Fr., *Ephélide, Lentille;* Ger., *Sommersprosse.*

Definition.—Freckles are circumscribed collections of pigment, varying in size from pinhead- to split-pea or larger, having a yellowish, brownish or darker color, and occurring usually on exposed surfaces, such as the face, neck, forearms, and hands.

Symptoms.—This condition is due to excessive and irregular deposit of pigment in the skin, producing the pinhead- to bean-sized spots of circinate or of irregular outline, frequently grouped or even confluent, usually symmetrically distributed on those parts of the body ordinarily exposed to the light and heat of the sun, such as the face, the neck, and the backs of the hands, in persons of both sexes. In those individuals whose bodies are to a greater extent similarly exposed they occur upon the chest, the back, and over the extremities. In other persons they may be seen upon parts not thus exposed, such as about the genital region and the inner surfaces of the thighs; a fact which indicates that freckles are not always the result of the operation of the agencies noted above. Knowles[1] found multiple freckle-like spots of varying size over the trunk in a child twelve years of age, the duration of the disease being eight years. The microscopic findings were those seen in ephelis.

The lesions vary in color from light-yellow, salmon, or red to the deepest brown, and are most noticeable in those having red hair and delicate skin. Freckles occur rarely in infancy, but as a rule make their appearance about the sixth or eighth year of life. They are commonly observed in mulattoes, individuals of a race particularly disposed to anomalies of pigment-distribution. Once developed, the lesions may last throughout life without marked alteration; or may fade with each recurrence of the season of winter; or in milder cases may disappear. They usually share in the atrophic changes of old age, and, when persisting to that period, may then spontaneously disappear. They are entirely devoid of subjective sensations.

A special form of lentigo frequently affects the covered parts of the body in fibroma molluscum, in xeroderma pigmentosum, and in cases where freckles are found having a unilateral distribution, arranged in streaks like a linear nevus.[1] This variety, in most instances, is in all probability a very superficial nevus. In doubtful cases it may be distinguished by a microscopic examination.

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[1] Jour. Cut. Dis., 1912, xxx, p. 83.

Etiology.—Freckles are unquestionably produced and aggravated at times by the action of the light and heat of the sun, as common experience suggests; but it is evident that these forces must act upon a susceptible skin. Of one hundred sailors exposed in precisely similar situations on a long cruise, some of the number will be uniformly tanned and others deeply freckled. Attention has been called to the occasional occurrence of freckles on the protected parts of the skin. It is said that exposure to sea air and fog, without the direct action of the sun's rays, is sufficient to produce these lesions.

Pathology.—The only pathological change noted in ephelis by Cohn (confirmed by Unna) is a deep pigmentation entirely limited to the regions of the lower layers of the prickle-cells, with an iron-free, melanotic pigment both intra- and extra-cellular, and a few granules and pigment-cells in the superficial portion of the papillary body.

Treatment.—The treatment of freckles is that of chloasma and other pigmentations of the surface. Wertheim, of Vienna, advises:

℞—Hydrarg. ammon.,	gr. lvj;	3	75
Bismuth. magister.,	gr. lij;	3	50
Ungt. glycerini,	℥j;	30	M.

Sig.—To be applied every other night.

Bulkley employs:

℞—Hydrarg. chlor. corros.,	gr. vj;		4
Acid. acetic, dilut.,	f ʒij;	8	
Sodii boratis,	℈ij;	2	66
Aq. ros.,	f ℥iv;	120	M.

Sig.—To be applied night and morning, at first with gentle brushing; afterward by rubbing.

Hardaway touches each freckle with a rather stiff needle connected with the negative pole of a galvanic battery, and he finds the results satisfactory.

Unna recommends peroxid of hydrogen and oxychlorid of zinc in an ointment.

The several lotions and ointments outlined in the treatment of chloasma may be employed here. Most of the methods employed by charlatans for the removal of freckles depend for their success upon thorough blistering of the surface. Inasmuch as by this process the epidermis is removed, the pigment is also measurably removed with it, and the new epidermis is for a time free from blemish. But in all such cases the ultimate result is a deeper and more persistent pigmentation than that which was previously visible.

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[1] MacLeod, Brit. Jour. Derm., 1913, xxv, p. 191: Pigmented Nevi-like Freckles. Case demonstration, Royal Soc. of Med.

CHLOASMA.

It is usual to describe under this title various pigmentary changes which occur both in localized patches or in more extensive areas, even to a diffuse discoloration of the entire cutaneous surface. The disorder may be idiopathic or symptomatic. When these areas are well defined and occur about the face, or even in other parts, they are popularly called "liver spots;" or, when the pigment is of a very dark or blackish shade, it is frequently known as *Melanoderma*. The color in all chloasmata varies from a scarcely perceptible staining of the skin, which requires a strong light for its detection, to a deep yellow, a yellowish-green, a chocolate-brown, or a blackish shade.

Chloasma Idiopathicum.—This variety of pigmentation is produced by agencies operating externally, in consequence of which an undue afflux of blood is persistently determined to any portion of the skin. Among these externally operating agencies may be named pressure and friction (as over the part covered by the pad of a truss); traumatism (as after the severe scratching of the skin affected with lice, eczema, or scabies); heat (as in diffuse tanning of the face or sunburn following exposure to the solar rays). Other agencies operating externally, frequently classed as toxic in their action, are applications of mustard, capsicum, cantharides, and other articles capable of producing either vesication or pustulation of the skin-surface. Persistent or even permanent pigmentation of the skin upon the face, shoulders, and breast, especially of young women, may be produced by the repeated applications of such topical medicaments.

Chloasma Symptomaticum.—The symptomatic varieties of pigmentation are the result of disorders either systemic or those involving internal organs. They occur as either circumscribed or diffused, localized or generalized spots, mottlings, stainings, or masks of the skin, and they vary in color from the lightest to the darkest shades. One of the most common, and at the same time the most important, of these varieties is

Chloasma Uterinum.—Chloasma uterinum is so called because of its frequent association with certain physiological or pathological conditions of the uterus, both among married and single women. Thus, in pregnancy, sterility, hysteria, chlorosis, ovarian disorders and tumors, and functional derangements of the uterus, there can be observed at times a facial discoloration extending equably over the forehead and reaching nearly to the line of the hairs at the scalp, in the form of a faint or a decidedly reddish-yellow or deep-brownish tinge. At other times the discoloration is macular and asymmetrical, involving the eyelids, the cheeks, the lips, or the chin. When the chloasma assumes the mask-like form, it is usually most pronounced over the forehead, but it may involve the whole facial region, being less distinctly defined below than above. Similarly, the well-known changes occur in the areola of the nipple, in the linea alba, and about the external genitalia. Extensive cases of pigmentation during pregnancy, which cleared up on the termination of this physiological state, are recorded by a number of observers. The pigmentation is unaccompanied by subjective sensations.

Other conditions producing symptomatic chloasma are tuberculosis, syphilis, cancer, chronic alcoholism, malaria, Addison's disease, Graves' disease, and other disorders. Among the cutaneous diseases capable of producing skin-pigmentation may be named scleroderma, lepra, lichen planus, psoriasis, eczema (especially *e. venis varicosis*), and pediculosis.

In **Addison's Disease** the pigmentation may be general or partial, and in the latter case is without definite lines of demarcation. It is most commonly pronounced over the face and neck, the scrotum, the groins, the axillæ, the nipple and areola. The hairs become coarse and dark; and dark- or grayish-brown patches are at times visible over the mucous surface of the lips, the gums, and other parts of the mouth. The bronze or mulatto-like color of the skin is intensified by stimulation or erosion of the cutaneous surface and by exposure to light. In these cases there are generally marked asthenia and a feeble pulse, with anorexia and other signs of gastro-intestinal disorder. When the result is fatal, there may or may not be recognized pathological alterations of the suprarenal capsules. When examined chemically, the pigment is seen to be iron-free.

In **Graves' Disease**[1] the pigmentation may occur as freckle-like patches or diffuse, usually most marked in regions which have normally more pigment than the general surface of the body.

Hemochromatosis indicates a pigmentation of the skin and viscera associated with hypertrophic cirrhosis of the liver and extensive sclerotic changes in the pancreas. It may terminate in glycosuria, and in the terminal stage it is called *Bronze diabetes*.

The long-continued administration of arsenic for the relief of nervous disorders in adults and children is frequently followed by a characteristic dull-brownish or dirty-colored discoloration of the skin of the neck and chest. In some instances the pigment may become generalized, with a tendency to persist. The pigmentations from this cause resemble somewhat closely that found in Addison's disease, but here the mucous membranes are free. In addition there may be, in connection with the arsenical pigmentations, palmar or plantar keratoses and hyperidrosis, as well as keratoses elsewhere on the body (*Cf.* section on Dermatitis Medicamentosa).

Mongolian Pigment Spots.[2]—This is a congenital condition characterized by the presence of dark-bluish spots on the lower sacral region, found more commonly in the darker races. The lesions appear as blackish, bluish, or mulberry-colored, smooth, non-elevated areas. The skin is normal in texture, and the lesions are unaccompanied by subjective symptoms. In shape they are roundish, oblong or squarish, and may be single or multiple (five or six or more). In size they vary from one-half a centimeter to 12 centimeters or more. They may be well defined, or gradually fade into the surrounding skin. Their common site is the lower spine or sacral region, and buttocks, but they may occur all over the body. They are present at birth, and disappear about the third or fourth year of life.

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[1] For a review of the cutaneous changes seen in Graves' disease, with bibliography, see Dore, Brit. Jour. Derm., 1900, xii, p. 353; Hyde and McEwen, Amer. Jour. Med. Sc., 1903, p. 1000; Hyde, Brit. Jour. Derm., 1908, xx, p. 33.

[2] American Anthropologist, March, 1907. Ashmead, A. S., Jour. Cut. Dis., 1905, xxiii, pp. 203 to 214: The mulberry-colored spots on the skin of the lower spine of Japanese and other dark races a sign of negro descent. Castellani and Chalmers, Tropical Medicine, 1913, 2d ed., p. 1615.

Etiology and Pathology.—The chloasmata are all due to excessive deposit of the natural pigment of the body in the rete mucosum of the epidermis. The causes vary, as outlined above, in the different varieties. The histology is that of lentigo, except that the pigment deposited, instead of being localized, is diffused, but found in similar location in the lowermost layers of the rete.

From the above discolorations, which are due solely to the deposition in excess of coloring matter normally existing in the skin, it is necessary to distinguish the various dyschromias which are due to the introduction into the integument of coloring substances, either supplied by other portions of the body or foreign to it. Thus, in icterus the bile may color the skin from a light-yellow to a dark-chrome shade, the duration and severity of the cutaneous symptoms depending upon the nature and gravity of the hepatic disease. This condition is frequently accompanied by itching in various grades of severity, the exact causes of which are obscure.

Diagnosis.—The diagnosis of the various chloasmata is readily effected by observing the persistence of the discoloration under pressure; the absence of all symptoms of hyperemia, inflammation, and secondary changes in the skin; and the characteristic shades of color presented to the eye. In tinea versicolor there is a slight furfuraceous desquamation, and the lesions occupy by choice the regions under the clothing. In addition, on microscopic examination, *Microsporon furfur* may be readily demonstrated. In determining to which particular category the chloasma belongs, a reference must be made to the various symptoms above described.

Treatment.—In all the symptomatic pigment-anomalies the indications for treatment are presented by the disease which begets the cutaneous disorder.

The local treatment of both the idiopathic and symptomatic varieties of the affection demands the use of external applications which will hasten the physiological reproduction of the epidermis, substituting thus new and unpigmented for old and pigmented epithelia. This process must also be accomplished without the artificial production of such a hyperemia as will tend to add to the very coloration which it is attempted to relieve. The substances used for the slow accomplishment of this end are borax, sulphur, tincture of iodin, potassium and sodium hydroxids (including the soaps of these alkalies), and the mercurials. None of these substances is more generally employed than corrosive sublimate, which constitutes the basis of most of the cosmetic lotions sold in the shops.

The following formulæ are given by White for use in the evening. The preparations in each case should be left upon the affected surface during the night, and be removed by a soap-and-water washing in the morning. They are to be used for weeks in succession, but only after a cautious preliminary testing of the sensitiveness of the skin to their action. To avoid the possibility of error, the practitioner would do well to order a poison-label upon all vials containing the sublimate:

℞—Hydrarg. am.,					
Bismuth magister.,	āā	ʒij;	āā	8	
Amyl.,					
Glycerin.,	āā	℥ss;	āā	15	M.

℞—Ammon. chlor.,	ʒss;	2	
Aq. Colognien.,	f℥j;	30	
Aq. dest.,	Oss;	240	M.

℞—Hydrarg. chlor. corros.,		gr. vj;			4
Acid. hydrochlor., dil.,		fʒj;		4	
Glycerin.,		f℥j;		30	
Alcoholis,					
Aq. ros.,	āā	f℥ij;	āā	60	
Aq. dest.,		f℥iv;		120	M.

The following formulæ for ointments are given by Kaposi:

℞—Hydrarg. ammon.,					
Sodii biboratis,	āā	℥ss;	āā	15	
Ol. rosmarin.,		gtt. x;			66
Ungent. simpl.,		℥j;		30	M.

℞—Acid. boric.,					
Cer. albæ.,	āā	ʒj;	āā	4	
Paraffin.,		ʒij;		8	
Ol amygd. dulc.,		℥j;		30	M.

Van Harlingen recommends:

℞—Hydrarg. chlor. corros.,		gr. vss;			4
Zinci sulphatis,					
Plumbi subacetat.,	āā	ʒss;	āā	2	
Aq. dest.,		f℥iv;		120	M.

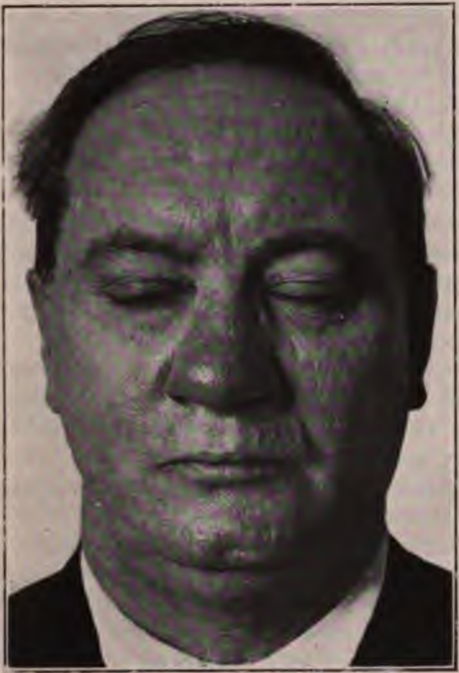
Sig.—Lotion for external use, morning and evening.

Other applications advised are: alcohol, followed by the use for several hours after of a plaster of ammoniated mercury; 2 parts of magnesium carbonate and zinc oxid, 4 parts of pure kaolin and glycerin, and 10 parts of vaselin; chloroform 100 parts, chrysarobin 15 parts (Leloir); hydrogen peroxid; diluted acetic, hydrochloric and nitric acids and phenol; 1 to 2 parts of salicylic acid, in paste or powder, to 20 parts of base; and solutions of mercuric chlorid in collodion, 1 part to 30, employed with great caution.

The rapid removal of pigmented patches is accomplished, in Vienna, by covering the part with strips of linen dipped in an aqueous or an alcoholic solution of mercuric chlorid of the strength of 4 grains (0.25) to the ounce (30.), with which solution the dressing is also occasionally moistened. Vesiculation is usually accomplished in about four hours, when the serum is evacuated by puncture, and the detached epidermis is covered with an inert dusting-powder. The resulting crusts fall in about eight days. The procedure is attended with danger of producing in the end the precise deformity which it seeks to remedy, a danger explained above.

Prognosis.—The prognosis is in all cases uncertain. There is strong reason to believe that the local treatment of these dyschromiæ is, in the long run, ineffective. Those methods which effectively and brilliantly accomplish the desired end temporarily are almost invariably followed by deeper pigmentation than that which it was attempted to remove; those operating more slowly have, probably, a less speedy but scarcely more disguised sequel. The treatment intelligently directed to the cause of each discoloration is that which in the end proves most satisfactory.

FIG. 178



Argyria of the nose due to the application of a silver nitrate pencil.

Argyria.—A bluish, bluish-gray, slate-colored, or bronzed coloration of the skin may result from ingestion of silver nitrate. Argyria is most commonly the result of the administration of the drug in the treatment of epilepsy, but it is said to have also resulted from the topical application of silver crayons to the throat, to the conjunctivæ, and even to the skin. Under what form the silver produces this effect, whether as an albuminate or other salt, is not known. The deposition, however, occurs in the form of minute particles of the metal in the connective tissue of the derma. The discolorations are most evident upon the parts of the skin exposed to the light, as the face and hands; but the chest and the lower extremities may be stained similarly. The connective tissue of the viscera is at times also involved, showing thus that the action of light is not essential to the production of the dyschromia. Two cases are reported as relieved by the administration of potassium iodid.

Localized argyria may follow the local application of any of the organic silver compounds. Olsen[1] recorded the case of a young woman in whom bluish-green and slate-gray pigmentation occurred on the right lower eyelid, the right side of the nose and cheek. The patient had previously suffered a fracture of the nose with injury at the same time to the soft tissues of the eyelid and cheek. Argyrol was instilled into the eye from which it permeated the injured tissues inducing the pigmentation. With the internal administration of hexamethylenamin the pigmentation was largely overcome.

Tattooing.—By the process of tattooing, mineral and vegetable substances are directly introduced into the corium by means of needles, for the production in the skin of various devices in colors. Individuals whose entire integument has been thus artificially covered with figures of different patterns by tattooing with indigo, vermilion, and cinnabar are from time to time publicly exhibited. The results are indelible. Post-mortem these pigments have been discovered not only in the derma, but also in the lymphatic ganglia nearest the site of their introduction.

The condition produced by *powder-stains* is practically identical with tattooing. Here particles of powder and other extraneous material are deposited in the skin.

Treatment.—A method of removing tattoo-marks and pigmented nevi successfully employed by French dermatologists consists in tattooing the region, previously rendered aseptic, with a solution of 30 parts of zinc chlorid to 40 parts of water. If properly done, the resulting inflammation is slight, and after a few days there forms a superficial crust, which remains about a week and then falls, leaving a slight scar, which becomes almost imperceptible. This method succeeds in a few cases, but requires skill and care in its application in order to obtain good results and to avoid suppuration and deep cicatrization.

Blue Pigmentation.—Gottheil[2] reported a case of blue pigmentation, associated with atrophy of the skin, following hypodermic injections of cocain. Other cases are recorded of bluish pigmentation following injections of morphin and of cocain, and are attributed either to extraneous pigment, such as carbon or impurities contained in the drug itself, or to particles of metal from the needles used by patients.

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[1] Trans. Amer. Med. Assoc., Sect. on Derm., 1917, p. 115. Argyria localis due to organic silver preparations. Literature.

[2] Jour. Cut. Dis., 1912, xxx, p. 1 (with references to allied cases).

Treatment.—The treatment of all these conditions is most unsatisfactory. Various methods have been employed with varying success.

Generalized Pigmentation.—A form of generalized pigmentation was described by Wende and Bauckus[1] as occurring in two children of the same family. The pigmentary change began at the end of the first year in both cases. The areas first involved were the face and scalp, then the trunk and finally the entire cutaneous surface. In the older child the pigment increased for four years, then gradually faded. In certain areas, face and trunk, the color became almost black. At different times and in different areas the color change varied from yellow-bronze to negro-black. A peculiarity in both instances was a lessening in the degree of the discoloration in the presence of light. The younger child was exhibiting the same progress history as the older at the time of the report. Histologically the epidermis showed much increase in pigment in the cells of the rete as well as many migratory pigmented cells. In the corium large numbers of abnormal pigmented cells were seen throughout its entire area. The collagen and elastin also showed changes. The fibers were short, tortuous, dense, thick and compact. In the papillary layer it appeared as though the fibrous tissue was undergoing hyalin degeneration. In certain areas chromatophores could be traced through the corium into the epidermis. The deeper glands, sebaceous and sweat, were surrounded by pigmented cells. Plasma and mast-cells were absent, and only an occasional epithelioid cell or lymphocyte was seen.

The occurrence of a generalized pigmentation of this type presenting the peculiar light changes is unique.

Albinismus.—**Synonyms.**—Albinism, Congenital leukasmus, Congenital achromia, Congenital leukopathia, Congenital leucoderma. Fr., *Albinisme.*

Definition.—A congenital loss of pigment, partial or complete, involving the skin, hair and eyes.

Symptoms.—In partial albinismus or leukoderma, a congenital absence of pigment is noted in variously sized and shaped areas. The areas are usually circular, oval or irregular. The skin in the area is white or may present a pink tinge. The areas are commonly of irregular distribution, though rarely they may follow the course of the cutaneous nerves. The patches are usually well defined and are not surrounded by a hyperpigmented border. Hairs in the affected regions are white. As a rule, the patches remain without change throughout life. Rarely they enlarge or multiply. Very rarely the patches become pigmented. The term "pied" or "piebald" has been applied to negroes suffering with partial albinism.

Complete albinismus is seen in persons known as "Albinos" (Kakerlaken; Dondos). Here the entire cutaneous surface is devoid of pigment and is milky white or pink tinted. The pink or rosy tinge is imparted from the color of the blood, and may be of fine texture. The hairs are silky white, yellowish-white or, in rare instances, red. The iris is transparent or pinkish. The absence of pigment in the choroid renders the pupil red or pinkish-red. Photophobia and nystagmus are commonly present.

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[1] Jour. Cut. Dis., 1919, xxxvii, p. 685.

Aside from the absence of pigment the skin shows no changes, either structurally or physiologically. The subjects of complete albinismus may be normal, but more often they exhibit imperfect mental development and are subnormal physically. The condition remains unchanged, as a rule, throughout life.

The disorder often occurs in families; usually several members are affected. It rarely occurs in succeeding generations. Alternation in birth of normally pigmented and Albinos in the Negro race is recorded. Marcy[1] reported the following: The parents were Negroes; the first two children were males and black; the next two females Albinos; then a black female child and, lastly, a male Albino. Sym's[2] recorded a family in which the first, third, fifth and seventh children were Albinos, the others normal.

The condition occurs in the white as well as the colored races, and aside from its occurrence in families, suggesting hereditary influences, no cause is known and the condition is remediless.

Vitiligo.—**Synonyms.**—Acquired leukoderma, Leukopathia, Leukasmus, Acromia, Piebald skin.

Definition.—Vitiligo is an acquired cutaneous achromia characterized by variously sized and shaped single or multiple patches of milk white color, usually presenting hyperpigmented borders and a tendency to enlarge peripherally.

Symptoms.—Absence of pigment producing color changes is the only symptom in vitiligo. The patches are round, oval, or irregular in shape, of milky-white color, and have well-defined hyperpigmented borders. They are neither elevated nor depressed below the general surface. They vary in size from small spots to palm-sized areas and enlarge slowly but continuously as a rule. New areas form and by coalescence extensive areas become invaded, which in rare instances become generalized. The patches may be few or numerous and the sites of selection are the face, neck and backs of the hands. After these the genitals, trunk and the extremities. The hairs in the affected areas may or may not be depigmented. Usually they are and when occurring in the scalp the whitened hairs in patches are conspicuous. The course of the affection is slow, after progressing for a certain length of time it may remain stationary for long periods, or the gradual enlargement of the areas is almost imperceptible. Occasionally enlargement of the patches is comparatively rapid. The condition is more apparent in the summer months on exposed surfaces, due to the increase of pigment in the unaffected areas. It is more conspicuous in dark-complexioned persons and very conspicuous in the colored race. While it is more or less limited in its development as a rule, generalized cases have been recorded by Hall,[1] Lévi,[2] Hardaway,[3] Simon[4] and Stelwagon.[5]

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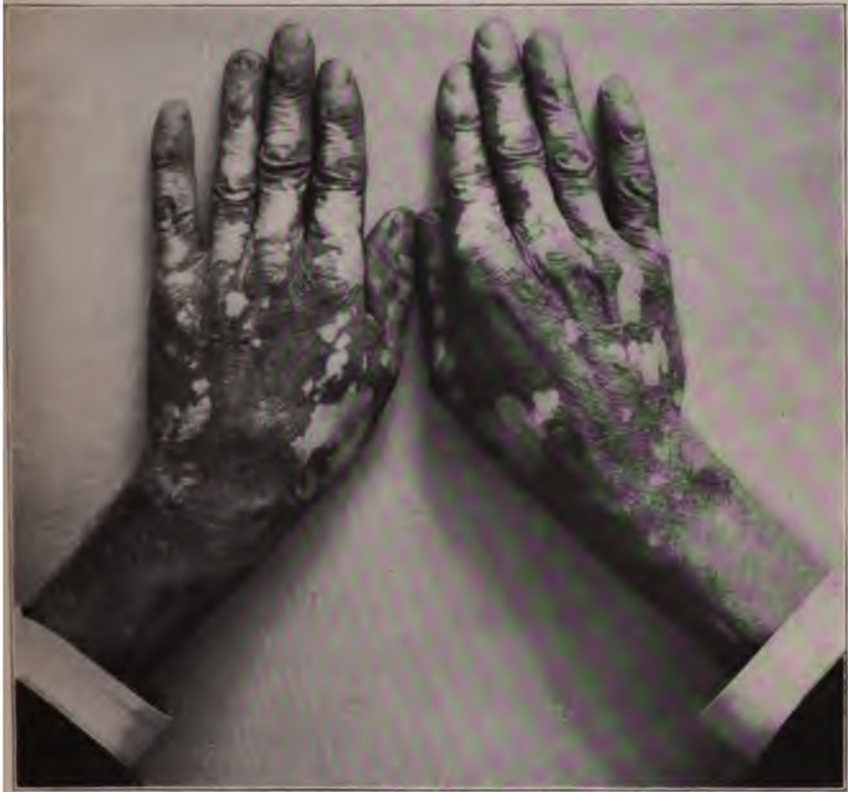
[1] Amer. Jour. Med. Sc., 1839, xxiv, p. 517.

[2] Trans. London Ophthal. Soc., 1891, xi, p. 218.

Occasionally through enlargement of many patches, either in limited areas such as the face and hands, or even on the trunk, the pigment is so far removed that only streaks or bands of normally pigmented skin are left which present a puzzling picture. These are at times mistaken for chloasma,[6] the affected areas here being considered the normal.

FIG. 179



Vitiligo. (G. H. Fox's Atlas of Skin Diseases.)

Occasionally a patch of vitiligo presents in its center a red papule or brown maculo-papule. Several such areas may be exhibited in the same patient.[7]

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[1] Louisville Med. News, 1888, x, p. 148.

[2] Receuil de Mém. de Méd., de Chir. et de Pharm. mil., 1865, p. 193.

[3] Manual of Skin Diseases, 2d Ed., p. 280.

[4] Deutsch-klinik, 1881, p. 399.

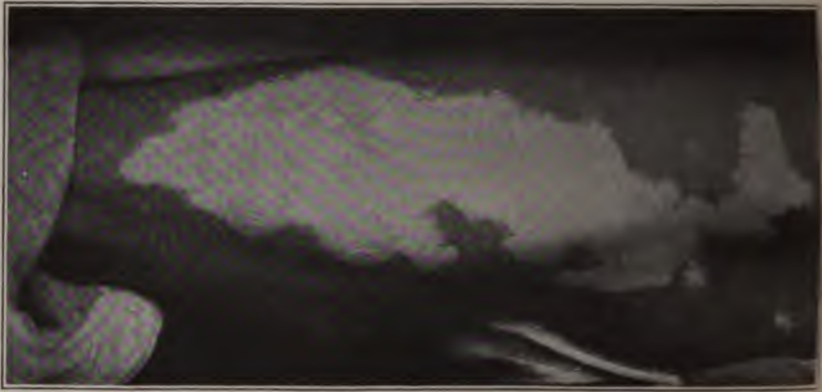
[5] Amer. Jour. Med. Sci., 1885, xc, p. 168, and Trans. College of Physicians, Philadelphia, 1894.

[6] Jackson, Jour. Cut. Dis., 1911, xxix, p. 555.

[7] Hyde, Jour. Cut. Dis., 1906, xxiv, p. 84; Sutton, Ibid., 1916, xxxiv, p. 797; Shelmire, Ibid., 1917, xxv, p. 163; Bunch, Brit. Jour. Derm., 1918, xxx, p. 203.

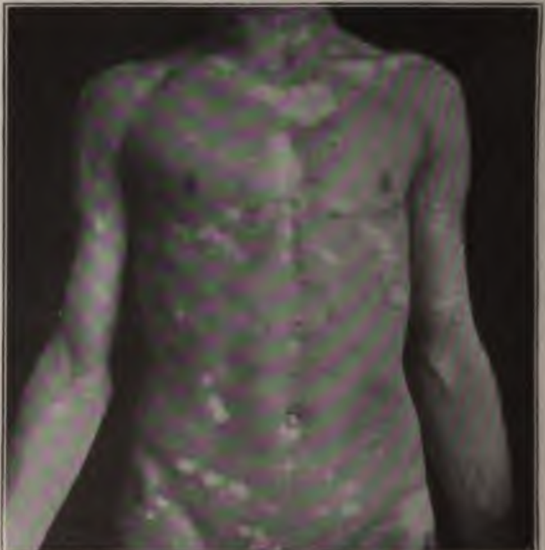
The skin in vitiligo presents no textural changes and is normal in every way except a sensitiveness to solar irritation. Severe burning occurs frequently during the summer months, owing to lack of protective pigment.

Fig. 180



Vitiligo.

Fig. 181

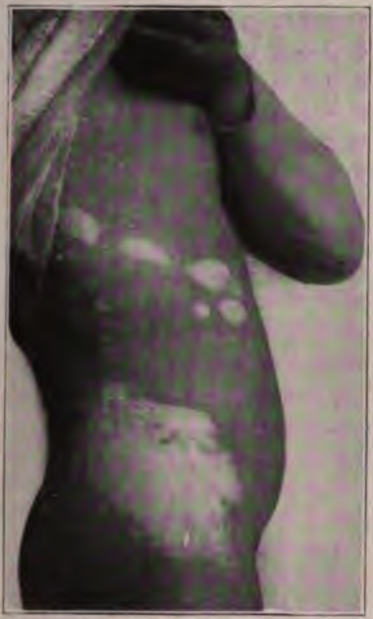


Vitiligo.

Etiology.—The cause of vitiligo is unknown. It occurs most commonly during the second and third decades of life. Crocker recorded an example in a child four years of age. It occurs in association with or follows certain cutaneous diseases such as lichen planus, psoriasis,[1] alopecia areata and local pruritus. It occurs in myxedema, Graves' disease,[2] and syphilis.[3] Gaucher[4] and others believe it to be of toxic origin. It frequently occurs in neurotic persons. It occasionally follows local irritation, trauma through scratching and irritant applications such as formalin.[5]

Pathology.—In the affected patches the epidermis is devoid of pigment. It is otherwise unchanged. In the corium some pigment cells are present. In the surrounding area the epidermis is hyperpigmented and the bloodvessels, follicles, and glands of the corium are surrounded by numerous oval, stellate and branched pigment cells.

FIG. 182



Vitiligo. (Douglass W. Montgomery.)

Diagnosis.—The white patches surrounded by a hyperpigmented border presenting no structural changes are characteristic. Chloasma presents hyperpigmented areas and can only be mistaken for vitiligo when the latter disorder is extensive and the hyperpigmented borders are mistaken to be the pathological portions. By remembering that the pathological portions, whether white or hyperpigmented, have convex margins, errors cannot occur. In tinea versicolor the presence of scaling, the location of the patches and the finding of *Microsporon furfur* microscopically in the scales readily establishes the diagnosis. From morphea, and the white patches occurring in anesthetic lepra, the structural changes in the skin are evident and make the differentiation. The white patches of partial albinismus are distinguished by their congenital history, lack of hyperpigmented border, and their lack of tendency to enlarge.

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[1] Hartzell, Diseases of the Skin, 2d Ed., p. 535.

[2] Dore, Brit. Jour. Derm., 1900, xii, p. 353; Hyde and McEwen, Amer. Jour. Med. Sc., 1903, cxxv, p. 1000; Schamburg, Jour. Amer. Med. Assoc., 1919, lxxiii, p. 30. In discussion of vitiligo and syphilis.

[3] Lane, Jour. Amer. Med. Assoc., 1919, lxxiii, p. 27. Discussion of vitiligo and syphilis with pertinent references. Ravitch and Steinberg, Amer. Jour. Syph., 1918, xi, p. 478. Vitiligo syphilitica. Brown, Dujardin and Van Haecke, Brit. Jour. Derm. and Syph., 1919, xxxi, p. 1. Vitiligo and its relation to syphilis, acquired or hereditary.

[4] Annales, 1902, s. iv, iii, p. 1113.

[5] Castellani and Chalmers' Manual of Tropical Med., 1913, 2d Ed., p. 1609.

Treatment.—The treatment of the disorder is almost uniformly unsuccessful so, therefore, no method can be recommended. Arsenic and iron are reported to have been occasionally successful. Montgomery[1] reported the return of pigment in vitiligo following the use of the Finsen light. With[2] and others have succeeded in producing pigmented spots and areas in patches of vitiligo with Finsen light. Stein[3] reported a similar result by using the Kromayer quartz lamp and by freezing with carbon dioxid snow. Ochs[4] applied tincture of nux vomica on areas of vitiligo as a placebo, with a return of the pigment in the areas treated.

Prognosis.—The disease progresses to a certain grade of development and then remains unchanged. The disfigurement causes some distress in sensitive persons.

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[1] Jour. Cut. Dis., 1904, xxii, p. 17.

[2] Brit. Jour. Derm. and Syph., 1920, xxxii, p. 145. Studies on the effect of light on vitiligo. Literature.

[3] Archiv, xcvii, p. 163, abstr. Brit. Jour. Derm., 1910, xxii, p. 102.

[4] Jour. Cut. Dis., 1911, xxix, p. 111.

CLASS VI.

NEW GROWTHS.

CICATRIX.

Synonyms.—Scar. Fr., *Cicatrice;* Ger., *Narbe.*

Definition.—A cicatrix is a connective-tissue new-formation of the skin, replacing normal structures which have been lost by traumatism, by ulceration, or by some other pathological process. Most cicatrices are reparative in character, as, for example, those following the ulcerations of syphilis, the operations of the surgeon, or the dermatitis produced by a severe burn.

Symptoms.—Scars vary greatly in shape, size, color, and other features. They may be smooth, glossy, shining, scaling, dull-whitish in color, or pinkish from vascularization of the surface. They may be linear, fan-shaped, circular, corded, ridged, dotted, crateriform, or tumor-like. They may be raised above the skin, be on a level with it, or be depressed below it. They may be deeply attached to periosteum or to bone, or be readily movable over the panniculus adiposus. They are of deeper color when young, and increase in whiteness with age, and are unprovided, as a rule, with hairs, or with coil- or sebaceous glands.

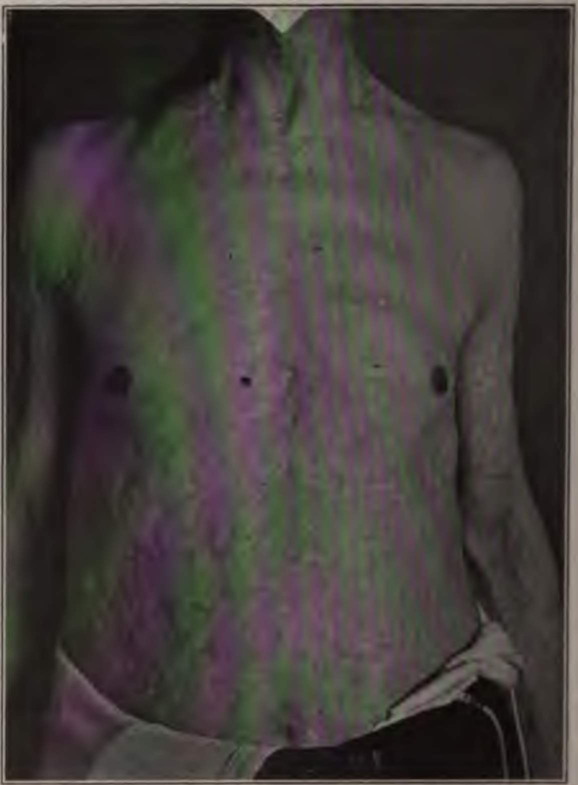
The most insignificant cicatrices are those resulting from clean, incised, and punctured wounds and lesions of similar grade. Certain peculiarities of cicatrices are seen in the special disorders in which they are produced. Circular, oval, reniform, horseshoe-shaped, S-shaped, and figure-of-eight-shaped scars, thin and flexible, are characteristic of syphilis. The cicatrices of variola, zona, and ecthyma are slightly different each from the other, though all are of small size and depressed. Those of tuberculosis and burns of severe grade are exceedingly irregular and often corded, and when occurring about the joints produce deformity through contraction.

Hypertrophy of cicatrices is the condition elsewhere described as keloid. Here there is a tumor-like development of the cicatrix, in the form of a ridge, button, knob, indurated fold, or puckered and irregularly circumscribed, whitish or reddish lesions. In certain individuals these lesions may follow almost every traumatism and destructive process to which the integument is liable.

Keloid-like cicatrix of the cheeks following acne is far from uncommon. Its lesion is usually smoothed down in the process of time, after the disappearance of the sebaceous-gland disorder, until the deformity is lessened greatly, and often scarcely noticeable. Colloid degeneration occurring in scar-tissue and producing lesions whi clinically resemble those of xanthoma is described by Juliusberg[1] an Dubendorfer.[2]

Etiology.—The formation of cicatrix is always preceded by destru tion of at least a portion of the papillary body of the corium. Th loss of tissue may be due to various causes: trauma, burns, ulce and atrophy caused by pressure of new-growths. Hypertrophi cicatrix may result from slight but continued or frequently repeat irritation of a healing surface, the repair of which is thus greatly d layed, but it occurs chiefly in the form of cicatricial keloid.

FIG. 183



Scars following hypodermic injections of morphin. (Fordyce.)

Pathology.—Histologically, scars are made up of connective-tiss bundles, which interlace in all directions with great irregularity. young scars the fibers are finer and the tissue is vascular, but as t scar grows older the fibers usually become coarser and contract a the vessels disappear. There is complete absence of the hair-follicles, glands, and furrows of normal skin. The scar-tissue proper is covered with a very thin epidermis, and Heitzmann claims that shallow and irregular papillæ are always present. Other observers report in scars an entire absence of both papillæ and rete-pegs.

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[1] Archiv, 1902, lxi, p. 175 (with bibliography of colloid degeneration, and of pse xanthoma).

[2] Ibid., 1903, lxiv, p. 175.

Diagnosis.—The distinction between hypertrophied cicatrix and keloid is one chiefly of degree, and needless from a practical point of view.

Treatment.—Treatment is of very little value in the depressed scars following variola and other similar affections. The prevention of scarring by proper treatment of the preceding disorder is highly important. The disfiguring scars following burns and some other destructive processes may be largely avoided by forestalling pus infection. The management of hypertrophic scars is outlined under treatment of keloid.

KELOID.

Synonyms.—Cheloid, Kelis, Alibert's keloid. Fr., *Chéloïde;* Ger., *Knollenkrebs.*

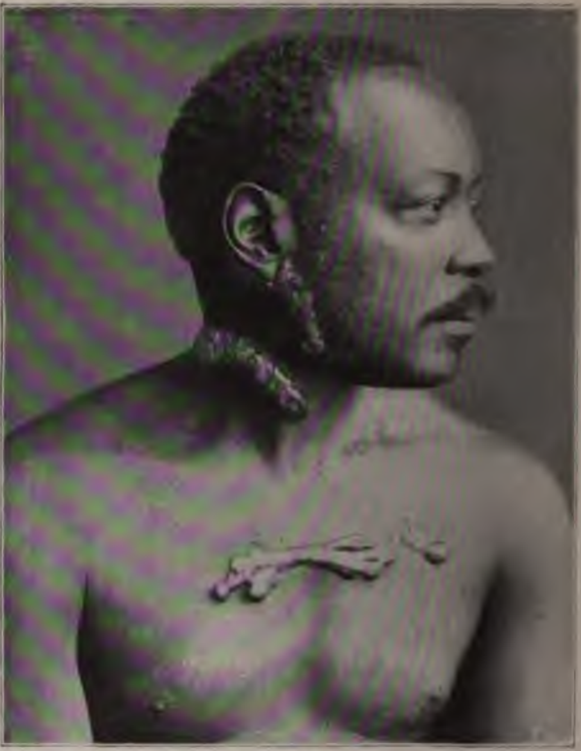
Definition.—Keloid is a neoplasm of the derma, usually following trauma, developing as one or multiple fibro-cellular elevations of the skin, irregularly shaped, smooth or corrugated, whitish or reddish in hue, and resembling a thickened and hypertrophied cicatrix.

The term keloid, first given to the disease by Alibert, should be restricted to the above defined disorder. The keloid of Addison is now known to be scleroderma.

Authors have described two varieties of the disease: the "true," "spontaneous," or idiopathic form; and the "false," "spurious," or cicatricial form, which develops in the scar produced by a traumatism. There is no anatomico-pathological separation between the two. Cases of so-called spontaneous keloid are usually instances of development of the growth following such slight wounds as those inflicted by mosquitoes, by scratching, contusion, traction, or pressure.

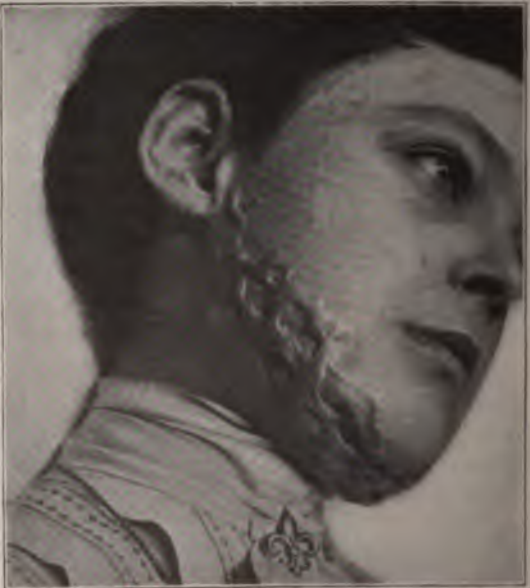
Symptoms.—The new-formations of this disease are dense, generally elastic nodules imbedded in the corium, or projecting above the level of the skin and firmly attached to it. They are usually slow of evolution, and having once attained full development and assumed one of the several shapes which they affect often persist for a lifetime. These forms are whitish or reddish, globular or semiglobular nodules, buttons, or plaques, with roundish or ovoid outline; linear, elevated striæ, bands, ridges resembling cords, ribbons, or tapes, in irregular outline and disposition; or combinations of two or more of these figures. A common form over the sternum or other situations where the development of the growth in one direction is not impeded is that of a larger central mass with two or more diminishing and declining prolongations, bearing a remote resemblance to the body and claws of a crab. The lesions vary in size from that of a small pea to that of a large plate, the largest including the outlying points of the limbs or

FIG. 184



Keloid.

FIG. 185



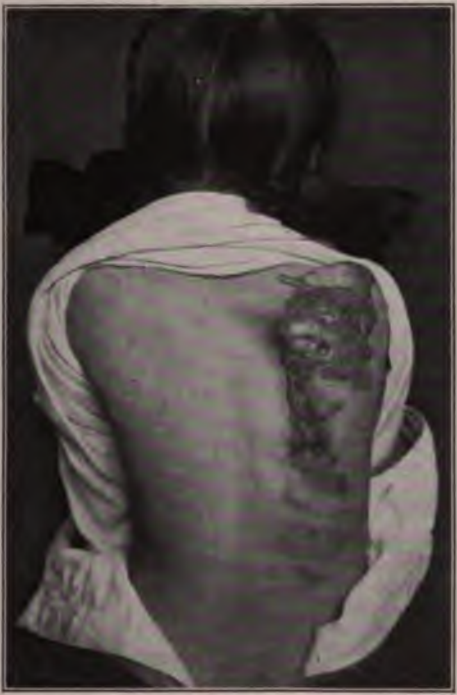
Hypertrophic scars (keloid) following burn.

radiating ridges. The lesions may be white, grayish-white, light- to deep- or even vivid-red in color. In the colored race the lesions are either blackish or entirely devoid of pigment. Their surface is smooth, hairless, and frequently shows telangiectatic bloodvessels.

The subjective sensations aroused are commonly trifling or inappreciable. At times the growths are the seat of severe pain or burning, and occasionally are hypersensitive to pressure and heat.

The most frequent site of the disease is the anterior surface of the chest, but it is observed also upon the face, neck, ears, breast, hands, between the scapulæ, and on the extremities. Though frequently multiple, there are rarely more than a score of these growths visible at one time upon the skin of an individual. The disease is far more common in the colored than in the white races.

FIG. 186



Keloid following burn.

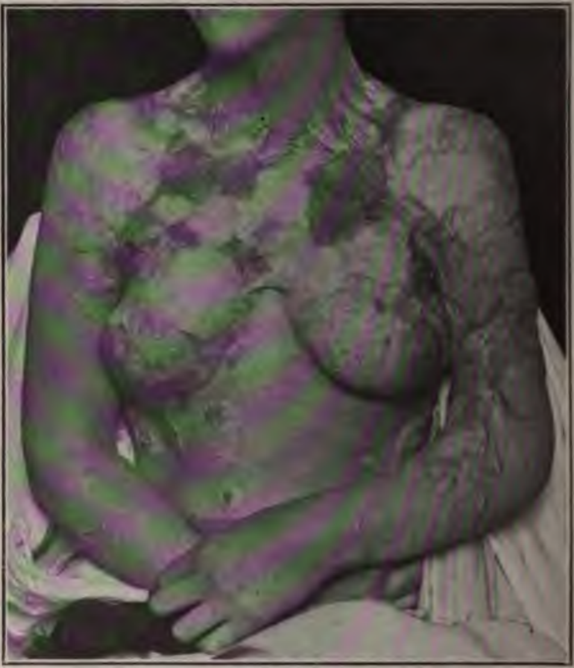
The usual course of the disease is toward the production of tumors of a medium size, after which few changes are to be recognized. Involution and complete disappearance are rare. These results, however, have been secured in a few cases.

Cicatricial Keloid (*Scar-keloid; Hypertrophic scar; Hypertrophic cicatrix*).—These terms are employed to indicate that the lesion has been preceded by scar-formation due either to disease or injury.

It thus follows the lesions of zoster, syphilis, and variola, as also traumatisms of all sorts, including those made by surgical operations and accidents. The presence of a large number of small, scattered keloids suggests to the mind of the experienced clinician the frequent use of the hypodermic syringe, as keloids may form at the point of puncture of the hypodermic needle.[1]

It is not every scar, even in susceptible individuals, which becomes hypertrophic. The tumors, as a rule, spring directly from scar-tissue, and after reaching a maximum of development do not surpass the limits of the original lesions. At times, however, the growths slowly develop at a distance from the original site of the injury or disease. Those growths remaining limited to the site of the original injury are more properly considered as hypertrophic scars.

FIG. 187



Extensive keloids following burns.

Scar-keloid often is found as a firm nodule in the lobe of each ear among women who have pierced the ears for the insertion of earrings. Lesions in this situation often assume huge proportions. It is seen also not rarely as a result of burns, whether produced by the application of caustic agents or heat. Lesions of this kind rarely develop symmetrically. They may be counted at times by the hundred;

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[1] Herzog, Jour. Amer. Med. Assoc., June 1, 1907, p. 1844.

commonly but one or two are seen in one person. They may persist after reaching a maximum development, or spontaneously disappear, wholly or in part, or ulcerate, or become the seat of malignant growths.

Acne Keloid.—Acne keloid is a term descriptive of a disease occurring usually on the nucha and scalp, formerly supposed to be a keloid (*Cf.* Dermatitis papillaris capillitii).

Etiology.—The origin of the disease is obscure. Neither age, sex, nor previous disease of the skin seems to have any bearing upon its production. It is seen in vigorous persons (more often in the negro race), but also in those who are weakly. Schramek[1] records multiple keloids in association with Mongolian spots, thus suggesting their congenital origin. Heredity and family influences[2] play a part in certain cases. The exciting cause of many keloidal growths is most frequently found in trauma, burns, and suppurative processes induced by many agencies, a common one being that occurring in connection with tuberculous adenitis of the neck. In many of the so-called idiopathic keloids, no preceding pathological condition is demonstrable. Krzysztalowicz[3] considers keloid to be a chronic inflammatory process, and that most spontaneous keloids are due to a chronic inflammation of the hair-follicles and sebaceous glands. He believes that the two forms are not separable.

Pathology.—The pathogenesis of the disorder is not understood. The histology shows a dense connective-tissue new-growth limited to the corium. In newer formations a larger number of connective-tissue cells are present; while later the mass becomes more dense and cells less frequent. Extending from the growths are masses of cells, usually surrounding bloodvessels, and it is due to the presence of these masses of cells that recurrence happens after supposed extirpation of the mass. According to Warren,[4] the keloid begins by a growth of round cells in the adventitia of the arterioles of the corium. These cells become fusiform, and finally develop into the connective-tissue fibers forming the tumor. According to Langerhans,[5] Warren,[6] Kaposi, and others, in all cases of keloid the papillary layer of the corium and the interpapillary projections of the rete downward are intact, the new-formations being strictly limited to the middle and lower portions of the corium, in which there are numerous dense collagenous bundles, disposed for the most part parallel with the surface of the skin. In cicatricial keloid, these observers found a partial or complete absence of the papillary and interpapillary processes. Babes, Crocker, and others, on the contrary, found that the papillæ and rete may be normal, modified, or absent in either form. Heidingsfeld[7] found none of the peculiarities above described holding true in all cases, and believes there is no histological distinction between the different varieties. The elastic tissue is described as being attenuated, absent, or in the process of new formation. Heimann[1] found evidences of regeneration of elastin. The sebaceous glands, coil-glands, hair-follicles, and muscles are pushed to one side by the new-growth and not infrequently become atrophied.

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[1] Archiv, 1910, xcix, p. 207; abstr. Jour. Cut. Dis., 1910, xxviii, p. 424.

[2] Jour. Cut. Dis., 1905, xxiii, p. 365.

[3] Monatshefte, 1909, xlix, p. 381; abstr. Brit. Jour. Derm., 1910, xxii, p. 145.

[4] Annales, 1888, ix, p. 573.

[5] Virchow's Archiv, Dritte Folge, Bd. xl, p. 334

[6] Loc. cit.

[7] Jour. Amer. Med. Assoc., October 16, 1909, p. 1276: Keloid: A comparative histological study. (In this study seven cases of spontaneous keloid, three of false keloid, one of soft multiple fibroma, two cases of hypertrophic scars, two of atrophic scars, two of scleroderma, and two of macular and striated atrophia cutis were examined.)

Diagnosis.—The situations of the lesions of keloid (often over the sternum), the peculiar claw-like elongations, and yellowish-white, reddish, or grayish-white color, all point to the nature of the disease.

Treatment.—The most satisfactory treatment for keloid and hypertrophic scars is found in radiotherapy. Ullmann, Taylor, Pusey,[2] and others report instances in which a keloid or thick scar has been removed wholly or in part by the use of *x*-rays. In a large number of cases of all varieties we have found much improvement, and in most instances obtained complete removal of the elevated, indurated lesion and its replacement by soft, smooth, though sometimes telangiectatic scar-tissue. In several instances in which hypertrophic scars due to burns from steam or fire were extensive and very disfiguring, the entire thickness of the growth was removed, leaving soft, pliable scars. Radium is also valuable for keloids of moderate dimensions. Pfahler[3] recommends radiotherapy combined with excision. Removal of keloid by cauterization and excision is not to be practised, as the growth commonly recurs. Vidal successfully employed multiple linear scarification. Lawrence[4] obtained good results by scarification, followed for several weeks by moderate pressure produced with rubber tubing held in place by adhesive plaster. Various stimulating applications may also be made with a view to promote resorption, such as the tinctura saponis mollis, iodized glycerin, iodin in ointment and tincture, and mercurial, salicylated, and lead plasters. These applications are to be made with caution. Where there is pain, anodyne ointments may be employed topically, such as freshly prepared belladonna plaster, or ointments of belladonna, stramonium, and opium. One that has been especially valuable and which is also capable of producing an alterative effect is an ointment of the oleate of mercury and morphin. Ularic and others report successful destruction of keloid with injections of 5 to 20 per cent. solutions of creosote in olive oil. Electrolysis has given good results in a few cases. Tousey, Newton, Crocker, Neisser, and others report excellent results from injections along the growths of from 10 to 20 minims (0.66–1.33) of a 10 per cent. solution of thiosinamin in equal parts of glycerin and water, or in alcohol. Mears[5] employed thiosinamin successfully in one case, in dosage of $\frac{1}{2}$ to 1 grain internally, in connection with a 5 to 8 per cent.

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[1] Archiv, 1910, cii, p. 65; abstr. Jour. Cut. Dis., 1911, xxix, p. 119.

[2] The Röntgen Rays in Therapeutics, p. 558.

[3] Archiv of Derm. and Syph., 1920, ii, p. 181. Roentgen-rays or radium combined with excision in the treatment of keloids.

[4] Brit. Med. Jour. 1898, ii, p. 151.

[5] Med. Record, 1910, lxxviii, No. 2, p. 902.

ointment of the same drug applied locally. Internally, quinin, strychnia, arsenic, and potassium iodid have been given with varying success.

Prognosis.—As regards the general condition of the patient, the prognosis is favorable. Very rarely there is spontaneous resorption of the nodule or tumor. Generally the latter may be expected to persist, after full evolution is attained, for an indefinite period of time.

FIBROMA.[1]

Synonyms.—Fibroma molluscum, Molluscum fibrosum, Molluscum simplex pendulum, Neurofibroma, von Recklinghausen's disease.

Definition.—A fibroma is a connective-tissue new-growth of the skin, which may occur as a hard or soft tumor, varying in size, shape, and consistency. The lesions may be single or multiple, congenital or acquired, and, in the cases with multiple lesions, are frequently accompanied by pigmentation.

Symptoms.—Multiple fibromata usually begin to develop early in life, or at about the age of puberty, and gradually progress, new lesions appearing from time to time, until in some cases large numbers are present.[2] They are irregularly disseminated over the cutaneous surface, being seen in highest development over the front and back of the trunk; in fewer numbers over the head, face, and limbs. The lesions are usually pea- to egg-sized tumors, and vary greatly according to their age and dimensions. In early development they present dome-shaped, pea- to bean-sized elevations, which are softish, semisolid or pasty, and with a normal epidermal covering. Later they are cone-shaped, frequently nipple-like, semisolid formations, sometimes capped by an opaque summit, which presents the appearance of a vesicle, and which bleeds freely when pricked. Wickham[3] emphasizes the occurrence of small, dome-shaped, elevated, soft, violaceous prominences and patches scattered throughout these lesions. Digital compression causes some some of these tumors to disappear through a ring in the skin, and when released they spring back into their normal position. Old tumors are less gelatinous, are firmer, harder, and not infrequently are mammilated and pedunculated. Small-sized tumors may recede, leaving an empty patch of skin. When diffuse, especially about the face, they draw the skin into extensive folds.

Under the title fibroma molluscum gravidarum Brickner[4] described an unusual form of the disease developing in women during pregnancy and subsequently disappearing. Hirst,[1] Sutton[2] and others have reported similar cases.

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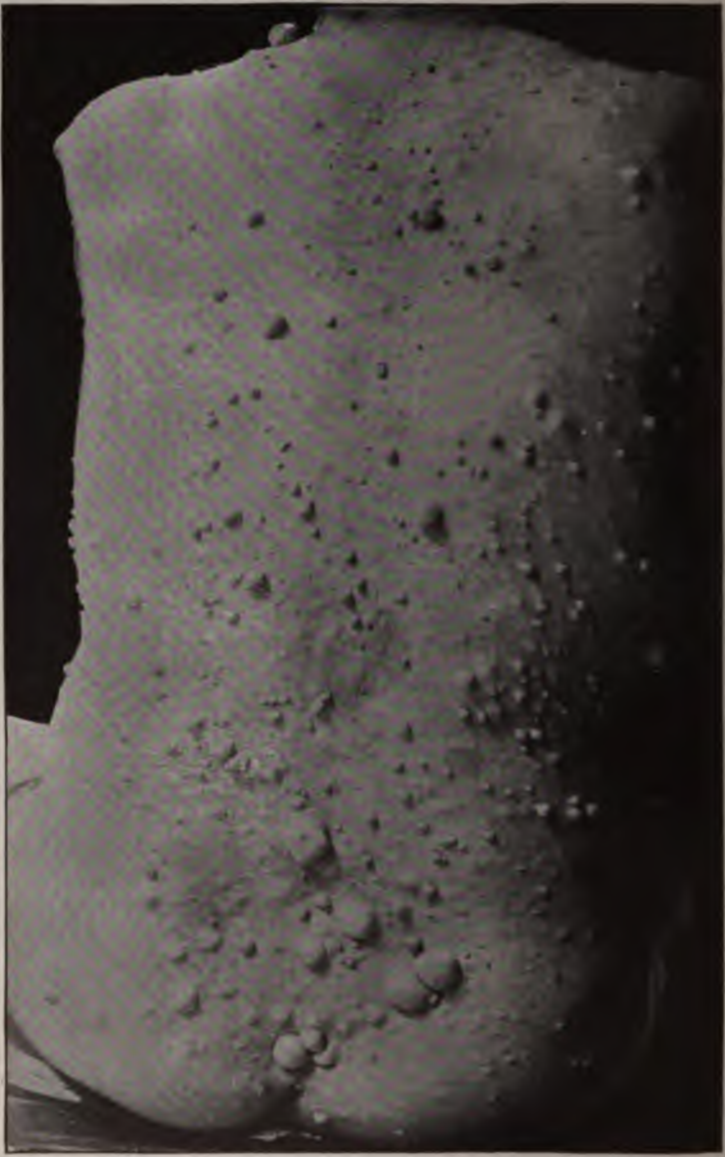
[1] Friedlander, Jour. Cut. Dis., 1910, xxviii, p. 497 (report of a case of multiple neurofibromata, with review of the subject, based on 262 cases reported in the literature). Ravogli, ibid., 1911, xxix, p. 71: Fibroma molluscum or universal neurofibromatosis (bibliography).

[2] Razumovski, Russki Joor. Koznikh E Venericheskikh Boleznei, February, 1913, xxv, No. 2, p. 103; abstr. Jour. Cut. Dis., 1913, xxxi, p. 1060 (a case with 5868 tumors scattered irregularly over the body). Anderson, Jour. Amer. Med. Assoc., 1920, lxxiv, p. 1018. Generalized neurofibromatosis. Case report more than 3000 variously sized tumors present.

[3] Brit. Jour. Derm., 1890, ii, p. 151.

[4] Amer. Jour. Obst., 1906, liii, p. 191. Fibroma molluscum gravidarum.

Fig. 188



Multiple fibromata of the back

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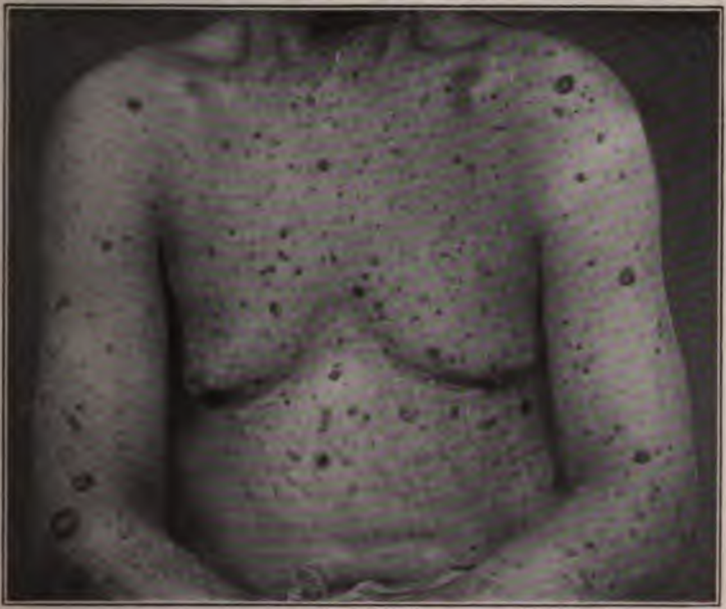
[1] Amer. Jour. Obst., 1911, lxiii, p. 256. A note on the etiological influence of pregnancy upon molluscum fibrosum.

[2] Amer. Jour. Med. Sc., cxlvii, p. 419. A clinical note on fibroma molluscum gravidarum.

Fibroma pendulum occurs most frequently about the face and sides of the neck, the occipital region, the arms, axillæ, breast, buttocks, thighs, and labia. Huge masses weighing several pounds are frequently developed, and the skin hangs in lax folds and not infrequently overlaps. The masses present to the palpating finger the sense merely of skin and fat. The skin, in addition to presenting a lax appearance, is frequently pigmented and covered with plugged sebaceous orifices. A remarkable example of this disorder is recorded by Crocker, the patient having been previously reported by Treves, and presented publicly as the "elephant man." In this case the skin was greatly thickened, hanging in folds, having irregular, lobulated masses of confluent tumors, and presenting the usual type of molluscum fibrosum. It is to these cases that the term *dermatolysis* has been applied. Somers[1] recorded a case in which a pendulous tumor, originating on the left side of the vulva and left buttock, hung nearly to the knee. The surface in these cases may show dilated bloodvessels and comedones, or present nevoid appearances. When occurring about the anal region, they present folds of tissue, which annoy the patient in defecation. Occasionally, a single large, pedunculated tumor is seen. In a given case tumors of all sizes and in all stages of development may be present.

FIG. 189



Multiple fibromata.

The tumors may affect the mucous membrane of the mouth, the large nerve-trunks, suprarenal capsules, intestines, and other internal organs. Cooke[1] removed a large number of tumors from the rectum, which were identical in histological structure with the molluscum tumors of the skin found in the same patient. Not infrequently, various bones may also become involved in the process. Patches of leucoderma, telangiectasia, patulous orifices of sebaceous glands, comedones, and fatty tumors are not uncommon.

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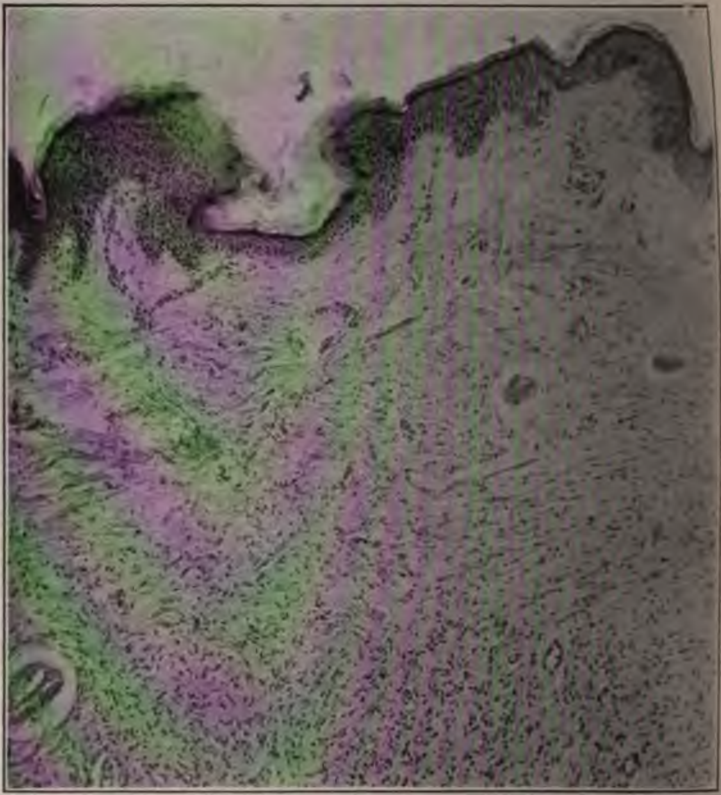
[1] Occidental Med. Times, January, 1904, p. 19.

FIG. 190



Photomicrograph of a section of von Recklinghausen's disease. (Stillians' section.)

Pigmentations are usually light- to dark-brown colored freckles sprinkled over the entire cutaneous surface, together with a few large patches, in some of which jet-black points are at times observed. The freckles may be discernible in the negro. Wickham[2] observed pigmentation in eight cases, and especially emphasizes its importance in this disorder. Oddo[3] has reported two cases in which there were pigmentations of the mucous membrane of the mouth, resembling those com-

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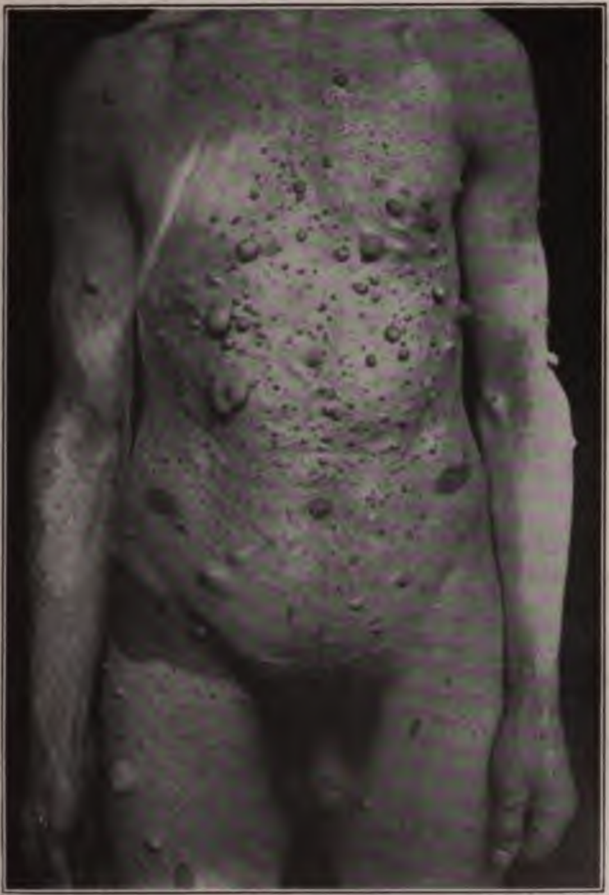
[1] American Medicine, November 21, 1903, p. 818.

[2] Loc. cit.

[3] Annales, 1906, s. iv, vii, p. 803.

y observed in the mouths of dogs. Weber[1] believes, in common
a few others, that there are anomalous cases of von Reckling-
n's disease in which the pigmentary are the only changes, at
for certain periods of time. In the case as originally reported
eber, only pigment was present; in the later report tumor-forma-
ad begun. Little[2] recorded a typical case in which the pigmenta-
receded the tumor-formations by six years.

FIG. 191



Neurofibroma (von Recklinghausen's disease).

bra called attention to the low standard of physical and mental
opment in the subjects of this disease seen by him, a fact observed
any and well illustrated in several patients under the observa-
of the author.

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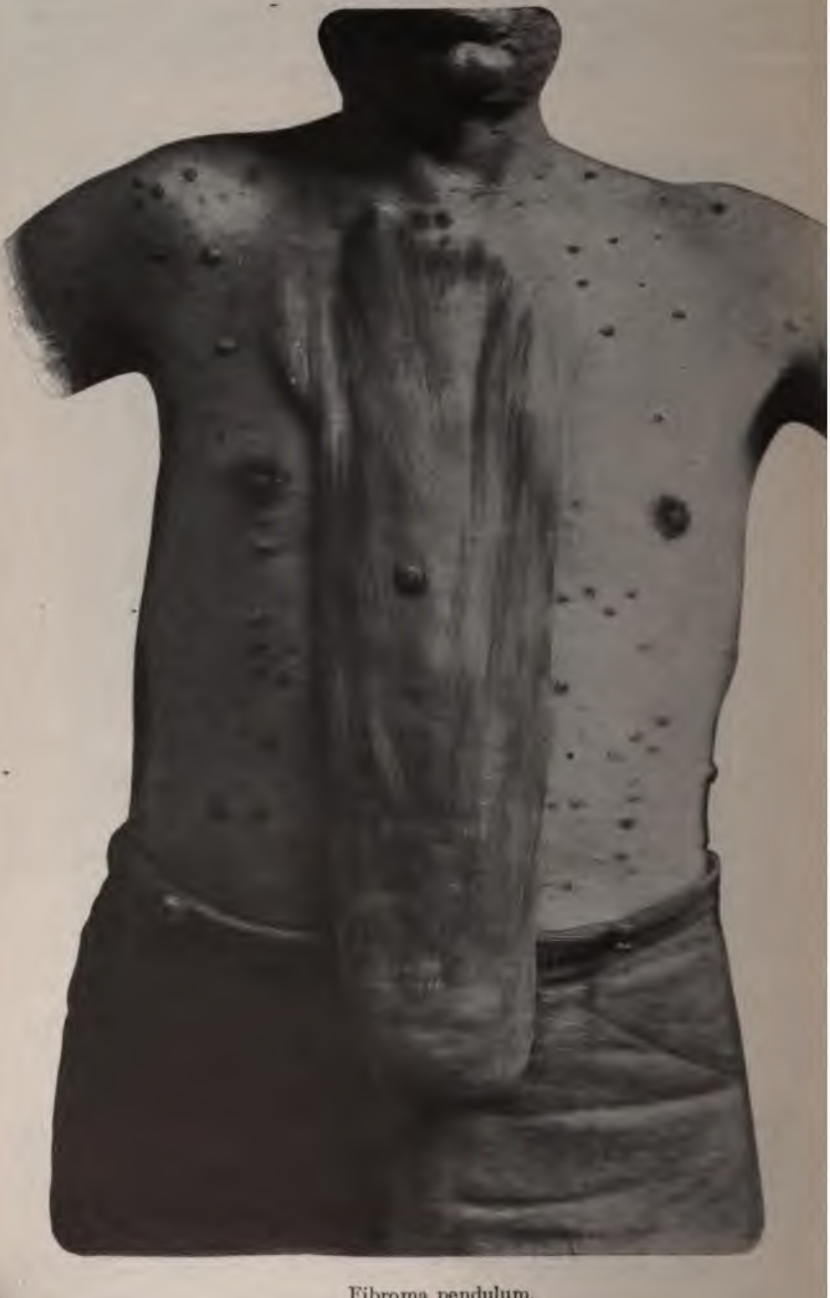
[1] Brit. Jour. Derm., 1905, xvii, p. 226; ibid., 1909, xxi, p. 49.

[2] Ibid., 1908, xx, p. 413.

The term *Achrochordon* (soft warts, *verrues charnues*) has been applied to that form of fibroma which occurs over the neck, face, and

FIG. 192



Fibroma pendulum.

between the shoulders, chiefly in elderly people, and characterized by pinhead- to pea-sized, soft, pedunculated, mole-like lesions, which, after undergoing involution, leave empty hernia-like sacs and small, wrinkled patches of the integument.

Hard fibromata (*desmoids*) usually occur singly, though several lesions may be present. They are noted on the trunk and extremities, and are usually round or oval in contour, firm, smooth, pea-sized nodules, deeply situated. They may occur at any age, even in childhood, and may be present at birth. They gradually develop and show no tendency to undergo involution.[1]

Etiology.—Multiple fibromata are not peculiar to either sex. The disease may be present at birth, or it may appear at puberty or even in early adult life. The cases with multiple lesions usually begin early, while those with single lesions are adult forms. The disorder may develop following surgical operations, during pregnancy, or after severe illness (malaria, dysentery), or it may develop in the absence of these factors. It has been described in association with certain developmental defects. Hintz[2] records von Recklinghausen's disease associated with adenoma sebaceum, and concludes that both disorders are due to faulty development. The association of adenoma sebaceum with the disease has also been recorded by Anderson[3] and Jamieson.[4] That heredity plays a part seems probable from the observation of cases in which the above defects of development and others are present; and also of families in which several members are afflicted, as well as those in which the disease has occurred in three successive generations. Trimble[5] records a case of mother and daughter with the disorder. Friedlander[6] says it is extremely probable that the disease is one of the nervous system, dating from intra-uterine life.

Pathology.—The tumor is a connective-tissue new-growth of peculiar character. It is described as myxomatous and as having a peculiar glassy appearance under the microscope. The fibers are usually finer and differ materially from the ordinary collagenous tissue of the corium. According to von Recklinghausen,[7] the tumor originates in the endoneurium of the nerve-trunks in the subcutaneous tissue and spreads upward along the nerves, the vessels, the muscles, and the follicles, dividing into fine bundles and extending toward the epidermis. The growth is well defined and composed of a gelatinous connective tissue, with fine fibers in the small tumors, while in the larger tumors, on the outer portion, the fibers are coarser; but in the central portion of the tumors a gelatinous consistency is maintained, with an abundance of cells. These are spindle-shaped and round connective-tissue cells, presenting well-marked nuclei. According to Unna,[1] the most striking constituent is a large mast-cell surrounded by a red halo, the latter being of the nature of spongioplasm and taking the same stain as the mast-cell granules. The blood- and lymph-vessels show dilatation. The glands of the skin are unaffected except as they are displaced by the new-growth. The epidermis shows no changes other than those due to compression from the tumor beneath.

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[1] Heidingsfeld, Jour. Cut. Dis., 1912, xxx, p. 332: Fibroma subcutis (report of a case with a hard, subcutaneous nodule on the finger, which histologically was a pure fibroma). Savatard, Brit. Jour. Derm. and Syph., 1920, xxxii, p. 126. Fibroma simplex. Case report. Single lesion. Typical microscopic appearance.

[2] Archiv, 1911, cvi, p. 277; abstr. Jour. Cut. Dis., 1912, xxx, p. 114.

[3] Brit. Jour. Derm., 1895, vii, p. 316.

[4] Ibid., 1906, xviii, p. 379.

[5] Jour. Cut. Dis., 1909, xxvii, p. 411.

[6] Loc. cit.

[7] Ueber die multiplen Fibrome der Haut, und ihre Beziehung zu den multiplen Neuromen. Berlin, 1882.

Diagnosis.—The tumors of molluscum fibrosum are to be distinguished from multiple cutaneous sarcomata by the violaceous or reddish color of the latter, with absence of pedunculation and greater tendency to ulceration, and by their evidently malignant character. From nodules of lepra they are distinguished by the entire absence of constitutional impairment and by their general development in far greater multiplicity. Tumors of molluscum epitheliale differ in their contents, their superficial location, and in the frequent presence of the dark puncta at their summits.

Neuroma is usually painful; lipoma less frequently multiple and pedunculated, commonly of larger size, having a lobulated structure, and presenting a "pillowy" sensation to the touch. Warty growths are readily distinguished by their verrucous summits; and the gummata of syphilis by the concomitant or prior symptoms of the existence of lues.

Treatment.—The treatment of large, single fibromata is surgical. Multiple lesions are often so numerous as to forbid such interference. In the major portion of cases treatment is of little avail. Fenton[2] reported improvement with fibrolysin; and Parounagen[3] reported improvement by the use of arsenic.

Prognosis.—Rarely one or more of the lesions disappears by spontaneous involution; more commonly they persist after their evolution is completed. Marasmus, tuberculosis, and a fatal result may occur. One or several of the tumors may become sources of danger from the occurrence in them of an active inflammation, with resulting degeneration and septicemic consequences. The disease, however, does not in many cases shorten life. In general, the prognosis of multiple fibromata may be regarded as unfavorable.

PARAFFINOMA.[4]

Since injections of paraffin have been made by many so-called "dermatological institutes" to remove wrinkles, form dimples, and otherwise change the normal contour of the face to suit the fancy of the patient, many untoward results are annually presented to the profession for correction. The proportion of patients who become victims of the peculiar new growths that sometimes follow such injections cannot be stated, but it appears to be small compared with the large number treated. Surgeons have for several years employed paraffin to replace lost tissue. Since Gesuny first injected a quantity of paraffin having a melting-point of 40° C., into the scrotum to replace lost testicles, for the purpose of hiding the deformity in a candidate who contemplated taking the physical examination for entrance into the army service, surgeons have utilized the method to overcome various deformities. It has been used in the correction of sphincter incontinence, in hernia, to separate nerve-ends, to correct cleft palate, and many facial deformities, especially those about the nose. It is to the cases in which unsightly new growths occur about the face following its use that this chapter is devoted. The following is a description of the clinical and pathological findings of several patients we have had under observation and treatment.

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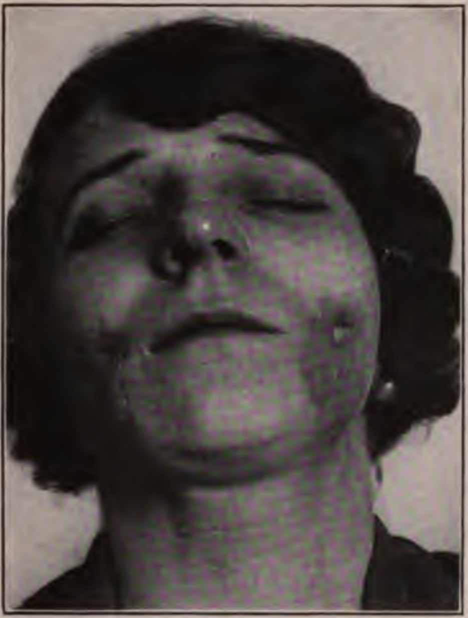
[1] Histopathology, p. 847.

[2] Brit. Jour. Derm., 1908, xx, p. 114.

[3] Jour. Cut. Dis., 1913, xxxi, p. 598.

[4] Literature: Heidingsfeld, Histopathology of Paraffin Prosthesis, Jour. Cut. Dis., 1906, xxiv, p. 513 (with many references); idem: A further contribution to the histology of paraffin prosthesis, Jour. Amer. Med. Assoc., 1908, Sec. 12, p. 2028; Ormsby, Tumor formations following paraffin injections, Jour. Cut. Dis., 1907, xxv, p. 277, Williams, Paraffinomata, Brit. Jour. Derm., 1907, xix, p. 432; White, Jour. Cut. Dis., 1909, xxvii, p. 313, case report; Goldenberg, Ibid., 1916, xxxiv, p. 401; Gaskell, ibid., 1918, xxxvi, p. 59, Ormsby, ibid., 1918, xxxvi, p. 469.

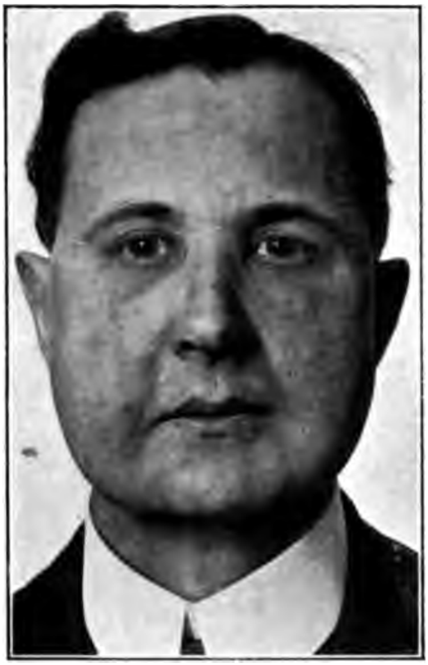
Fig. 193



Paraffinoma.

Symptoms.—There is commonly a period of time, varying from six to fifteen months, in which the tissues apparently do not rebel at the presence of the foreign material; then the new growths begins. The sites of these deformities are usually at the angles of the mouth, about the chin, near the junction of the wings of the nose with the face, between and beneath the eyes, and on the upper part of the neck. The masses vary in size from that of a pea to a hen's egg and larger. They are firm in consistence, attached to the overlying epidermis, and imbedded deeply in the subcutaneous tissues. In color they are bluish-red, at times brownish-red, often covered with dilated bloodvessels, occasionally they appear not unlike keloid. They are, as a rule, unaccompanied by subjective sensations, but occasionally some discomfort is experienced, due to the pressure over the bony prominences. When occurring near the angles of the mouth, the prominence extends into the oral cavity, producing bulging of the buccal mucous membrane.

FIG. 194



Vaselinomata of cheeks and chin.

Edema of varying degree is present in the surrounding tissues. The deformity produced in some of these patients is disfiguring to a marked extent and practically forbids their appearance among their fellows.

Tumors following injections of vaseline[1] occur in the same situations and are accompanied by practically the same symptoms as those described above. In one instance noted by the author the tumors began developing three years after the injection.

Camphorated oil tumors are reported by Mook and Wander[2] which very closely resemble the tumors produced by paraffin. These tumors, however, occur in a different situation, usually being found on the arms, thighs and breast.

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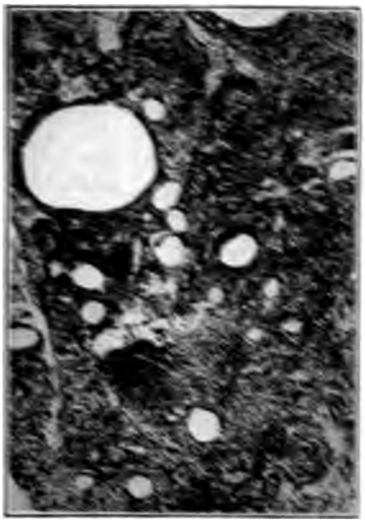
[1] Ormsby, Jour. Cut. Dis., 1917, xxxv, p. 612; "Tumors following injections of vaseline;" ibid., p. 843.

[2] Jour. Amer. Med. Assoc., 1919, lxxiii, p. 1340; "Camphorated oil tumors; clinical and histological report of six cases."

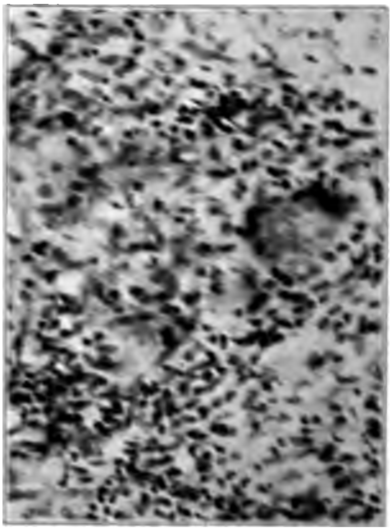
Histopathology.—The tumor is essentially a connective-tissue new growth, resembling very closely the granulomata. With the low power of the microscope it presents a series of oval and round cavities, not unlike well aërated Swiss cheese, as described by Heidingsfeld. These cavities represent spaces formerly occupied by the paraffin, which penetrated the tissues in numerous fine channels. The larger spaces were probably produced by the paraffin having been deposited in masses by rupture of the tissues. In addition to the fibrous bands and multinucleated cells, groups of connective-tissue cells are found. It appears probable that, owing to the highly vascular tissue in these areas, the foreign material is not encapsulated to such a degree as when deposited in the purely fatty tissue, such as occurs when injected in some of the regions referred to above, where surgeons employ it for cosmetic or other purposes. The histological structure of the vaseline tumors above noted was practically identical with that seen in the paraffin cases, and that described in connection with the camphorated oil tumors is also similar.

Fig. 195



Section from tumor induced by paraffin. Low power, showing Swiss-cheese appearance and cellular infiltration.

Fig. 196



Section same as Fig. 195. High power, showing giant and other cells.

Diagnosis.—In paraffin tumors about the face the lesions resemble keloid and sometimes sarcoid and occasionally even lupus vulgaris. The hardness of the growth and the absence of the soft, apple-jelly-like nodules of lupus distinguish it from this disease. The peculiar conformation, distribution, color and size of the mass, together with the history of previous treatment with paraffin serve as distinguishing features of these cases. The camphorated oil tumors have to be differentiated from syphilis, tuberculosis, or other possible new growths. Their location and the history of a previous illness when injections were necessary are important points in the diagnosis.

Treatment.—Surgical excision followed by radiotherapy has given the best results in the paraffin cases. Gaskell reported improvement with radiotherapy and high frequency, and Schamberg with the use of the galvanic current and incandescent bulb, the heat in the latter being considered efficient. In one of the author's patients much improvement followed the use of radium. It has been our experience that unless the paraffin has been totally removed there is soon a recurrence. It is, therefore, necessary to have the surgical removal very thoroughly accomplished. Other methods of treatment have been employed without much success.

Prognosis.—After observing these cases for many years no further changes have been noted. Heidingsfeld stated that the natural goal is complete fibrosis. Malignant changes have not occurred except in a single instance mentioned by Wise.[1] The growths remain unless removed surgically. The deformity is pronounced in some cases, causing much suffering and in one case self-destruction.

CHONDRODERMATITIS NODULARIS CHRONICA HELICIS.[2]

Synonym.—Painful, nodular growth of the ear (Foerster).

Cases recorded under the above titles were independently observed by Winkler of Luzerne and Foerster in this country. Winkler's description was founded on the observation of eight cases during a period of ten years in individuals ranging from twenty-eight to sixty-five years of age. The lesion described was a cherry-stone sized flat papule, with a central crust situated on the upper pole of the auricle. The margin was slightly elevated and somewhat hard. The color was that of the surrounding skin, or translucent, resembling an epithelioma. On the surface there occurred a crust which at times was firmly adherent. Removal of the crust was attended by sharp pain and left a well-defined ulcer of irregular contour, having a reddened floor and in places undermined walls. The nodules occasionally became infected, swollen, pustular and painful. After healing this inflammatory reaction recurred. Microscopically he found proliferation of the epithelium, lengthening of the interpapillary bodies, and both hyperkeratosis and parakeratosis. In one instance there was marked thickening of the epidermis with loss of the papillæ. In the corium there was a marked cellular infiltration, which localized into an abscess beneath the epithelium. At the periphery of the infiltration round cells predominated. The bloodvessels were dilated and their walls infiltrated. The pathogenesis was not clear, but it was thought that the disorder might be due to trauma followed by infection and chronic inflammation. The disorder was to be distinguished from epithelioma and pernio and excision was the method of treatment suggested.

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[1] Howard Fox, Jour. Cut. Dis., 1916, xxxiv, p. 759; "Paraffinoma." In the discussion Wise related a case in which sarcoma developed at the site of injection and proved fatal.

[2] Winkler, Archiv, April, 1915; reviewed in Practical Medicine Series, 1915, ix, p. 30. Foerster, Jour. Cut. Dis., 1918, xxxvi, p. 154.

Foerster's report is made on the observation of four cases. The growths are described as being single, ovoid, well-defined, rosy-red nodules, varying from 3 to 4 mm. in their longest diameter, imbedded in the skin, but in only one instance attached to the underlying cartilage. The nodule is flat-topped or slightly convex with a sloping margin and has a shallow central depression filled in with an adherent scale. In these instances the growths appeared suddenly without the history of preceding injury to the part, attained their maximum size within a few weeks, and then remained stationary. The duration of the disorder in this series varied from eight months to ten years, and the patients were from twenty-one to sixty-two years of age. The microscopic examination of one of the nodules showed a diffuse infiltrating and degenerative process of the corium associated with considerable epithelial hypertrophy. In the epidermis there was marked acanthosis, particularly at the center of the lesion, but there was no edema or degenerative change in this situation and the basal layer was everywhere intact. The entire corium was edematous, the lymph spaces were dilated, and there was thickening of the bloodvessels and perivascular cellular infiltration. The infiltration consisted chiefly of round cells and epithelioid cells with a few plasma cells. The collagen showed a granular degeneration and the elastic tissue was also degenerated, took a basic stain and showed fragmentation. There was no change in the cartilage. In two circumscribed areas of inflammatory reaction there were round cells, epithelioid cells, and polymorphonuclear leukocytes.

The growths are to be distinguished from keratoses and epitheliomata. Foerster considers the condition a localized chronic inflammatory process. Radiotherapy was employed and in addition the galvanocautery, electrolysis, or carbon dioxid snow were suggested as preferable methods of treatment.

LIPOMA.[1]

Fatty tumors occur in the corium and subcutaneous tissues and in the viscera, and are composed of fat-cells similar to those found normally. It is to those growths occurring in the corium and subcutaneous tissue that this chapter is devoted.

Symptoms.—These tumors may occur singly or as diffuse masses. The commonest sites are in the subcutaneous tissue over the shoulders and back. They also occur in the neck, both anteriorly and posteriorly; in the breast, gluteal region, and rarely on the face, scalp, scrotum, and labia. They may be small and flat, or nodular and lobulated, and vary in size up to that of a man's head. As a rule, the base is broad, but at times a pedunculated tumor is formed by the weight of the mass narrowing its base. The skin over the tumors is usually normal in color or slightly pigmented, and may be normal in thickness. The lobulated condition, which is characteristic of these growths, may be evident to the eye when they occur near the surface. As a rule, no subjective sensations are present, but at times, through pressure on the nerves, some pain is experienced. Secondary changes sometimes occur in the larger tumors, such as calcification, ossification, edemat changes, and liquefaction; oily cysts are thus sometimes fou The skin over the growth may necrose when secondary infect occurs, producing putrefaction.

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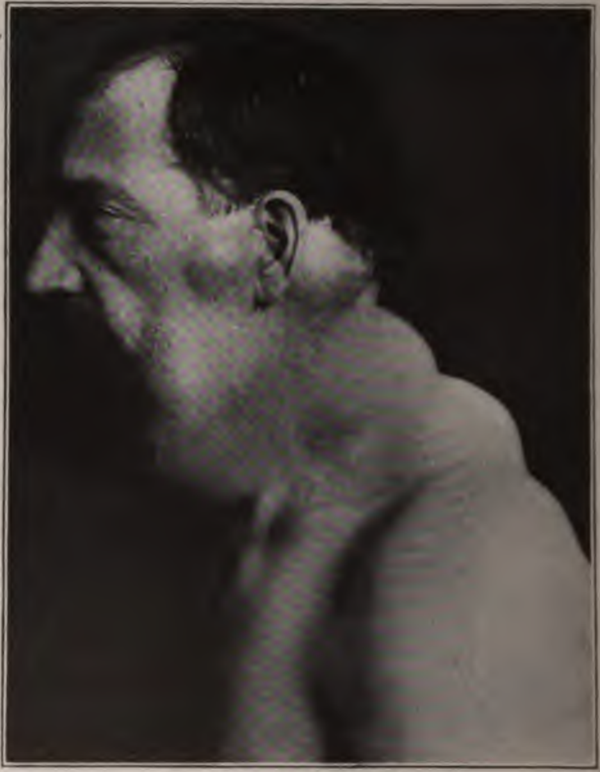
[1] Lexor-Bevan, General Surgery, pp. 796–805.

Fig. 197



Lipomata (fatty neck).

That these tumors, though composed of fatty tissue similar to normal, are independent of the general nutrition is shown by t persistence in wasting diseases (Virchow). They usually grow slo and cause little inconvenience, but the small multiple and symmetr variety often develops rapidly.

Etiology and Pathology.—The lipomata are rarely congenital; they usually develop between the ages of thirty-five and fifty years, and occur most commonly in the female sex. They are composed of groups of fat-cells, which are slightly larger than the normal, held together by a capillary network forming small lobules. These are not so distinct as in normal fatty tissue, but are united by connective-tissue trabeculæ into lobes and differently shaped masses. These tumors are enclosed within a connective-tissue capsule. The lobules are said to "bear the same relation to the nutrient artery as grapes do to the stem upon which they grow."

Diagnosis.—The important points in diagnosis are the position of the tumors, their slow growth, mobility, lobulation, and consistency.

Treatment.—The treatment consists in complete extirpation by surgical means.

Prognosis.—They are benign and do not recur after complete extirpation. They commonly grow to a given size, then remain stationary for years.

Adiposis Dolorosa[1] (*Dercum's Disease*).—This disease is commonly classed as a subvariety of obesity and is briefly referred to here for the reason that in some particulars the lesions resemble lipomata. It is a "disorder characterized by irregular symmetrical deposits of fatty masses in various portions of the body, preceded by or attended with pain." It occurs near middle life and in women. The fatty deposits may occur as large masses, which are soft in consistency. The hands, face, and feet are exempt. The true nature of the disorder is unknown. The neuralgic pains associated with fatty deposits are characteristic. Thin[2] describes two forms of multiple painful lipomata which he believes are closely related but not identical.

NEUROMA.[3]

Synonyms.—Tumor of the nerves. Fr., *Névrome;* Ger., *Neurom.*

Definition.—Neuroma is a new-growth, consisting of one or several tubercles, developed in the skin and composed of elastic, fibrous, and connective tissue, with fibers of nerves.

Symptoms.—The description appended is a summary of the symptoms detailed in the reports of Duhring,[4] Rump,[5] and Kosinski[6] of cases of neuroma affecting the skin primarily.

The patients were all men of middle life or advanced years, who exhibited upon the shoulders, arms, thighs, or buttocks numerous disseminated and defined, pinhead- to hazel-nut-sized, spherical or oval nodules. They were either painful from the beginning, or painless at the outset and painful later; Rump's case, which was an example of the false neuroma of Virchow (*fibroid tumor of the nerve*), was an exception, in that there was no pain throughout the course of the disease.

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[1] Dercum, Amer. Jour. Med. Sci., 1892, p. 521; Little, Brit. Jour. Derm., 1919, xxxi, p. 102. Case report.

[2] P. Thin: Adiposis Dolorosa and Painful Symmetrical Lipomata, Monatshefte, 1903, xxxvi, p. 281; abstr. Jour. Cut. Dis., 1903, xxi, p. 292. Osler, Modern Medicine, 1909, vi, p. 570.

[3] For a review of the subject, with full bibliography, see Krzysztalowicz, Monatshefte, 1903, xxxvi, p. 421.

[4] Amer. Jour. Med. Sci., 1873, lxvi, p. 413: Case of Painful Neuroma of the Skin; also supplement to the same, with cuts, ibid., 1881, lxxxii, p. 435.

[5] Arch. f. path. Anat. u. Phys., 1880, lxxx, p. 177.

[6] Centralb. f. Chir., 1874.

The nodules, which were not arranged along the course of nerves, were immovable, dense, and elastic; they were fixed in the corium and extended below it. In color they were purplish or pinkish, and the skin between them was unaltered, or like that enveloping the lesions —dry, uneven, and desquamative. The nodules were both tender and painful, the pain being excruciating, paroxysmal (usually lasting in Duhring's patient for an hour), and radiating. It was aggravated by temperature changes, mental emotion, and bodily movement.

Pathology.—These tumors are composed of a mixture of fine connective tissue with medullated and non-medullated nerve-fibers; and should properly be called neurofibromata. Sections of the growth in Duhring's case showed anatomically a connective-tissue stroma, interwoven with fibers, for the most part lying parallel with one another, each fiber composed of a finely granular central substance surrounded by a sheath containing numerous elongated, oval, somewhat granular nuclei. There were also yellow elastic tissue; bloodvessels with thickened and nucleated walls; and about the latter lymphoid, cell-like bodies. There was entire absence of unstriated muscular and fibrillar connective tissue. The specimen represented the true amyelinic neuromata of Virchow. In Kosinski's case non-medullated nerve-fibers and connective tissue were also discovered. In both cases exsection of a portion of nerve (brachial plexus of the one, and small sciatic of the other) was followed by considerable diminution of pain and almost entire disappearance of the growths. In Rump's case, which, as stated above, represented the fibromated and so-called fibro-nucleated tumors of Virchow, the nodules were strung upon the same nerve "like beads upon a rosary," and were similarly displayed upon its branches. Spinal, cerebral, and sympathetic fibers were all involved.

Duhring, in commenting upon these rare cases, calls attention to the distinction between purely cutaneous lesions and the generally solitary, movable, and "painful subcutaneous tubercle."

Knauss[1] reports a case in a girl of eleven years. There were over sixty tumors, varying in size from a cherry to a hen's egg. They were situated beneath the skin, were firm and elastic, and never painful. Histological examination showed them to be composed of medullated and non-medullated nerve-fibers and numerous ganglionic nerve-cells.

Little[2] recorded the case of a male patient aged thirty years, who presented numerous small bluish, reddish, hard, pea-sized, tender and painful nodules situated on the legs. Histologically the tumor was seen to be composed of nerve fibers.

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[1] Virchow's Archives, 1898, cliii, p. 29.

[2] Brit. Jour. Derm., 1919, xxxi, p. 102.

MYOMA.

Synonyms.—Leiomyoma, Dermato-myoma.

Cutaneous myomata are divided by Besnier[1] into two classes: simple myoma, or leiomyoma; and dartoic myoma.

Dartoic Myoma.—Dartoic myoma is much more common than the other form, and is of chief interest to the surgeon. It is usually single, though occasionally multiple, and occurs most frequently on the mammæ, the labia majora, the penis, and the scrotum. The tumor develops slowly, finally attaining a size varying from that of a small nut to that of an orange, and may be sessile or pedunculated. In most cases reported pain has been slight or absent, though it was marked in a case reported by Virchow. Under the influence of cold and local irritation, the tumor usually contracts, or may show a slow, vermicular motion. Some of these tumors are composed almost entirely of non-striped muscle-fibers; others are mixed with other tissues to form a *Fibromyoma*, an *Angiomyoma* (*Myoma telangiectodes*), or a *Lymphangiomyoma*.

Simple Myoma.—Simple myoma is rare.[2] Its lesions are usually multiple and occur most frequently on the upper extremities, affecting chiefly the extensor surfaces; but they may occur on other parts of the body. They begin as minute round or oval macules or papules, which develop slowly to the size of a small pea or bean, occasionally becoming larger. At first readily effaced with the finger, later they become firm and elastic to the touch. They are usually limited to one or two regions of the body, where they appear in patches without definite arrangement or grouping, and are pinkish, reddish, or of the color of the normal skin. In the beginning the growths are usually insensitive, but in most cases after slow evolution become painful on pressure, and in some instances they are the seat of paroxysms of severe pain, which occur spontaneously and at irregular intervals. Nearly all the cases reported have been in elderly people and in men. Some of the tumors may undergo involution, but usually they tend to increase in size and in number. Histological examination shows that they are limited to the derma proper, and are composed chiefly of unstriped muscle-fiber mixed with some elastic tissue, with a few vessels and nerves. They are frequently developed about the hair-follicle, and are said by different observers to arise from the erector pili muscles, the muscular coats of the bloodvessels, or from vascular tissue in connection with the glands. By many their derivation cannot be traced. Moberg[3] records a case of single myoma, accompanied by the usual symptoms, and springing, according to this observer, from the muscularis of the vessels. Sobotka[1] recorded the occurrence of an angiofibroma deeply situated, which was connected with the arrectores pilorum, and occurred in a patient with multiple myomata.

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[1] Annales, 1880, s. ii, i, p. 25; and Besnier-Doyon translation of Kaposi, ii, p. 346 (with reference to all reported cases).

[2] For a *résumé* of cases see Crocker, Brit. Jour. Derm., 1897, ix, pp. 1 and 47; Roberts, ibid., 1900, xii, p. 115; Morris, ibid., 1901, xiii, p. 8 (a case shown before the London Dermatological Society); Marschalko, Monatshefte, 1900, xxxi, p. 313 (with survey of most of the previously published cases); Sobotka, Archiv, 1908, lxxxix, p. 323; Beatty Wallace, Brit. Jour. Derm., 1907, xix, p. 1: A case of multiple leiomyomata of the skin (a clinical and histological study, with illustrations and review of literature to date); Heidingsfeld, Jour. Amer. Med. Assoc., February 16, 1907, p. 562 (clinical and histological study. Review of the literature).

[3] Nord. Med. Arkiv., 1911, abt. ii, p. 65; abstr. Brit. Jour. Derm., 1912, xxiv, p. 375.

In a case under observation by Dr. Hyde, multiple pinhead- to large-bean-sized congenital tumors were situated near the sterno-cleido-mastoid muscle of a girl nineteen years old. These were exquisitely sensitive to pressure, were capable of slight vermicular motion when irritated, and examination of the largest, after removal, disclosed smooth muscular fibers, and, in small proportion, terminal filaments of cutaneous nerves.

Sequeira[2] recorded the case of a woman aged thirty-two, who presented a collection of millet seed to a pea-sized, rounded, pink-red painful tumors under the skin of the forehead. They had always been present. Histologically the tumors were composed of unstriped muscle around a hair root arising from the erector muscles. In the case of a man, aged forty-eight years, recorded by Savatard,[3] pinhead, to cherry-sized, dull red, or yellowish-brown, nodules occurred over the right malar region, and on the external surface of the right forearm. They were painful, and sensitive to pressure and changes of atmosphere and temperature.

Diagnosis.—The diagnosis in well-marked cases is not difficult, but in some instances the recognition of the disease must depend upon a microscopical examination. Myomata have been mistaken for xanthoma tuberosum, for keloid, for lymphangioma tuberosum multiplex, and for neurofibroma.

Treatment.—The only successful treatment is by excision.

OSTEOMA CUTIS.[4]

Synonym.—Osteosis cutis.

Bony deposits in the skin and subcutaneous tissue may occur secondarily in certain diseases, such as syphilis, and also as the result of trauma, examples of which may be seen in hod carriers, in whom bony new growths are sometimes found under the skin of the shoulder where the weight of the hod falls. Osseous lesions occurring spontaneously in the skin are rare. In a few cases recorded the nature of the affection was not discovered until the microscopic examination was made. These new growths may be single and localized, or multiple, with a more or less generalized distribution. As to origin, some of the cases point strongly to their being due to misplaced embryonal cells, while others, especially those following disease or produced by trauma, may be examples of metaplasia. Coleman records the case of a patient six years of age who had a plaque of cartilaginous hardness, studded with nodules occupying about one-third of the external plantar surface of the left foot. The lesion at that time had existed for about two and a half years. This growth recurred after surgical removal.

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[1] Archiv, 1913, cxvi, p. 79; abstr. Jour. Cut. Dis., 1913, xxxi, p. 592.

[2] Brit. Jour. Derm., 1914, xxvi, p. 331, Leiomyoma.

[3] Brit. Jour. Derm. and Syph., 1920, xxxii, p. 229. Leiomyoma. Literature.

[4] Literature: Coleman, Warren: Osteosis of the Skin of the Foot; Jour. Cut. Dis., 1894, xii, p. 185. Salzer: Osteoma of the Skin; Langenbeck's Archiv, xxxiii, No. 1. Pusey: The Principles and Practice of Dermatology, p. 847. Taylor, S. and MacKenna, R. W., Liverpool: Osteoma Cutis; Jour. Cut. Dis., 1908, xxvi, p. 449. Heidingsfeld: Osteoma cutis; Archiv, 1908, xcii, p 337. Pollitzer: Ossification in a Case of Scleroderma; Jour. Cut. Dis., 1918, xxxvi, p. 271. Trimble: Osseous Formation in Lupus Erythematosus; Archiv. of Derm. and Syph., 1920, n. s. I, p. 296.

In Salser's patient there was a nodule in the scalp which in the course of four or five years attained the size of a small coin. This lesion was freely movable and was excised on account of the loss of hair in the area.

Pusey recorded a case noted by F. G. Harris, in which bone formation was found in a scar following a laparotomy wound.

Under the title *Osteoma cutis*, Taylor and MacKenna described this unusual condition: The patient, a female child aged fifteen months, had several bony deposits situated in the skin of the limbs, scalp and trunk. The largest occurred on the thigh and measured about one inch by three-quarters of an inch. The overlying skin was purplish in color and had several millet-seed-sized pearly spots on its surface. On palpation the plaque appeared well defined, hard, resilient and elastic. When bent, it would resume its former shape after the pressure was removed.

Heidingsfeld reported finding osseous formation in a pigmented nevus, occurring on the chin of a male patient aged twenty-one years. In this study numerous bony particles were found, and the author ascribes the condition to misplaced embryonal rests.

In the case of a patient described by Pollitzer calcareous deposits were discovered in patches of scleroderma. Some of these deposits were situated in ulcers. The lesions occurred upon the side of the face, the temporal region and the neck below the mastoid. True bone formation was demonstrated in some of these calcified areas.

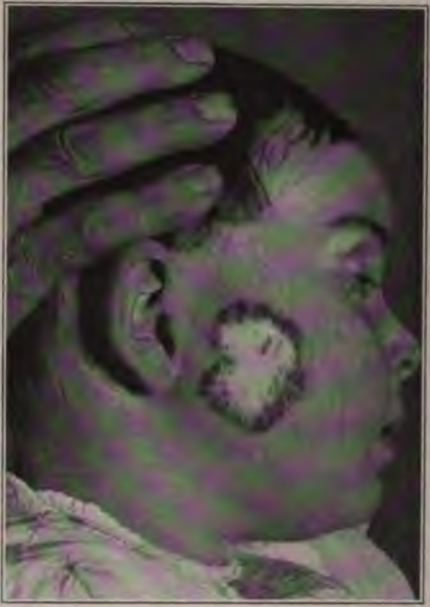
Trimble recorded the discovery of bone formation in an indurated plaque situated in the gluteal region in the case of a patient with lupus erythematosus of the face. The plaque was irregular in outline, measuring about three inches in one diameter and four in the other, densely indurated, and its surface was dead ivory white.

ANGIOMA.

The affections considered under this heading occur in two main groups: (1) angioma, which is characterized by a new-growth of the vessels; (2) telangiectasis, in which dilatation of preëxisting vessels occurs without new-vessel formation. Several classifications have been made from different standpoints. Kaposi, according to their varying characteristics, distinguished four classes: (1) telangiectasis; (2) nevus vascularis; (3) angioma elephantiasis; (4) tumor cavernosus. Unna makes still further subdivisions. Crocker and others describe these conditions under two headings: nevus vasculosus and telangiectasis.

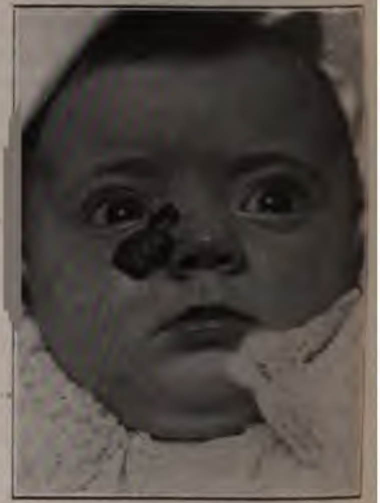
Nevus Vasculosus.—**Synonyms:** Nevus vascularis, Nevus sanguineus, Birthmark, Angioma. Fr., *Angiome, Nevus vasculaire;* Ger., *Gefässmal.* The common type of angioma of the skin is variously known as *angioma simplex, angioma simplex hyperplasticum, capillary nevus, and cutaneous nevus.* These are present at or begin shortly after birth; are round or irregular in contour; and are elevated in varying degrees, at times to a marked extent. They vary in color from a bright-red to a deep-purple, and in size from a pin's-head to a large coin or larger. They are found on the face, head, neck, arms, and other portions of the body. They may be minute at birth and gradually grow to a certain size, then remain stationary. Occasionally, in the scalp of infants comparatively rapid growth occurs. Rarely, they undergo involution, leaving delicate atrophic scars. When the lesions are composed of capillaries, they are more superficial and of a light-red color; when of veins, they are more deeply situated, and may be of large size, and smooth or lobulated, frequently the latter. They are frequently turgescent, erectile, and may be pulsating. When extending to the deeper tissues, nodular, polypoid, and lobulated masses occur, from which the blood may be pressed out, revealing a delicate skin covering the deeper, firmer portions of the tumor. This form has been called *elephantiasis telangiectodes* and also *angio-*

Fig. 198



Angioma undergoing spontaneous cicatrization. (Fordyce.)

Fig. 199

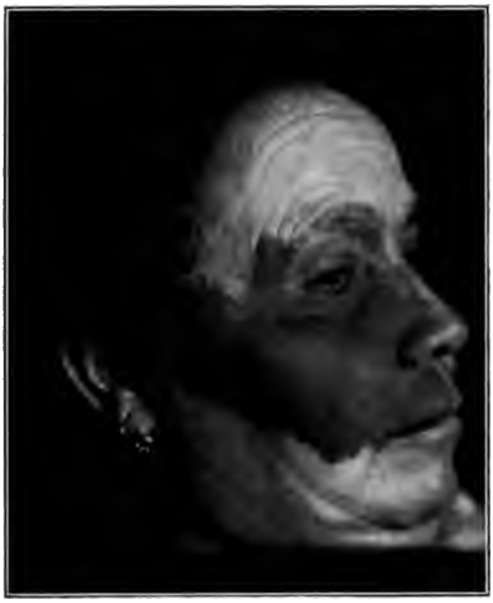


Hemangioma.

elephantiasis (*Virchow*). When occurring upon the upper lip, the latter hangs down as an irregular, bluish fold over the mouth, and when occurring upon the ear may produce quite a deformity. The lesions are temporarily increased in size by crying, excitement, or when placed in a dependent position.

Extensive angiomata of the type above described have been recorded by Ullmann,[1] in which millet-seed- to pea-sized, compressible nodules occurred. Kopp[2] recorded a similar case, with smaller nodules, situated on the scrotum and flexor surface of the limbs. Post[3] recorded one with large nodules and lobulated masses occurring along the extent of one arm, with a few smaller lesions on the chest. Histologically, they were pure hemangiomata. Occasionally, mixed cases occur, in which lymph- and bloodvessels are simultaneously involved (*angio-lymphangioma*), or bloodvessels and fatty tissue (*angio-lipoma*).

Fig. 200



Nevus vasculosus. (Fordyce.)

Another less common form is the port-wine stain (port-wine mark, birthmark, *nevus flammeus*, *Feuermal*, *tache de feu*), seen usually on the face and neck, less frequently on the trunk and limbs. The lesions may be of small size or cover large areas. On the face they are frequently unilateral and of large extent, sometimes invading the mucous membrane of the mouth. The surface is either smooth or dotted with small nodular tumors, with, in older cases, wart-like eleva tions at times. They are purplish-red or violet in color, and roun or irregular in outline. Crying, coughing, and exposure to the col produce changes in the color. With the diascope the latter may b entirely removed except for a slight brownish stain. A similar lesion, slightly developed, occurs very frequently on the back of the neck. According to Pollitzer and Depaul, this slight development of vascular nevus is present in about one-third of all new-born infants, but sometimes disappears spontaneously.

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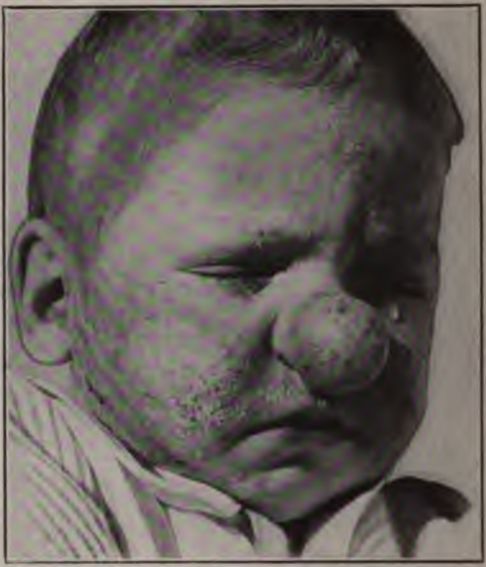
[1] Archiv, 1896, xxxv, p. 100 (photo).

[2] Ibid., 1897, xxxviii, p. 69; abstr. Brit. Jour. Derm., 1897, ix, p. 416.

[3] Jour. Cut. Dis., 1903, xxi, p. 498 (multiple angiomata).

FIG. 201



Cavernous angioma of nose. Infantile eczema of cheeks. Suitable case for radium therapy. (Morrow.)

These nevi may occur singly or there may be a number, an ceptionally extensive development may occur. Pollitzer[1] rec such a case, in which practically the entire cutaneous surfac covered with dime-sized lesions, so closely packed as almost to a network. Crocker[2] pictured another occupying the right side face, with extension over nearly three-fourths of the bod face.

Cavernous angiomata usually develop soon after birth, more later in life. They may be either superficial or deep-seated, ci scribed or diffuse. They are lobulated masses, blue or bluish- in color; and when deeply situated present merely a bluish, fla vation, covered by normal epidermis. When not encapsulated, spread into the surrounding tissues, involving even cartilage and

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[1] Internat. Atlas of Rare Skin Diseases; Fasc. xiv, Plate XLII.

[2] Atlas, Plate LVII, Nos. 3 and 4.

In structure they closely resemble the *corpus cavernosum* (Winiwarter).

Etiology and Pathology.—The cause of these various conditions is not known. Vascular nevi occur in females more frequently than in males. The popular conception of prenatal maternal impressions, although apparently supported by evidence, is believed to have no significance, the striking examples being coincidences, rather than instances of cause and effect. Unna's theory of intrauterine pressure is supported by the occurrence of the congenital angiomata in regions where the fetal skin, such as that over the frontal and occipital regions and the surface of the knees, comes in contact with the bony prominences of the pelvis. Pollitzer[1] adds to this the compression of the cutaneous vessels from an unusual degree of flexion or extension of the head or limbs. This observer found lesions on the back of the head in 40 out of 114 new-born infants he examined. Depaul (quoted by Crocker), in a similar investigation, found the lesions in this situation in one-third of the cases. Virchow's fissural theory is supported by the occurrence of these lesions most abundantly in the position of the earlier fetal clefts.

Histologically, the vascular nevi have their seat chiefly in the superficial layers of the corium. In some instances they extend deeply and involve the hypoderm. The venous capillaries are chiefly implicated in the formation of vascular nevi (Virchow). The walls of the capillaries become thickened and, owing to endothelial proliferation, resemble the veins of the subcutaneous tissue. In the cavernous angioma the proliferation is most marked about the sweat-glands, hair-follicles, and fat-globules (Billroth). Embryonal vascular growths spring from these and as they multiply and develop are enforced by proliferation of connective tissue. Winiwarter[2] believes that they are developed in the same way as erectile tissue; namely, by a process of budding of solid vascular bodies from the walls of the veins, which become excavated and lined by endothelium.

Diagnosis.—The ordinary lesions of angioma are readily recognized by their color, size, shape, and obvious vascular constituents. Anderson calls attention to the importance of differentiating encephalocele due to the failure of ossification of the ethmoid and frontal bones at the root of the nose. Operations upon such tumors supposed to be angiomatous in character have resulted fatally. Lobulation, great distention (when a child is crying), a superficial rather than deep and complete vascularization of the smooth and glossy skin of the tumor, and a double pulsation, all point to frontal encephalocele.

Treatment.—The treatment of vascular nevi varies according to the type of lesion. The methods that are most satisfactory and chiefly employed are the following: refrigeration with carbon-dioxid snow or liquid air; cauterization by means of chemicals, electricity, or the actual cautery; radiotherapy, using x-rays or radium; electrolysis, and surgical procedures other than those above mentioned, such as excision and ligation.

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[1] Amer. Textbook of Gen.-Urin. Dis., Syph., and Dis. of the Skin. Bangs and Hardaway, 1898, p. 1006.

[2] Die Chirurgischen Krankheiten der Haut. Stuttgart, 1892. Quoted by MacLeod, p. 261.

In the capillary nevi, carbon-dioxid snow furnishes a simple and efficient means of treatment.[1] A stick or cylinder of the snow is moulded in shape to correspond with the lesion and is applied for from three or five to ten or twenty or more seconds. Care must be taken not to freeze the lesion too deeply, as this produces destruction of tissue further than is necessary, being followed by the usual symptoms of a congelation, dermatitis, and possibly sloughing. Only moderate pressure should be used and the freezing carried only to the point of whitening the angiomatous tissue. The color returns in a few seconds, after which reaction occurs, depending on the severity of the treatment. A crust commonly forms within three days, which falls between the seventh and fourteenth day, after which time, in case it is necessary, the treatment may be repeated. The most important factors in this treatment are the time of exposure and the amount of pressure exerted.

Liquid air may be substituted for carbon-dioxid snow, in case it is obtainable, and may be of greater service in certain cases. Its chief disadvantages are: (1) the difficulty or impossibility of obtaining it; and (2) its mode of application. Being a liquid, it has to be applied on a cotton or other applicator, and it is therefore somewhat difficult to limit the application to the lesion.

Electrolysis may be employed with advantage in certain of these cases, the technique being that employed for hypertrichosis (which see), except that the current is not broken until the needle or needles have been removed, the latter being gradually withdrawn, the electrolytic process being kept up until the needle makes its exit from the tissue, thus sealing the opening and preventing the hemorrhage which otherwise would occur.

In certain deeper cases x-rays may be employed, and in combination with other methods are of distinct value. Radium is highly recommended in all types of the disorder by Wickham.[2] After several years' experience with radium by the author it has become the method of choice in the circumscribed elevated hemangioma type. The disadvantage in the use of x-rays and radium is the possible future development of telangiectasia in the area, which is disfiguring to a certain degree.

Electrical desiccation has been employed successfully in certain types by Pfahler[3] and others. Clark[4] obtained good results with ultraviolet rays employed with compression.

In the simple and superficial cases of angioma, pressure by means of flexile collodion, alone or combined with puncture, was recommended by Stelwagon,[1] who used a triangular-edged needle, making the punctures from one-eighth to one-fourth of an inch apart, and employed proper aseptic precautions.

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[1] *Cf.* section on Therapeutics for technique of preparation.

[2] Wickham and Degrais, Radiumthérapie, Paris, 1909. (For technique, *Cf.* section on General Therapeutics.)

[3] Jour. Cut. Dis., 1916, xxxiv, p. 466.

[4] Therapeutic Gazette, 1916, xxxii, p. 312.

Various acids have been employed, using either a needle or pointed stick in making several punctures over the area, after which the lesion is dressed with an ordinary surgical dressing. Sherwell[2] employs multiple puncture with a set of fine needles in a needle-holder similar to that used for electrolysis. These are dipped in a 25 to 50 per cent. solution of chromic acid, and then made to penetrate the part to be attacked. The bleeding is readily arrested by pressure, after which the patch is covered with several layers of flexible collodion. This procedure is of value in circumscribed patches of superficial character and relatively limited area. By it one can succeed in removing port-wine marks, with the result of producing a somewhat irregular cicatriform tissue much less disfiguring than the original blemish.

Other methods employed are the ligature, when practicable; puncture with incandescent needles; topical application of caustics other than those named above, such as potassium hydroxid, phenol, and corrosive sublimate; and total excision, the latter being practicable in relatively small growths. Larger growths also can be removed and the surface covered with skin-grafts. The galvano-cautery and the thermo-cautery are both valuable in the destruction of capillaries. The old method of multiple vaccination about and upon the involved area is sometimes followed by good results, but whether in consequence of the retraction of tissue under the influence of the inflammation excited, or of the destructive results of the suppuration induced, or of an indefinite caustic effect, is not clear.

The treatment of port-wine marks is only moderately satisfactory. Improvement may be made by several of the above methods. Their entire eradication is difficult. When perceptibly elevated, much more can be done than is otherwise the case. Liquid air, carbon-dioxid snow, and radium have given best results in this type.

Angioma cavernosum and other deeply situated angiomata require surgical interference.

Prognosis.—The prognosis may be formulated from what precedes. Excellent results, with practically no sequels, are frequently obtained in capillary nevi by treatment. The deeper lesions are followed by some scar-formation, and in rare instances may recur after surgical removal.[3] In port-wine stains the prognosis is guarded. Improvement can be promised, but complete eradication without atrophy is not possible in most instances.

Telangiectasis (*Acquired vascular dilatation*).—Telangiectases occur as localized or occasionally generalized vascular dilatations of pre-existing bloodvessels, associated at times with the development of new vessels. One of the common forms is that described as *nevus araneus*, "spider nevus," or the so-called "spider cancer." It is characterized by a central, slightly elevated, red dot or spot, pinhead-sized or slightly larger, from which fine bloodvessels radiate like the spokes of a wheel. Early it is hardly perceptible, but gradually becomes distinct, until a small, permanent patch is formed. Rarely the lesions may assume larger proportions. From one to several lesions occur as a rule, situated most frequently on the cheeks, beneath the eyes and on the nose; occasionally, they are seen on the neck, chest, and other parts of the body. They may develop spontaneously or follow slight trauma, such as a mosquito-bite,[1] or the injury caused by the forcible removal of a comedone. They are usually seen in women and children with delicate skins. Crocker[2] records a case beginning in a boy of five, in whom they were still developing at the age of fourteen; and quotes a more generalized case recorded by Mandelbaum.[3]

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[1] Diseases of the Skin, p. 685.

[2] Amer. Archives of Derm., 1879, v, p. 354.

[3] Post, loc. cit.

Telangiectasia occurs in association with rosacea, xeroderma pigmentosum, chronic *x*-ray dermatitis, lupus erythematosus, angiokeratoma, morphea, syphilis, and other cutaneous disorders. It has been recorded in connection with exophthalmic goiter.[4] It occurs also following continued exposure of the face to wind and sun, and in persons in whom a chronic congestion of the face is present from various causes.

Multiple telangiectatic areas on the face and body have been noted occurring in several members of one family, such a group having been studied by the author. A group of cases in which multiple telangiectases occurred in connection with epistaxis and other hemorrhages has been recorded by Osler, Weber, and others. These not infrequently occur in several members of a family.[5]

Another form, papillary ectasis, or varices, occurs in people past middle life, chiefly about the trunk and upper part of the chest, as slightly raised, hempseed-sized, bright-red or purplish areas, resembling blood extravasation (Crocker), but consisting of tufts of dilated capillaries.

A very extensive case of telangiectasis was recorded by Frick.[6]

Treatment.—The treatment of telangiectasis is that of capillary angiomata, previously described. Electrolysis is the best method in most cases. In the telangiectatic vessels occurring in connection with various diseases, such as rosacea, this method gives excellent results. It is well not to employ it in cases of rosacea until after the inflammatory processes have been cleared up by other methods of treatment. In the so-called "spider cancer," the electrolytic needle is inserted in the center of the lesion, which commonly seals the surrounding vessels at the same time. Only a moderate exposure to this process should be employed, on account of its scar-formation. A current of from one to one and one-half milliamperes may be used, and the needle should be removed as soon as the area becomes whitened, which only requires a few seconds, the needle being removed in the same manner as described in connection with the angiomata. Multiple scarification, as described in connection with lupus, is of value in many cases, and may be employed particularly in the marked telangiectasia occurring with rosacea. In the generalized cases treatment cannot be employed.

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[1] Whitfield, Allbutt and Rolleston's System, p. 587.

[2] Diseases of the Skin, p. 967.

[3] Vierteljahr, 1882, ix, p. 213.

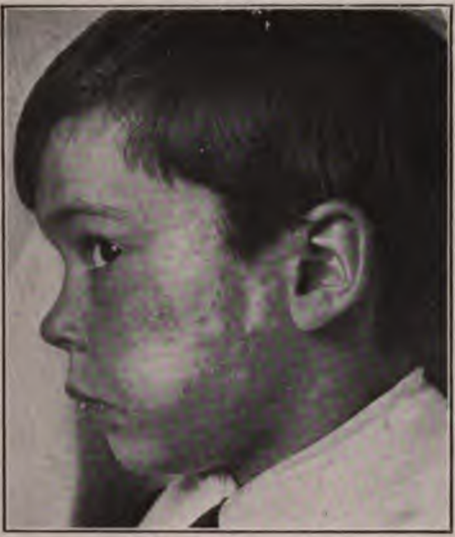
[4] Hyde, Brit. Jour. Derm., 1908, xx, p. 33.

[5] Colcott Fox, Brit. Jour. Derm., 1908, xx, p. 145 (a case of bilateral telangiectasis of the trunk, with a history of marked epistaxis in childhood and a recent rectal hemorrhage; with review of the literature).

[6] Jour. Cut. Dis., 1912, xxx, p. 334.

Nevus Anemicus.—Under this title Vörner[1] described an affection of the skin not previously recognized. Several examples have since been recorded in this country.[2]

FIG. 202



Nevus anemicus.

The disorder is characterized by white macules of variable size in which no change is noted in the skin other than the loss of normal color. The lesions have varied from pea- to silver-quarter-size, and larger, and have been noted on the back, chest, face and other areas. No subjective symptoms are present. The affection is to be distinguished chiefly from vitiligo. The absence of a pigmented border is important in making the distinction. Harris suggested rubbing the surface with ice, which accentuates the color change by inducing redness about the lesions.

Histologically, partial or complete absence of bloodvessels was noted in the area. Stein[3] found no histological changes demonstrable, and quotes Fischer as attributing the condition to a nervous disturbance of the vessel formation.

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[1] Archiv, December, 1906, lxxxii, p. 391.

[2] Lane, Jour. Cut. Dis., 1916, xxxiv, p. 602; case report with literature. Harris, ibid., 1917, xxxv, pp. 45, 280 and 852; case reports. MacKee, ibid., 1918, xxxvi, p. 200; case report. Goldenberg, ibid., 1919, xxxvii, p. 142; case report.

[3] Ibid., 1910, ci, p. 411: Ueber, Nevus anemicus.

The affection persists and usually treatment is not sought, its presence being detected when the skin is examined for other conditions.

ANGIOMA SERPIGINOSUM.[1]

Synonyms.—Infective angioma, Nevus lupus.

This rare disease was first described and figured by Hutchinson,[2] under the title "A Peculiar Form of Serpiginous and Infective Nevoid Disease." Crocker[3] described the disease under the title of "Angioma Serpiginosum."

The lesions of the disorder occur in several forms, which may, in certain cases, be restricted to one or more of the varieties, or in others to a combination of the entire group. The most striking lesions, according to most writers, are bright-reddish puncta, resembling grains of cayenne pepper, arranged in oval or circular rings, which are definitely outlined and are of varying sizes. Other lesions in the neighborhood, termed "infective satellites," may be seen when the disease is spreading. Wise divides the lesions into seven types: (1) bright-red, pin-point dots, barely visible to the naked eye, but on account of their vivid tint standing out prominently and distinctly in contrast to the surrounding diffusely reddened skin; (2) areas of diffuse, uniform redness; (3) slightly raised vascular papules; (4) delicate perivascular rings; (5) meshworks of irregularly curved and crooked lines; (6) pigmented lesions; and (7) atrophic spots. In addition to these, there may occur some branny desquamation. The eruption begins insidiously and progresses slowly, and any portion of the cutaneous surface may be attacked. The lesions, as a rule, are symmetrical, and entirely devoid of subjective sensations. The increase in the eruption is by the formation of new areas in the neighborhood of older lesions, with very gradual fusion. Crocker states that the cayenne-pepper-like grains, which consist of bright-red vascular points imbedded in the skin, are formed into groups, which spread peripherally, clearing in the center, thus forming rings. In Wise's case the eruption increased by coalescence of new lesions springing from different centers. In the older lesions the skin presents a glistening, atrophic appearance. The eruptive manifestations of the disease sometimes begin in a vascular nevus. In a case observed by Hyde, in a female infant, the lesions developed as a consequence of a congenital nevus of the vulva. Others begin independently of such a lesion. Although in distribution the disease may become nsive, it produces no effect on the general health of the ient.

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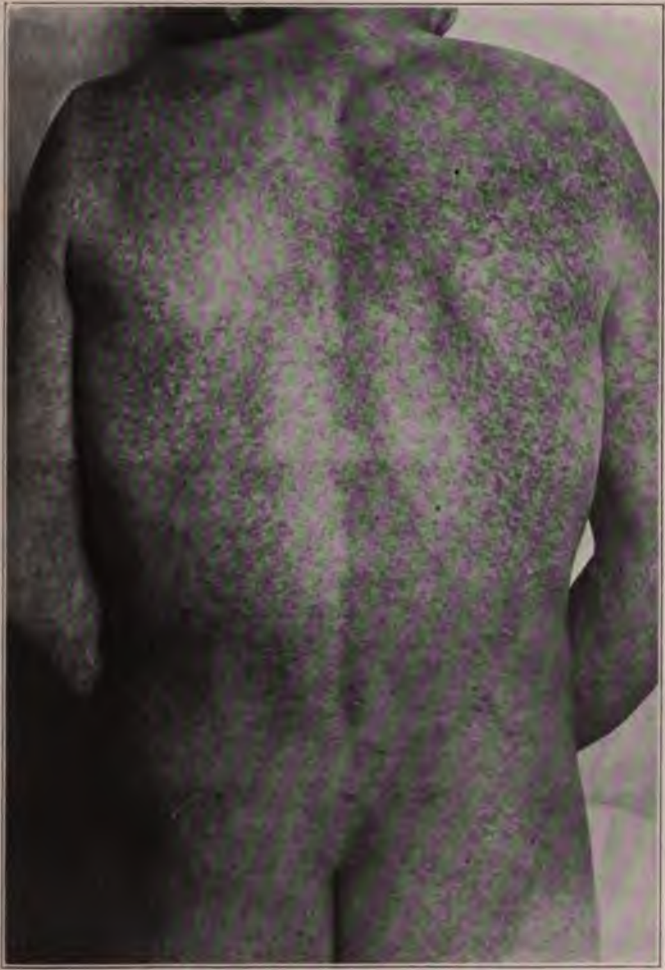
[1] Wise, Jour. Cut. Dis., 1913, xxxi, p. 725, and Transactions of the Section on Dermatology of the American Medical Association, 1913, p. 177. In this article three cases are described, the clinical part by Wise, the histological by Pollitzer. In addition, there is a review of all the recorded cases (25) and the allied conditions, with a comprehensive study of the entire subject.

[2] Archives of Surgery, 1899, i, p. 289, Plate IX.

[3] Brit. Jour. Derm., 1894, vi, p. 367.

tiology.—Nothing is known definitely concerning the cause of the rder. One case was supposed to have originated in violent muscular rcise. In several of the reported cases the starting point of the

Fig. 203

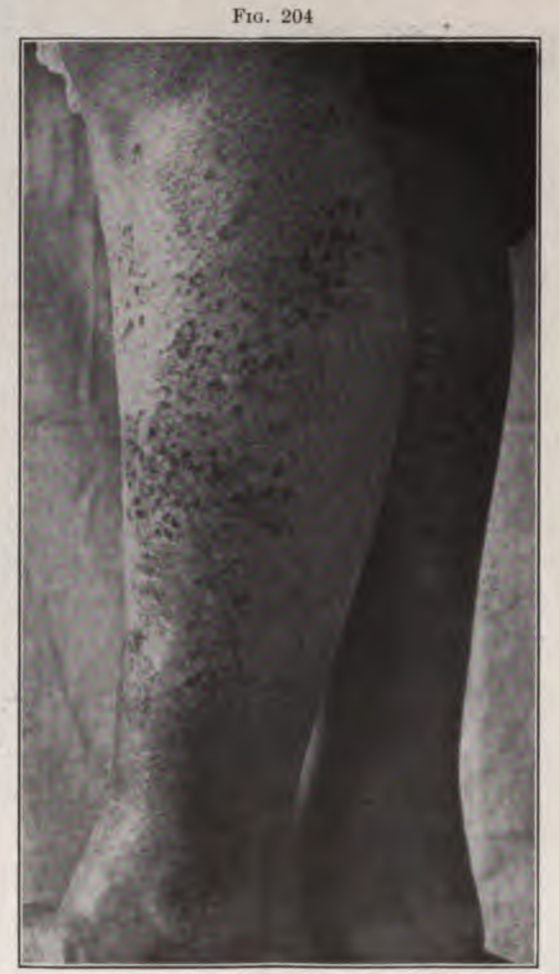


Angioma serpiginosum.

tion has been in a port-wine mark. The major portion of the s have occurred in females, and it may occur at any age from ncy to sixty years.

athology.—Past histological studies have revealed very little of ortance relative to the disorder. In Pollitzer's study, however, the findings are more conclusive. He sums these up as follows: There is a progressive perivascular infiltration of the papillary and subpapillary layers in circumscribed areas, corresponding to the small, deep-red papules and the rings of the annular lesions. In these areas there is a proliferation of capillaries, as in inflammatory tissue, and there are marked endothelial proliferation and swelling. The elastic tissue appears normal; the collagenous tissue is somewhat degenerated. The epidermis, over the areas of infiltration, shows a high degree of intercellular edema, affecting the lower layers of the rete, and extending with diminished intensity as far as the stratum granulosum. The lower layers of the rete are invaded by the lymphocytic cells over the papillary infiltration. A few polymorphonuclear leukocytes are

FIG. 204



Angioma serpiginosum. (Fred Wise.)

present, and many of the epidermic cells have undergone a vesicular and hydropic degeneration. There is no sign whatever of extravasated blood, as in a hemorrhagic process, or of widely dilated capillaries and veins, as in some forms of vascular nevus.

From these findings, the writer concludes that the essential process is a low-grade inflammation, affecting primarily the capillary areas of the papillary and subpapillary regions, with secondary effects in the epidermis.

Diagnosis.—The disease is to be recognized by the vascular puncta, and by their special tendency to grouping; the formation of circinate lesions; and by the peculiar manner in which the lesion gradually spreads. It is to be differentiated from purpura annularis telangiectodes, which disease is classed with it in Crocker's description. The latter occurs almost exclusively on the legs and is characterized by the presence of purpuric lesions and a history of the disappearance and recurrence of the lesions, points which do not occur in angioma serpiginosum. From simple telangiectases and certain cases of nevus vascularis it must also be differentiated.

Treatment.—The treatment consists only of surgical measures, either ablation or destructive cauterization.

Prognosis.—The disease in some cases, after reaching a certain stage of development, becomes arrested and remains stationary. In others the lesions disappear spontaneously, at least to a certain extent.

GRANULOMA PYOGENICUM.

Synonyms.—Botryomycosis hominis, *Granulomes à pédicule bénin* (Frédéric), Telangiectatic granuloma (Kuttner).

The disease described under the above titles consists of small pedunculated tumors, frequently occurring on the site of an injury. A group of four cases described as *Botryomycose humaine* was presented by Poncet and Dor[1] at the Congress of Surgery in Paris, in 1897. These authors identified the affection described by them with that described in 1870 by Bollinger as *Botryomycosis*, a disorder characterized by inflammatory tumors occurring on the scrotum and spermatic cord of horses following castration. Crocker[2] described the disease in 1903, under the title *Granuloma pyogenicum*. Hartzell[3] first described the condition in America, reporting four cases. Wile[4] recorded two cases and described and discussed the pathology and bacteriology. Lenormont[5] and Heuck[6] have recently made comprehensive reports of this disease.[7]

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[1] Congrès de Chirurgie de Paris, October 18, 1897; and Lyon Méd., October 24, 1897, and January 30, February 6, 1898 (abstr. Brit. Jour. Derm., 1898, x, p. 209).

[2] Diseases of the Skin, 3d ed., 1903, p. 1081.

[3] Jour. Cut. Dis., 1904, xxii, p. 520 (*résumé* and literature).

[4] Ibid., 1910, xxviii, p. 663 (*résumé* of French and German cases).

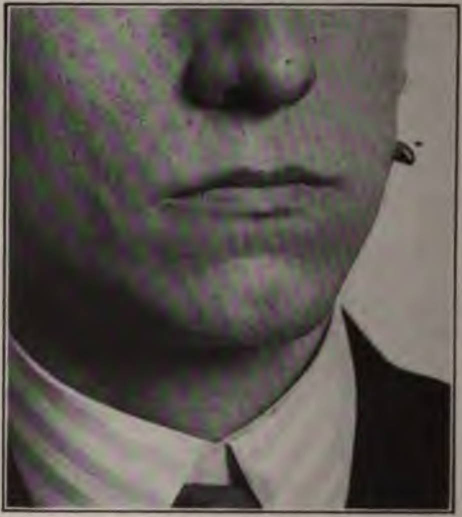
[5] Annales, 1910, i, p. 161.

[6] Zeitschrift, 1912, pp. 221, 324, 404.

[7] Montgomery and Culver, Jour. Cut. Dis., 1917, xxxv, p. 338, Granuloma pyogenicum and High Blood-pressure. Report of five cases.

Symptoms.—The disease is characterized by rapidly growing lesions, often occurring in the site of an injury. The lesions are described as pedunculated tumors, varying in size from that of a pea to a cherry or slightly larger. They are vascular and bleed easily. In color they are bright-red, bluish-red, or brownish-red. They are frequently moist and fleshy, or they may be mushroom-like. At times the surface presents areas of ulceration and necrosis, and is bathed in a purulent secretion. The tumors may secrete a foul-smelling, seropurulent exudate, or may have some crust-formation. The surface is smooth, or may show crevices filled with pus, or be frambesiform. The lesions are benign connective tissue new-growths, but tend to recur. Crocker states that they are exaggerations of what is popularly known as "proud flesh." The sites of predilection are the hand and foot, the lip, cheek, chin, shoulder, back, and umbilical region.

FIG. 205



Granuloma pyogenicum.

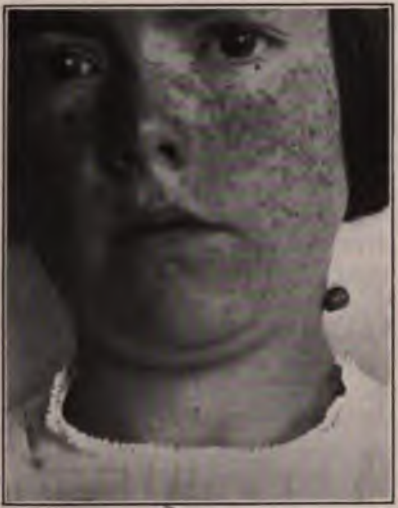
Etiology.—The cause is not definitely proven. Traumatism, followed by infection, is strongly suggested. Crocker believes they are produced by pus-cocci. Hartzell found only the ordinary yellow staphylococci of suppuration. Wile says: "The etiological factor is not a fungus, but probably *Staphylococcus aureus,* perhaps in an unfavorable soil or in an attenuated form." In the original cases in animals, yellow granular masses were present, termed *botryomyces,* which were thought to be the causative agent. Similar findings were made in the early cases occurring in man. After further research, this hypothesis was abandoned.

The disease has occurred at all ages, and about equally in the sexes. About one-half of the cases reported followed traumatism. There is no evidence of transference of the disease either from animal or man to man.

Pathology.—Practically all observers agree on the histological findings. The neoplasm is composed of granulation tissue, largely composed of young connective tissue and new-formed bloodvessels. The bloodvessels may be so large and so numerous as to suggest an angioma. The cellular infiltration is made up largely of connective-tissue cells. There are also varying numbers of mast-cells, polynuclear leucocytes, plasma and small round cells. In the different cases clumps of micrococci are also found in varying situations.

In addition to the microörganisms mentioned under etiology, the following have been found: *streptococci*, *Bacillus coli, Bacillus proteus*, and, finally, an *ameba*.

FIG. 206



Granuloma pyogenicum. (The small tumor is the lesion.)

Diagnosis.—These neoplasms are to be distinguished from angiomata and papillomata.

Treatment.—Excision, followed by cauterization, is recommended. Radiotherapy has given perfect results in several cases treated by the author.

Prognosis.—The prognosis is good, but the possible recurrence should be remembered.

LYMPHANGIOMA.

In the present state of knowledge of this subject, it is not always possible to draw sharp dividing lines between lymphatic new-growths on the one side and simple lymphangiectasis on the other. It is probable that the two processes often are associated.[1]

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[1] For review of literature of the subject, consult Francis, Brit. Jour. Derm., 1893, v, pp. 33 and 65.

Lymphangiectasis.—Lymphangiectasis, uncomplicated by growth of new vessels, may occur in the superficial or deep lymphatics. When superficial, pinhead- to pea-sized, isolated or grouped vesicles form, which have the color of the normal skin, and disappear temporarily under pressure, and which do not break easily, but on rupture give exit to a continuous or intermittent flow of lymphatic fluid. Elliot[1] describes a case of this kind in which the vesicles bordered old scar-tissue and were seemingly identical in character with the lesions of lymphangioma circumscriptum, but histological examination showed them to be formed by simple dilatation of the lymphatic capillaries, due, probably, to mechanical obstruction.

Lymphangiectasis of the deeper vessels often produces no change visible on the skin and can then only be recognized by palpation; or it may be displayed in raised, irregular cords, or in chains of nodules. Türk[2] records an idiopathic lymphangiectasis occurring on the penis of a patient, exhibited as a nodule 2 cm. long by 0.5 cm. in height. The histology was characteristic. Müller[3] records a case of lymphangiectasis with lymphorrhea of the scrotum and elephantiasis of the leg. Following injuries or inflammation, it may be acute, but usually it is chronic, and occurs most frequently on the lower extremities and in parts in which the return current of the circulation is in some way impeded. The skin may become the seat of soft nodules, which may rupture and form lymphatic fistulæ; but more frequently the greatest changes occur in the deeper structures, resulting in elephantiasis, in phlegmon, or in lesions of periosteum and bone, the skin of the affected region being edematous, infiltrated, ulcerating, or cicatricial.

Simple Lymphangioma.—Simple lymphangioma may occur upon any part of the body in the form of circumscribed, elastic tumors, made up of enlarged lymphatics, which are partly the result of dilatation of previously existing vessels and partly of new-formation. The skin over such tumors may be unchanged, or it may be reddened and thickened. Upon the tongue the condition is called *Macroglossia*, and upon the lips *Macrochilia*. In more extensive cases there is hypertrophy of the surrounding tissues, as in deep-seated lymphangiectasis. Many of the diffuse forms of lymphangioma constitute firm or lax tumors of such size as to be termed *Elephantiasis lymphangiectatica* or *Pachydermia lymphangiectatica*. These tumors often contain large lymph-filled sacs or lacunæ, enveloped in hypertrophied muscular and connective tissue, and an edematous integument. Some of the elephantiasic deformities of this character are fully as enormous as the extreme distortions of elephantiasis proper. In many instances there occurs an association of hemangioma and lymphangioma in the same area, the former receding, the latter progressing in time. An unusual example of lymphangioma with lesions simulating those of xanthoma is recorded by Gottheil.[4] He states that a similar case was previously observed by Thibierge. In the former case, degeneration of elastic tissue, characteristic of pseudo-xanthoma elasticum, was discovered in combination with the lymphangioma.

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[1] Jour. Cut. Dis., 1894, xii, p. 137.

[2] Zeitschrift, 1912, xix, No. 2, p. 138; abstr. Jour. Cut. Dis., 1912, xxx, p. 692.

[3] Archiv, lxxxii, 1906, p. 111; abstr. Brit. Jour. Derm., 1908, xx, p. 19.

[4] Jour. Cut. Dis., 1909, xxvii, p. 277.

Simple lymphangiomata may be congenital.[1] Their cause is unknown. It is supposed that they are produced by toxic or other irritating influences. They are often the seat of a recurrent, circumscribed inflammation of erysipelatous type. Anatomically, the lesions are found to consist of greatly developed lymphatic vessels and spaces, lined with epithelium and enveloped in small-celled connective tissue stroma. The treatment of the larger lesions only is surgical.

Lymphadenectasia is a name given by Virchow to tumors, usually in the axillary or inguinal regions, where the lymphatic vessels in the lymphatic glands dilate or multiply so as to form large lesions. The lymph scrotum due to the presence of *Filaria sanguinis hominis* is described elsewhere.

Cystic Lymphangioma belongs to the domain of surgery. It occurs in the form of multilocular cysts, usually congenital in origin and most frequently situated in the neck.

Lymphangioma Circumscriptum (*Lymphangioma cavernosum; Lymphangiectodes; Lymphangioma capillare varicosum; Lupus lymphaticus.* Fr., *Angiome cystique*). This is practically the only form of lymphangioma entitled to special consideration by the dermatologist. It is a rare form of skin disease and is illustrated well in the case reported by Morris.[2] Cases have been reported by Török,[3] White, Francis,[4] Hartzell, Elliot, Gilchrist, Brocq and Bernard,[5] Schnabel,[6] and others. A number of typical examples have been presented before the New York, Philadelphia, Boston, and Chicago dermatological societies during the past fifteen years.

Symptoms. — The characteristic lesions are small, deep-seated vesicles, generally described as resembling frog's spawn. They are usually closely crowded in irregularly shaped groups, from 8 to 20 millimeters in diameter, with normal skin between. These groups have no regular arrangement or distribution. There are sometimes a few scattered vesicles about or between the borders of the groups, which may coalesce to form new patches. There are usually several of these groups, but they are confined, as a rule, to one small region of the body. The most common sites, according to Francis, who has collated reports of twenty-eight cases, are on the upper parts of the extremities and the mucous membrane of the mouth, pharynx, and tongue. A site not infrequently attacked is the axillary and scapular region (Bowen), a fact borne out in several of the American cases. In a large majority of the cases reported the lesions occurred on the left side of the body. The vesicles are deep seated, with thick walls, and vary in size from that of a pinhead to that of a small pea. The newer and scattered vesicles may be colorless or have a yellowish or pinkish tinge. The skin over the older lesions may hypertrophy and produce growths that are easily mistaken for warts, and may even result in decidedly warty projections. Other lesions may be more or less covered with telangiectases and vascular dots or tufts, which may be present to such an extent as to obscure the primary vesicle-formation. When punctured, the lesions give exit to clear, colorless fluid, in greater quantity than the vesicles contain, which may be tinged with blood, the result of hemorrhage.

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[1] Vollmer, Archiv, 1903, lxv, p. 343 (report of a rare case, with illustrations, histology, and bibliography).

[2] Internat. Atlas, 1889, No. 1.

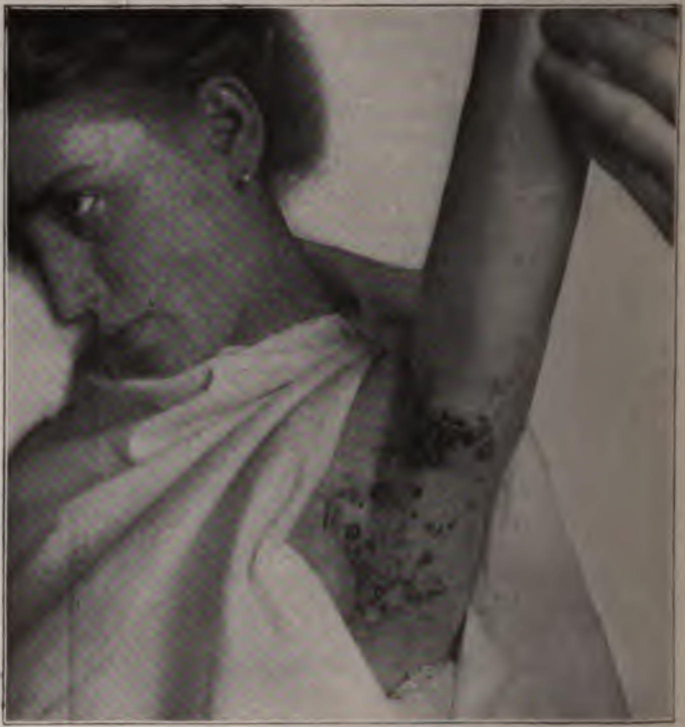
[3] Monatshefte, 1892, xiv, p. 169 (with critical review of previously published cases).

[4] Brit. Jour. Derm., 1893, v, p. 33 (with review of literature).

[5] Annales, 1898, s. iii, ix, p. 305 (full discussion of the subject, with review of the literature).

[6] Archiv, 1901, lvi, p. 177 (with histology and references).

Fig. 207



Lymphangiectodes. (McEwen.)

In some cases the lesions and skin about them become the seat of a recurrent inflammation of erysipelatous type,[1] such as not infrequently complicates other forms of lymphangioma. Probably as a result of these attacks of inflammation, there are often infiltration, ing, and even true hypertrophy of the deeper layers of the rming a sort of local elephantiasis.

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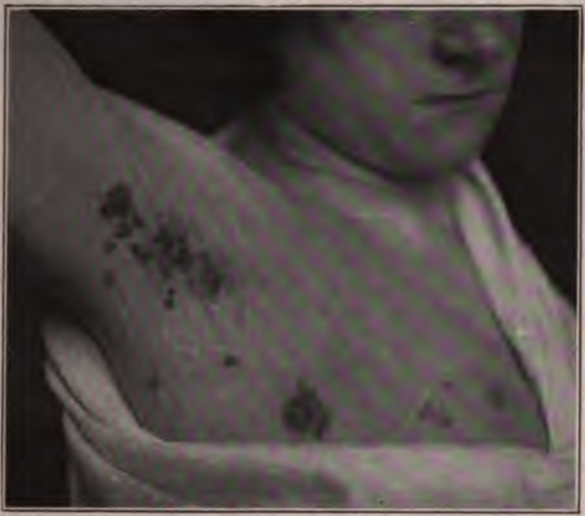
[1] *Cf.* White's report, Jour. Cut. Dis., 1894, xii, p. 474; also Bowen's article in Twentieth Century Practice, v, p. 687.

disease in most cases reported began in early childhood and ed very slowly, often remaining stationary for years. In but e has spontaneous involution been recorded.

gy.—As the disease usually makes its appearance in infancy childhood, it is probable that its origin is due to some congenital It has appeared a number of times in connection with nevi, s followed surgical operations, bordering the scars produced by rator. It is possible that such cases are simple lymphangi- of the capillary vessels due to blocking of the larger channels scar-tissue. The disorder not infrequently develops over or e site of a deeply situated hemangioma or lymphangioma, some instances after surgical removal of the deeper lesion.

Fig. 208



Lymphangiectodes.

logy.—The vesicles, or cysts, are found on section to be l in the upper part of the corium. These cysts are shown to n endothelial lining, and undoubtedly are dilated or newly- lymph-capillaries. Immediately about the cysts and dilated tics in an early uncomplicated lesion Bowen found considerable ion of round cells, but no other changes in the corium, while dermis was slightly thinned. In older lesions there is hyper- of the epidermal layers, and sometimes of the deeper parts corium. In other cases there are more or less dilatation and t new-growth of the blood-capillaries. This change in the ssels may be slight, or so marked as to form the chief feature disease both clinically and pathologically. In consequence, confusing reports have been made by different observers regarding the structure and origin of these growths, many of which seem entitled to the name *hemato-lymphangioma.*

Diagnosis.—The disease can usually be recognized by the presence of the peculiar vesicles which give exit to lymph when punctured; by its occurrence in early life; by its slow and persistent course; by the presence often of warty and telangiectatic areas over its surface; and by its history of recurrence after most methods of treatment.

Treatment.—Radiotherapy offers the best hope of removal of the lesions without recurrence. Cases so treated successfully are recorded by Engman and Mook,[1] Hartzell and Knowles, the author,[2] and by Dore.[3] Trimble[4] recorded successful treatment in three instances with radium. Other methods more or less successful are the removal of the lesions by surgical measures and by electrolysis.

Prognosis.—The disease tends to persist after developing to a certain extent, varying in different cases. The intercurrent attacks of inflammatory processes are to be remembered. With treatment with *x*-rays, promise of complete eradication is good.

MOLLUSCUM CONTAGIOSUM.[5]

Synonyms.—Molluscum sebaceum; Epithelioma contagiosum; Molluscum epitheliale; *Acné varioliforme* (Bazin).

Definition.—Molluscum contagiosum is a mildly contagious autoinoculable disease of the skin characterized by pinhead- to pea-sized waxy lesions, usually presenting a central depression.

The disorder was described in 1817 by Bateman under the title given above, and has been the subject of much study by many observers.

Symptoms.—The lesions begin a pinhead-sized elevations of the skin which slowly or rapidly increase in size until they reach the dimension of a small pea or larger. When fully developed they are rounded, semiglobular or slightly flattened, moderately elevated, waxy whitish, well circumscribed, firm, small, button-like lesions, which may be few and localized, or numerous and generally distributed. In the center of the lesion there exists a small, dark-colored opening from which a milky and curd-like substance may be expressed. Occasionally this material desiccates and protrudes from the orifice. Sometimes more than one opening is present. Owing to the waxy appearance with the central dark-colored opening, Hutchinson likened the lesions to small pearl buttons. This appearance may suggest the lesion of variola, which accounts for the title given the disorder by Bazin (*Acné varioliforme*).

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[1] Jour. Cut. Dis., 1913, xxxi, p. 266.

[2] Ibid., p. 267.

[3] Brit. Jour. Derm., 1913, xxv, p. 191 (a case of Sir Malcolm Morris' entirely cleared up with four *x*-ray exposures).

[4] Jour. Cut. Dis., 1919, xxxvii, p. 409.

[5] For complete review of the subject, with bibliography and additional research in the pathology and bacteriology of the disease, see White and Robey, Jour. Med. Research, 1902, vii, p. 255.

The lesions are usually discrete, but occasionally they are grouped, forming irregular lobulated elevations, and in rare instances tumor masses are produced.[1]

The lesions commonly develop slowly, requiring several weeks to attain the size of a pea, after which they may remain for an indefinite period unless removed spontaneously, through irritation and infection, or otherwise. The increase in number is usually by gradual multiplication of lesions, but occasionally the whole process is rapid and large numbers of ordinary sized lesions develop in a short time. The face, arms and genital region are sites of predilection, though the neck, trunk and other regions are not infrequently involved.

The disorder has been observed by the author much more frequently recently than formerly, and often in adults in whom the lesions were at times developed in large numbers and occurred chiefly on the trunk, the upper and lower extremities.

Etiology.—The disease occurs more frequently in children. It occasionally occurs in epidemics in schools or institutions where young people are confined.[2]

The disorder is more frequent in England[3] than on the Continent of Europe. The contagiousness of the disorder was early established through experimental inoculation. Retzius,[4] Vidal,[5] Pick,[6] and others produced the disease by inoculation of contents of molluscous tumors. Stellwagon[7] collected and classified reports concerning inoculation experiments which seemed to leave no doubt as to the parasitic nature of the disease. Ehrmann[8] suggested the possibility of the virus being transferred from one person to another by *Pediculus pubis*. Fox[9] cites seven cases in which operation wounds were infected with molluscum contagiosum. Examples of infection in Turkish baths are recorded by Crocker,[10] Morris[11] and Little.[12] Sweating and itching may induce dissemination of the lesions in affected individuals.

Although undoubtedly of parasitic origin, no parasite has been reported which has been accepted as a cause of the disorder.

Recently important experimental work has been done by Wile and Kingery[13] who successfully reproduced the disease by intracutaneous injections of the sterile filtrate of typical lesions. They concluded that the disease is due to a filtrable virus.

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[1] Smith, Dublin Jour. Med. Sci., 1878, lxvi, p. 371; Molluscum sebaceum (Giant molluscum). Larche, abstr., Jour. Cut. Dis., 1885, iii, p. 64. Fox, T. C., Brit. Jour. Derm., 1902, xiv, p. 216; and many others.

[2] Knowles, Jour. Amer. Med. Assoc., 1909, vol. liii, p. 671 (report of 59 cases in one institution); Hartzell, Medical Record, 1912, lxxxi, p. 1171; An epidemic of epithelioma (molluscum contagiosum), with some new observations concerning the molluscum bodies.

[3] For discussion of distribution and frequency of occurrence see Little, Brit. Jour. Derm., 1910, xxii, p. 180; Norman Walker, ibid., p. 284 and MacLeod, 1915, xxvii, p. 12.

[4] Deutsch. Klinik, 1872, p. 39.

[5] Progrès méd., 1878, p. 478.

[6] Monatshefte, 1892, xv, p. 133.

[7] Jour. Cut. Dis., 1895, xiii, p. 50.

[8] Zweiter Internat. Derm. Cong., Wien., 1892, p. 284.

[9] Westminster Hosp. Rep., 1909, xvi; abstr., Brit. Jour. Derm., 1909, xxi, p. 400

[10] Diseases of the Skin, 3d Ed., p. 732.

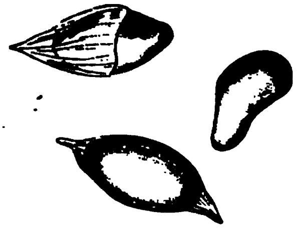
[11] Ibid., 5th Ed., p. 677.

[12] Brit. Jour. Derm., 1910, xxii, p. 180.

[13] Jour. Cut. Dis., 1919, xxxvii, p. 431; The etiology of molluscum contagiosum. Preliminary report of experimental study. Literature.

Pathology.[1]—The lesion of molluscum contagiosum is a benign epithelial new growth. It begins by proliferation of the cells of the rete mucosum in one or several areas, which causes a downgrowth of the epidermis into the corium, in the form of blunt, lobulated projections, producing a well-defined globular tumor, surrounded except at the surface by a layer of fibrous tissue. As the growth progresses downward the papillæ become flattened and disappear. A cavity begins at the surface, usually near the center of the growth, and extends downward with branches extending into each of the lobules composing the tumor. When fully developed the tumor is seen to be composed of wedge-shaped lobules, separated by fine connective tissue partitions, with the narrow end directed toward the central cavity. The various layers of the epidermis may be made out in the lobules, but many of the cells are altered through degeneration or other processes inducing characteristic changes. The cells of the basal layer show little change, but beginning in the second or third row above changes begin which continue to increase until the stratum corneum is reached, which here is represented in the central lining of the cavity. The granular layer is well formed, as a rule, and presents large granules of keratohyalin and the stratum lucidum can usually be differentiated.

Fig. 209



Molluscum corpuscles.
(After Kaposi.)

The degenerative changes occurring in the cells produces the "molluscum corpuscles." These cells are usually oval and have a double contoured wall, and their protoplasm shows degenerative changes. It may be granular or reticulated or reduced to a homogenous mass. The nucleus may be eccentrically placed, fragmented or absent.

Lining the cavity these cells present a horny capsule and contain a homogenous mass of degenerated protoplasm which forms the curd-like material which may be squeezed out of the small tumors. These cells were once believed to be parasites. In addition to the cells above described, Hartzell[2] noted a previously undescribed cell in the study of an early lesion. This was smaller than the "molluscum corpuscle," had a double contoured wall, was completely oval, and contained a mass of fine fibrils and no nucleus.

The character of the degenerative process occurring in the molluscum bodies is also a much debated subject. Török and Tammasoli[3] definitely settled that they were not coccidia when they demonstrated the capsule to be composed of keratin. Most observers believe the degeneration to be a peculiar form of colloid or hyalin degeneration. White,[4]

[1] Kingery, Archiv of Derm. and Syph., 1920, ii, p. 144. The histogenesis of molluscum contagiosum.

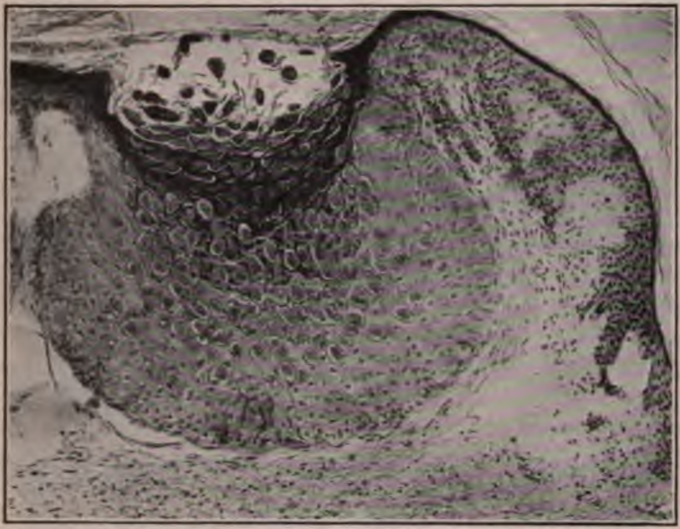
[2] Loc. cit.

[3] Monatshefte, 1890, x, p. 149; quoted by MacLeod.

[4] Loc. cit.

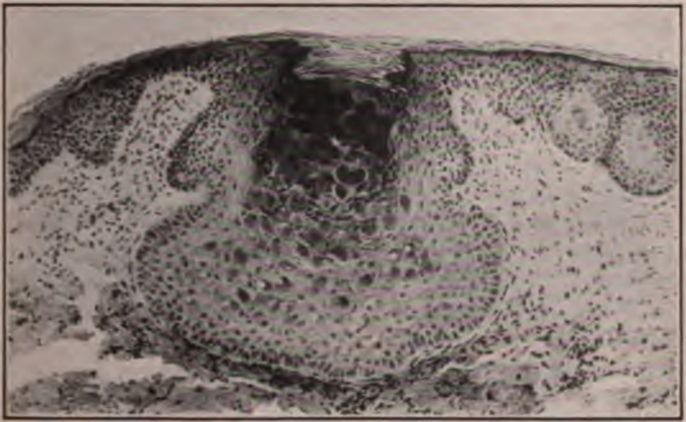
after an extended study, concluded that rather than a colloid or hyalin degeneration the molluscum bodies represent an extraordinary metamorphosis of rete cells into keratin.

FIG. 210



Photomicrograph of a section of molluscum contagiosum. (Wile and Kingery.)

FIG. 211



Photomicrograph of a section of molluscum contagiosum produced experimentally by inoculation of the material from the lesion shown above. (Wile and Kingery.)

Diagnosis.—The disorder is readily recognized when occurring in typical development. The small, pearly, semitranslucent, ovoid tumors presenting a dark central depression are characteristic. Ordinary verrucæ might on a superficial examination be mistaken, but the evident hyperkeratotic, irregular surface of the latter, readily distinguish them. The rare, large tumor masses in the absence of associated typical lesions, might require a microscopic examination to determine their nature. In occasional instances patients mistake these lesions when situated upon the genitalia for some form of venereal infection, but this is soon corrected when examined by a physician.

Treatment.—An efficient method of treatment consists in making multiple punctures in each lesion with a clean needle. Slight hemorrhage occurs. After several days the lesions disappear. When the tumors are few in number they may be removed by pressing out the contents through the central orifice. In some instances this slight operation is facilitated and rendered less painful by first making a linear incision over the growth. In children and others sensitive to the pain, the surface may be rendered anesthetic by the use of ice or ethylchlorid spray. Bleeding is arrested easily with a pledget of lint. Occasionally, after removal of the contents, the point of a crayon of silver nitrate may be introduced either to check hemorrhage or insure destruction of the cyst, or phenol may be introduced on the end of a pointed stick.

When the lesions are numerous, they may be made to exfoliate and disappear by the local application of green soap. Stelwagon recommended in such cases the use of an ointment containing 20 to 40 grains (1.3 to 2.6) of white precipitate or of sulphur to 1 ounce (30.), the ointment being rubbed vigorously into the affected parts once or twice a day. Davis[1] regards trichloracetic acid as most efficient.

XANTHOMA.

Synonyms. Xanthelasma, Vitiligoidea. Fr., *Plaques jaunâtres des paupières.*

This disease was first described by Rayer,[2] under the French title given above; by Addison and Gull[3] as *vitiligoidea*; by Erasmus Wilson as *xanthelasma*; and by W. F. Smith, in 1869, as *xanthoma*, the name now generally accepted by writers. Through the work of modern investigators, clear distinctions have been made between the many varieties described, and in this text they will be treated under separate titles as distinct disorders, which the findings appear to justify and which the consensus of opinion upholds: first, xanthoma, a neoplastic disorder; second, xanthelasma (eyelid cases), a degenerative process; third, xanthoma diabeticorum, a form associated with glycosuria. These disorders are related chiefly in the common occurrence of a fatty substance in the lesions.

Xanthoma (*Xanthoma multiplex*).—Xanthoma is characterized by yellowish lesions, occurring as nodules, plaques, or striæ, situated chiefly about the large joints (elbows, knees, hips), unaccompanied by subjective sensations, and composed of fatty and fibrous elements.

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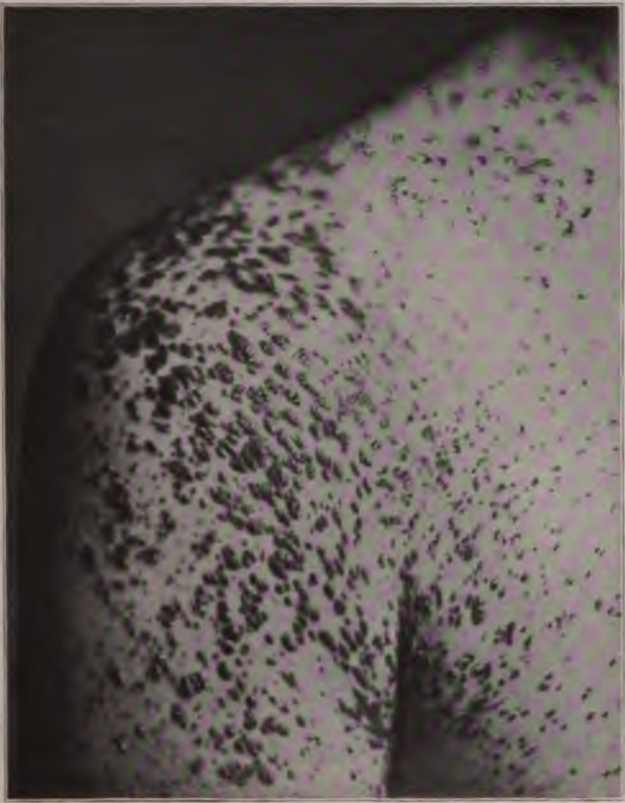
[1] Jour. Cut. Dis., 1910, xxviii, p. 303.

[2] Traité prat. des Maladies de la Peau, Paris, 1836.

[3] Guy's Hosp. Reports, 1851, 2d Series, vii, p. 268.

Symptoms.—The lesions occur as papules, tubercles, nodules, tumors, infiltrated plaques, striæ, or non-elevated, smooth, plane areas. The lesions of all the types are yellow or yellowish-red in color. They are usually grouped about the large joints (elbows, knees, and hips), but occur also over the small joints of the hands, the palmar and plantar surfaces, scattered irregularly over the trunk, and rarely, even in severe cases, upon the face. In certain cases, papules, nodules, plaques, and tumors may coexist along with plane lesions; and in these the greatest development will be seen about the joints, while upon the palms and soles striated lesions are commonly found. In rare instances, by enlargement and coalescence, the tubercles or nodules may form sessile or pedunculated, hazel-nut- to hen's-egg-sized tumors, which, as a rule, are firmer than the smaller lesions.[1] The

Fig. 212



Xanthoma.

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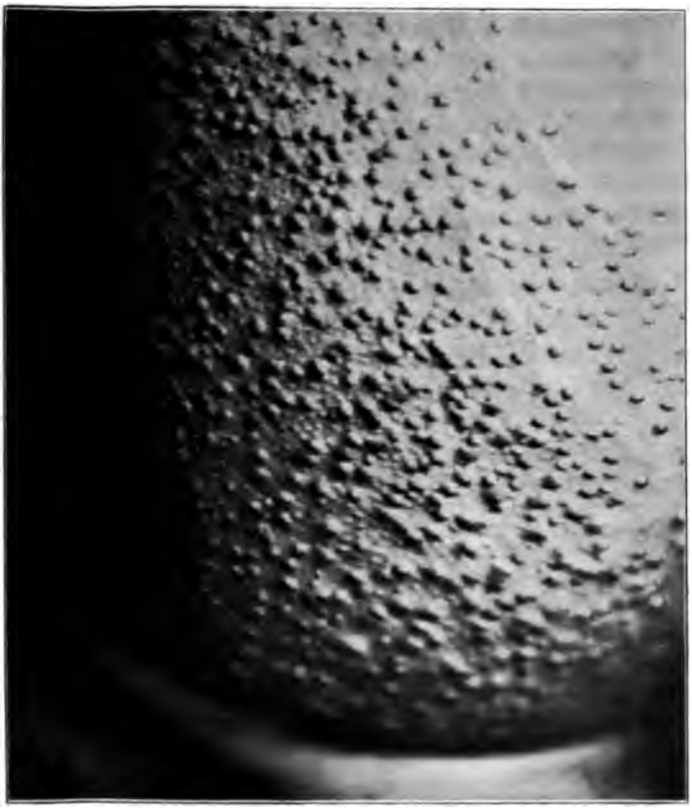
[1] Carry, Annales, 1880, s. ii, i, p. 75; Chambard, Arch. de Phys. norm. et path., 1879, s. ii, vi, p. 330.

surface of the plaques or tumors may be irregularly furrowed or lobulated or become mushroom-like in shape. Not infrequently extensive cases occur in children.[1] In these the eruption is apt to be generalized, the lesions being papules or flat tubercles of varying shapes. Winfield[2] describes the development of a lesion of this type as follows: "First, it appeared as a pea-sized, brownish-colored macule, but one month later a pinhead-sized papule, which was hard, smooth, glistening, and pinkish-red, developed upon the site of the macule. It gradually enlarged, became darker in color and less firm in consistency, and when it had reached its full development (in about six weeks) was about the size of a large pea, soft and flabby in consistency, so that it could be picked up and rolled between the fingers. It was no longer glisten-

Fig. 213



Xanthoma.

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[1] Winfield, Jour. Cut. Dis., 1909, xxvii, p. 112; Abrahams, ibid, 1909, xxvii, p. 224; Fischkin, ibid., 1910, xxviii, p. 419.

[2] Loc. cit.

ing, but dull and furrowed, and the color ranged from a bright lemon-yellow to orange and saffron."

A remarkable case was recorded by MacLeod,[1] in which extensive xanthomatous sheets occurred in association with tumors about the joints. In this as well as in other extensive cases lesions were found on the eyelids. (It will be of interest to determine by a histological examination whether the eyelid lesions in such cases belong to the degenerative or the neoplastic type.)

On the palms and soles more or less extensive discoloration occurs. The lesions occurring in these locations are striking in certain cases. They occur as yellowish discolorations in lines in the normal creases of these regions, at times being sufficiently wide to give the appearance of narrow ribbons. While not elevated, they stand out in bold relief, on account of their peculiar coloration. In certain cases, the disease is accompanied by generalized coloration of the skin, of a peculiar yellowish shade, which has been variously interpreted as a xanthomatous dischromia and as a true icterus. (A woman presenting one of the extreme phases of this icteroid xanthomatous condition of the skin was shown at the International Dermatological Congress in London, in 1896.) Other organs of the body may be affected. Nodules may appear on the conjunctiva and cornea, in the mouth, and on the palate. In Rhode's case,[2] nodules developed in the trachea, causing obstruction to such a degree that tracheotomy became necessary, and the patient wore a tracheotomy-tube continuously. Nodules may also form in the bronchi, in the esophagus, the gastro-intestinal tract, on the peritoneum, pericardium, spleen, and large arteries.[3] Lieberthal exhibited a young patient before the Chicago Dermatological Association with multiple lesions of xanthoma, in whom the sheath of the tendo Achillis was involved.

In rare instances, the disease may be exhibited as a single lesion, the so-called *Xanthoma solitarium*, and this may be situated in any of the locations commonly selected by the disorder.

The *Xanthoma planum* of writers indicates the same process in lesions that are distinguished by being imbedded in the skin rather than elevated above it.

Etiology.—The causes of xanthoma are obscure. In a few cases, the lesions have been observed first in early childhood, though they are encountered chiefly in adults. Women are affected more often than men. Many instances are on record in which several members of a family were affected. Török and T. C. Fox have each recorded families in which members of three generations presented the disease. The association of xanthoma with diseases of the liver, rheumatism, gout, ovarian disease, migraine, syphilis, carcinoma, hydatids, and other disorders cannot be denied, but it frequently occurs without such association. In rare instances, the disease is congenital, xanthomatous tumors being present at birth. McDonough[1] described a congenital xanthoma of endothelial origin. The lesions were lentil-sized; and at first were red in color, later becoming yellow. Some sort of hepatic disease has been described in about 50 per cent. of the recorded cases. Little[2] described a patient with xanthoma who was suffering with chronic cirrhosis of the liver. In a recent study Burns[3] found an increased amount of cholesterol in the blood of a patient the subject of xanthoma multiplex and concludes that "it seems probable that xanthoma multiplex is a dermatosis whose origin lies in the increased amount of cholesterol in the blood, the skin lesions of which are caused by its deposition in this tissue where it induces a peculiar secondary connective tissue hyperplasia."

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[1] Brit. Jour. Derm., 1913, xxv, p. 344.

[2] Laryngoscope, October, 1906.

[3] Cranston Low, Brit. Jour. Derm., 1910, xxii, p. 109: Xanthoma Tuberosum Multiplex, with Lesions in the Heart and Tendon Sheaths.

Pathology.—The anatomy of xanthoma has been investigated especially by Chambard, Balzer, Touton, Török, Pollitzer, Whitehouse,[4] Johnston, and others. The structure as described by all is similar, except as this changes at varying periods of the disease. In the interpretation of the findings wide divergence of opinion has been expressed. The chief changes noted are the presence of a large amount of fat contained in special cells, termed "xanthoma-cells," and in the increased connective-tissue growth, particularly in the later stages. The process by most authors is considered neoplastic rather than inflammatory. In a recent study made by Pollitzer and Wile,[5] the authors state that there occurs in the earliest lesions proliferation of the cells in the immediate neighborhood of the papillary and subpapillary vessels, together with an infiltration of a fatty substance into the perivascular and intercellular lymph-spaces. This is present also in the new-formed cells and in the endothelium of the capillaries and here and there in the basal layer of the epidermis. These new-formed cells are regarded by the authors as small, young xanthoma-cells, which in a lesion of larger size were found to contain two or more nuclei, and in certain instances a number, in which case they are considered xanthoma giant-cells. With proper stains, these cells were found to be almost entirely filled with a fatty substance, upon the removal of which the cells were seen to have a reticulated appearance. In the connective tissue surrounding these nests of xanthoma-cells, fibroblasts were present in large numbers. These also were seen to be filled with fat, in fine droplets and coarse clumps, upon either side of the nucleus, tapering out to fill the fusiform contour of the cell. In sections from older specimens, the xanthoma-cells had been reduced to a small number in proportion to the fibrous elements. Concerning the pathogenesis of this process, these authors conclude that xanthoma multiplex represents an irritative connective-tissue hyperplasia, in which extravasation of cholesterol-fatty-acid-ester present in excess in the blood serves as the stimulus. The occurrence of the above-mentioned lipoid in the blood in icterus and jaundice, together with its presence in great quantity in the tumors in xanthoma multiplex, affords, the authors believe, an explanation of the relation existing between these systemic disturbances and the cutaneous lesions. MacLeod,[1] in a histological study of his extensive case, confirms the findings of Pollitzer and Wile.

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[1] Brit. Jour. Derm., 1911, xxiii, p. 115.

[2] Ibid., 1909, xxi, p. 27.

[3] Archiv of Derm. and Syph., 1920, ii, p. 415. A contribution to the study of the etiology of xanthoma multiplex.

[4] Jour. Cut. Dis., 1904, xxii, p. 470.

[5] Ibid., 1912, xxx, p. 235.

Török concludes that the ordinary xanthoma-cell is formed of heterotopic adipose tissue, and that it is constituted, by reason of that heterotopia, of fat-cells of incomplete, interrupted evolution. Other observers look upon the process as being primarily an inflammation, which is followed by fatty degeneration of the cells.[2]

The histology of the palmar striæ was studied by Johnston in the case of Whitehouse,[3] and corresponds in most particulars to that of the ordinary multiplex variety described above; the peculiarity of the process lying only in the fact that so small an amount of fatty deposit could produce so pronounced a coloration as was evident clinically.

Diagnosis.—The diagnosis from all other lesions is readily made when the peculiar yellowish or saffron-like changes of xanthoma, and the common situation, form, and general characteristics of its various lesions are considered. The difficulty lies chiefly in distinguishing the cases occurring in infants from urticaria pigmentosa with xanthoma-like lesions. A number of errors are recorded in this connection. As a rule, the cases of urticaria pigmentosa are mistakenly called xanthoma. The reverse of this, however, occasionally happens, as was seen in the case of Winfield.[4] In urticaria pigmentosa the presence of dermographism, possible itching, pigmented areas, and, most important, the swelling, reddening and enlargement of the lesion when irritated, are important features. Finally, microscopically, the presence of a mast-cell tumor in the one and fatty masses in the other settles the diagnosis. Involution of the lesions of xanthoma is said to occur rarely. The major portion develop to a certain degree and then remain stationary throughout life. The chief concern is, first, the appearance of the lesions, and, next, the possibility of large tumors causing inconvenience.

Treatment.—Surgical procedure is indicated for the removal of large tumors. Other measures suggested in the treatment of xanthelasma may be employed in appropriate cases.

Xanthelasma (*Eyelid xanthoma*). — The term *xanthelasma* was adopted for this variety of xanthoma by the American Dermatological Association at the thirty-eighth annual meeting, in 1914. Pollitzer[5] had previously stated that xanthelasma would be an appropriate term for the eyelid form of the disorder. The disease is characterized by the occurrence in patients at or past middle age of one or several pinhead- to split-pea- or larger sized, yellowish, oval, soft lesions on the eyelids, usually near the canthi, with a tendency to remain permanently.

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[1] Loc. cit.

[2] Discussion of this question and a *résumé* of the literature, Brit. Jour. Derm., 1892, iv, p. 237.

[3] Loc. cit.

[4] Loc. cit.

[5] Jour. Cut. Dis., 1910, xxviii, p. 633.

Symptoms.—Xanthelasma occurs in the major portion of cases on the skin of the eyelids, rarely on other parts of the face or neck. The lesions are pinhead- to pea-sized or larger, chrome-yellow in color, very slightly raised above the adjacent surface, and are of the same consistency as the neighboring tissue. They occur most frequently on the upper lid, at the inner canthus, in the form of a small patch, which may or may not be followed by several others near the outer canthus and on both lids of both eyes. The patches extend slowly, and, according to Pollitzer, in the direction of the long fibers of the orbicularis muscle. Occasionally, the chamois-leather-like areas may affect the entire upper lid, or the lower lid, or encircle the eye. In rare instances, these lesions develop on the nose, the cheeks, the ears, and the nucha. In one instance, Pollitzer saw a similar lesion over a large area on the outer anterior aspect of the neck below the jaw. The lesions develop very slowly and in time become stationary, and when once established do not disappear. They are rarely productive of subjective sensations, but are occasionally the seat of slight itching. As a rule, this variety develops at or after middle life.

Etiology.—Xanthelasma practically always occurs in people past middle life, and, as has been proven by Pollitzer, is a degenerative process; otherwise, no etiological factor can be suggested. In one case lesions of this type developed on the eyelid following an accidental contact with corrosive-sublimate solution (Hyde).

Pathology.—The process in xanthelasma is quite different from that described in xanthoma. Pollitzer[1] first called attention to the fact that this was a degenerative process, and not neoplastic. In a subsequent article,[2] this author confirms his previous work, and his findings are briefly as follows: In xanthelasma the greater part of the cutis is literally filled with peculiar cell-like masses and bodies, known as xanthoma-cells. They are fragmented and degenerated remains of muscle-fibers with proliferated sarcolemma and nuclei. The xanthelasma cells resemble the cells of sebaceous glands, showing a reticulated structure filled with a granulo-fatty mass, with one or more round or oval, deeply staining nuclei. The fat, however, does not form a single large drop, which is common in the cells of sebaceous glands. In the larger cells there is frequently little structure, the cell being represented by the homogeneous granulo-fatty mass, within which there may be one or several nuclei lying without any definite arrangement. The various forms of these cells and their development from the muscle are well described by Pollitzer in this article. There also occurs a certain amount of yellowish pigment in various situations. The same author from his studies concludes that xanthelasma is in no way related to xanthoma multiplex, that it is the product of a

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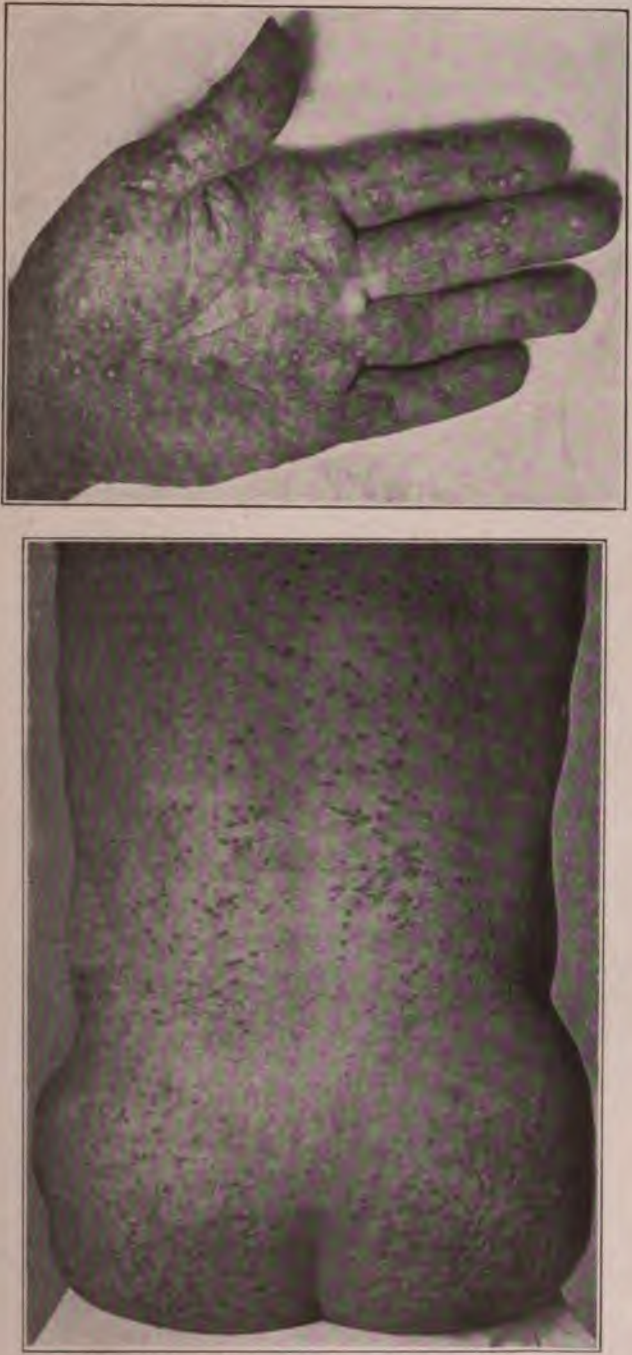
[1] New York Med. Jour., 1899, ii, p. 73.

[2] Jour. Cut. Dis., 1910, xxviii, p. 633.



Xanthoma Diabeticorum.

peculiar fatty degeneration of muscle-fibers of the eyelids, and that it belongs not to the neoplasms but to the degenerations.

From his investigations of the disorder Pollitzer concludes that the myogenetic origin of xanthelasma explains (1) the absence of any clinical signs of tumors; (2) the practically exclusive occurrence in the eyelids, where peculiar muscular conditions exist; (3) its occurrence in symmetrical, discrete patches at the canthi of the lids; (4) its arrangement in elongated plaques, whose long axes are parallel to the course of the orbicularis fibers; (5) its usual development after middle age, when degenerative processes are apt to occur; (6) the peculiar yellow pigment, an invariable accompaniment of muscles undergoing fatty degeneration; (7) the disappearance in the affected area of the greater portion of the muscular tissue normally present in the eyelids; and (8) the microscopical structure of the tissue itself.

Diagnosis.—The presence of soft, yellowish lesions, limited to the eyelid region, occurring in adults in middle life, can scarcely be confused with any other disorder.

Treatment.—Erasion and excision are the usual methods of removing xanthelasma. Care should be taken in such operations to avoid a consequent ectropion, when the operation is performed upon the skin of the eyelids. The Paquelin knife is objectionable on account of the radiation of heat to the globe of the eye. With the tumor slipped through an aperture in a thin sheet of asbestos paper, such as is now found on the market, this danger may be obviated. Morrow employs 25 per cent. salicylic-acid plaster. Roberts makes a salicylated collodion paint—2 parts of salicylic acid, 1 each of chrysarobin and castor-oil, and 4 of flexible collodion.

The modern method of treatment by electrolysis, however, is preferable to others. The technique is the same as that employed for hypertrichosis and for the removal of soft moles. Besnier employs phosphorus internally, followed by turpentine, by which the course of the disease is said to have been relieved. Wilson, with the same end in view, employed nitro-hydrochloric acid, bitters, and blue pill. McGuire reports the removal of lesions by applications of monochloracetic acid. Liquid air has been used with success by Dade.[1] Carbon-dioxid snow is of questionable value, but may be used.

Prognosis.—In xanthelasma the lesions without treatment remain permanently.

Xanthoma Diabeticorum.—Xanthoma diabeticorum is a rare cutaneous disease occurring, as a rule, in the subjects of glycosuria, characterized by the development on the skin of multiple whitish or yellowish globoid papules, with a reddish base, resembling pustules, and accompanied by mild subjective symptoms. Dilated capillaries are often noted over the papules, giving them a reddened appearance.

The disease was first described by Addison and Gull, in 1851, but was not separated from other forms until 1883, when Morris,[2] describing the fourth case, clearly differentiated it from other cutaneous affections.

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[1] Jour. Cut. Dis., 1909, xxvii, p. 311.

[2] Pathological Transactions, London, 1883.

Symptoms.—The lesions are usually multiple and exceedingly numerous, discrete or confluent, and not rarely grouped, pinhead- to pea-sized, firm, well-defined, conical or acuminate papules. At the apex may be recognized a yellowish center with reddish areola, which may be made to disappear temporarily under pressure. The appearance, when viewed at some distance, is suggestive of a pustule. The lesions occur particularly over the buttocks, loins, elbows, knees, and extensor faces of the limbs in general, the scalp, face (brows, lips, nose), about the angles and over the mucous surface of the mouth, and the palms and soles. The eruption occurs suddenly, and the lesions are abundant at the outset. They may be firm, but are generally soft and compressible to the touch. Occasionally, they occur as punctate, linear, ribbon-shaped, or flattened lesions. Upon their involution, no trace of their existence is left. They may persist from several months to several years, or disappear comparatively soon. Under appropriate treatment, they undergo involution with surprising rapidity.

Etiology.—In the major portion of cases recorded, glycosuria has been recognized. This condition may follow the appearance of the lesions, or may precede it, the latter being the rule. Johnston calls attention to the fact that in nearly every case the patient has been described as stout, florid, or obese. The major portion of the patients have been male subjects and usually in a condition of fair nutrition. In other cases, especially in young subjects, there is malnutrition, and even cachexia. In yet other subjects, albuminuria, nephritis, and jaundice have been noted.

Pathology.—This has been studied by Morris and Clark,[1] Crocker,[2] Török,[3] Schamberg,[4] Johnston,[5] Robinson, and others. Histologically, there is some resemblance between this disorder and xanthoma multiplex. The process is an inflammatory one rather than neoplastic. The xanthoma-cells, which are typically present, are fewer in number, and in the later stages there is less connective-tissue formation. Török states that the lesions of xanthoma diabeticorum are caused by a local irritative process, resulting in a granulo-fatty degeneration. This view is confirmed by Johnston, whose findings may be summed up by the statement that the process in the diabetic variety is inflammatory, resulting in the degeneration *en masse* of the central portion of the papule. The xanthoma-cells, he states, are in reality giant-cells containing minute droplets of oil. From these findings, he believes there is no reason to class the two varieties together. In a later study, Johnston[6] states that the nodule of diabetic xanthoma begins and remains localized throughout its whole course in the corium, appearing first as a sheath about the bloodvessels of the reticular portion and extending along them to the papillæ and subcutaneous tissue. These cellular sheaths consist of mononuclear cells of varied origin, and morphologically exhibited as lymphocytes, plasma-cells, fibroblasts, and endothelial cells. The latter are the so-called xanthoma-cells, from which giant-cells are produced by fusion or nuclear division. It is these cells which have undergone the fatty degeneration seen in this disorder. The connective-tissue cells become edematous and in some areas undergo hyalin degeneration; in other places they are replaced by free fat. The elastic tissue shows no changes. In conclusion, the author states that the process is an exudative inflammation, in which fatty change occurs, free fat infiltrating the tissues.

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[1] Brit. Jour. Derm., 1892, iv, p. 237 (report of new case and review of the twelve in literature).

[2] Ibid., p. 253 (new case, with histology).

[3] Quoted by Johnston.

[4] Jour. Cut. Dis., 1895, xiii, p. 202.

[5] Ibid., p. 401.

[6] Ibid., 1900, xviii, p. 387.

Diagnosis.—The important points in diagnosis in xanthoma diabeticorum may be summed up as follows: the sudden evolution and involution of the lesions; the presence of firm and solid, inflammatory, discrete or confluent papules resembling pustules; the yellowish color at the top of the papule, which may not be visible early; moderate subjective symptoms; the presence of glycosuria, as a rule; and the occurrence of the lesions, in addition to the regions mentioned, in the neighborhood of hair-follicles and sebaceous glands. Xanthoma multiplex differs from this chiefly in its slow development and the permanency of the lesions. The lesions of the latter are usually soft, and frequently striæ and patches occur. The yellow coloration is marked and distinctive. The subjective symptoms are absent. Jaundice may be present and glycosuria is not an accompaniment. Histologically, one is an inflammatory process with degeneration, the other a neoplastic growth.

Treatment.—The treatment of the disease, medicinal and dietetic, is largely that of glycosuria. Robinson's patient recovered after the use of small doses of Fowler's solution. Local treatment may be employed as indicated in any case.

Prognosis.—The prognosis is favorable. The majority of patients eventually recover.

PSEUDO-XANTHOMA ELASTICUM.

Synonym.—Xanthoma elasticum.

This is among the rarest of cutaneous disorders, only a few cases having been recorded, the first by Balzer.[1] Other reports on the subject have been made by Besnier and Doyen,[2] Bodin,[3] Little,[4] and Dübendorfer.[5]

The lesions described in this disorder have been pinhead- to pea-sized, discrete, irregularly outlined papules, which may by fusion form patches of varying size up to that of the palm of the hand, meshlike or linear in outline. Their color is yellowish, somewhat resembling xanthoma. They occur on the abdomen, below the clavicles, near the axillæ, about the elbows, and occasionally on the thighs. The face, including the eyelids, the hands, and other areas selected by the xanthomata, have been free.

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[1] Arch. de Phys., 1884, s. iii, p. 65.

[2] Translation of Kaposi, ii, p. 336.

[3] Annales, October, 1900, p. 1073; abstr. Brit. Jour. Derm., 1901, xiii, p. 231.

[4] Brit. Jour. Derm., 1908, xx, p. 194 (Pseudo-xanthoma of Balzer. Case report

[5] Archiv, 1903, lxiv, p. 175; abst. Brit. Jour. Derm., 1903, xv, p. 223 (Pseudo-xanthoma Elasticum and Colloid Degeneration in Scars).

In a histological study of the disorder it has been found to be a degenerative process affecting the elastin. A peculiar mononuclear giant-cell was observed by Bodin, and also hyalin degeneration. Balzer described the elastic tissue as occurring in large coils, chiefly around the follicles, forming the larger part of the tumor. The fibers were swollen, degenerated, and in parts broken and segmented.

The disease is distinguished from xanthoma clinically by the character of the lesions, which occur in areas not usually selected by xanthoma; and histologically by the absence of the peculiar xanthoma-cells and other evidence of fatty degeneration.

COLLOID DEGENERATION OF THE SKIN.[1]

Synonyms.—Colloid milium (Wagner); Hyaloma. Fr., *Colloïdome miliaire* (Besnier); Ger., *Hyalom der Haut.*

This disorder is among the rarer cutaneous diseases. It was first described by Wagner, in 1866, under the title of *Colloid milium.* According to Hartzell, twenty-two cases are on record. It is a disorder characterized by lemon-yellow colored elevations of the skin, occurring usually upon the face.

Symptoms.—The lesions consist of pinhead- to millet-seed- or split-pea-sized, sharply circumscribed, irregularly rounded, flattish elevations of the skin, lemon-yellow in color, having a peculiar glistening, translucent appearance, suggestive of vesicles. They project but slightly from the skin, and on puncture give exit to a soft, gelatinous mass, at times accompanied by a droplet of blood. Some of them may be surrounded by very slight telangiectases. They develop slowly, often in groups, the individual papules remaining distinct even when two or more unite. Frequently, a papule becomes depressed in the center; or becomes inflamed and covered with a crust, which falls and leaves a shallow depression, but not a true scar. The lesions, as a rule, occur on the upper two-thirds of the face, especially on the forehead and about the orbits. In C. J. White's[2] case the backs of the hands also were involved. In Hartzell's case the lesions extended over both malar eminences and across the nose, and there was some discoloration about the lower lip at the junction of the skin and the mucous membrane. In one of two cases recorded by Johnson and Paul[3] lesions were present on the neck and forearms as well as the hands. There are no subjective symptoms.

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[1] For full bibliography, *cf.* Hartzell, Jour. Cut. Dis., 1914, xxxii, p. 683: Report of a case of So-called Colloid Degeneration of the Skin.

[2] Jour. Cut. Dis., 1902, xx, p. 49 (with review of the literature).

[3] Med. Jour. of Australia, 1916, i, p. 304; abstr., Jour. Cut. Dis., 1916, xxxiv, p. 870.

Etiology.—The cause of the disease is not known. It occurs alike in men and women, usually after the forty-fifth year of age. A male patient aged twenty-five was seen at the clinic by Dr. Hyde.

Pathology.—The pathology has been studied by a number of observers.[1] The chief and characteristic change found is the presence of colloid degeneration to a greater or lesser extent in the corium. This is seen to affect the connective tissue and elastin, which may become involved over considerable areas. The changes are especially noticeable about the vessels and nerves and about the sebaceous and coil-glands. The glands themselves and all the epithelial structures except the endothelia of vessels (Balzer) usually escape. In sections examined by Hyde and Montgomery in the case mentioned above, a few rete-cells and a few cells of the coil-gland ducts were transformed into or infiltrated with colloid substance. The major portion of observers report colloid degeneration as occurring only in the mesoblastic structures. Hartzell summarizes the changes in his case as follows: (1) a marked thinning of the epidermis, the disappearance of a considerable number of the basal-cell layers, and colloid degeneration of the prickle-cells in various places; (2) complete disappearance of the papillary and subpapillary layers of the corium and their replacement by colloid material; (3) varying degrees of degeneration and destruction of the elastic tissue. He further states that the collagen and elastin are affected in an equal degree, and that his case demonstrated the fact that the cells of the epidermis share in the degeneration.

Diagnosis.—The disease may be confused with xanthoma, hydrocystoma, adenoma sebaceum, and multiple benign cystic epithelioma. A careful study of the characteristics of these various disorders, however, will in the major portion of cases be sufficient to make the diagnosis. In case of doubt, a histological examination should be made.

Treatment.—The nodules may be removed by surgical measures with a curette or by electrolysis.

CALCIFICATION OF THE SKIN.

This unusual condition was described first by Thimm[2] as occurring in the case of a male patient, aged twenty-three years. There was a hard, moderately elevated tumor of eight years' duration, one cm. in diameter, situated on the dorsal aspect of the proximal phalanx of the left little finger. The lesion was described as yellowish-white in color, having a warty center, and formed primarily by the coalescence of hemp-seed-sized, whitish nodules.

The microscopical examination revealed masses of calcareous material in the situations of the pilo-sebaceous follicles, and also between the fibrous bundles of the corium. The fibrous elements formed in areas a framework for the chalky deposits. There was, in addition, the usual picture indicative of a chronic inflammatory process. The writer's conclusion was that, as the result of a sebaceous-gland change, retention cysts formed, which later underwent calcareous degeneration.

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[1] For a discussion of the findings by the various writers and a consideration of the proper placement of these various cases, *Cf.* Hartzell, loc. cit.

[2] On Calcification of the Skin, Archiv, 1902, lxii, p. 163; abstr. Brit. Jour. Derm., 1903, xv, p. 223.

In a case reported by Reines,[1] the rim of the right ear was involved. Histologically, the entire thickness of the cutis was occupied by a deposition of calcareous masses in a net-like granulation-tissue. Giant-cells were present in abundance. The pathogenesis of the condition he explains by assuming a degenerative change in the tissue preceding the calcification, which change was induced by some form of chronic irritation (pressure during sleep) acting upon a part having low resisting powers.

Calcareous degeneration is described by Gilchrist[2] and Stokes in peculiar bodies found in the lupus-like tissue. Calcareous deposits have also been noted in connection with milia. In addition, we have seen similar deposits about the elbows and other situations in scleroderma. Scholefield and Weber[3] report a similar instance.

ADENOMA SEBACEUM.

Synonyms.—Adenoma of the sebaceous glands.[4] Fr., *Adénomes sébacés* (Balzer and Menetrier), *Adénomes sébacés cancroïdaux; Acné cancroïdale.*

Definition.—This rare disease was first described by Balzer, but no importance was attached to the subject until Pringle[5] described a case clinically, with a report of his histological study, and collected from the literature five similar cases. The disease is characterized by the occurrence (usually on the face, but also occasionally on other parts of the body) of small papules, telangiectasia, and occasional angiomata, associated with other nevoid conditions.

Symptoms.—The disease commonly begins in early childhood, although many cases are described in which it began at a much later date. The lesions are pinhead- to bean-sized papules or nodules, varying in color from yellowish-white to deep brownish-red. Many lesions are the color of the normal skin. Often their surface is vascularized by the presence of minute capillaries. They are usually discrete, but confluence sometimes occurs. They are situated chiefly on the face, over the cheeks, forehead, and particularly in the furrows beside the nose. In the majority of cases, other defects in the skin, such as warts, nevi, small papillomata, and pigment spots, are present; and many patients reported have been mentally deficient or epileptic. In a number of the earlier cases recorded, congenital anomalies were noted, and in addition a low state of mentality has also been observed.

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[1] Archiv, 1907, lxxxviii, p. 267 (with *résumé* of the reported cases).

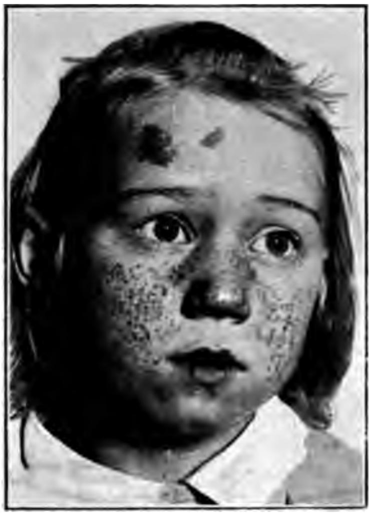
[2] Jour. Cut. Dis., 1903, xxi, p. 463: The Presence of Peculiar Calcified Bodies in Lupus-like Tissue.

[3] Brit. Jour. Derm., 1912, xxiv, p. 200.

[4] Darier, La Pratique Derm., tome i, p. 284. Jackson, Jour. Cut. Dis., 1909, xxvii, p. 307 (case demonstration). Hubbard, ibid., p. 410 (case demonstration). Gottheil, ibid., 1911, xxix, pp. 452 and 553 (case demonstration, mixed type). Sequeira, Brit. Jour. Derm., 1911, xxiii, p. 59 (case demonstration). Little, ibid., 1909, xxi, p. 327 (case demonstration, normal mentality). Colwell, ibid., 1909, xxi, p. 362 (case, with sections, demonstrated before the British Medical Association). Fordyce and MacKee, Jour. Cut. Dis., 1915; Adenoma sebaceum, case report. Lesions unilateral in distribution.

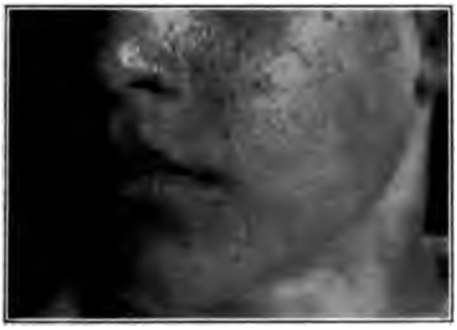
[5] Brit. Jour. Derm., 1890, ii, p. 1.

Fig. 214



Adenoma sebaceum. Three cases in one family. Aged seven years. Duration, two years.

Fig. 215

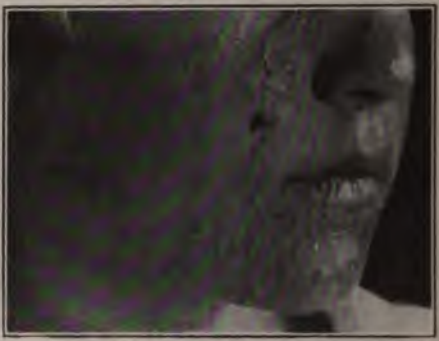


Adenoma sebaceum.

More recently, numbers of cases have been seen in people whose mentality was normal. The condition is said to be commonly found in asylums for the feeble-minded. Crocker states that a large proportion of the cases are chronic epileptics or imbeciles, and the disease

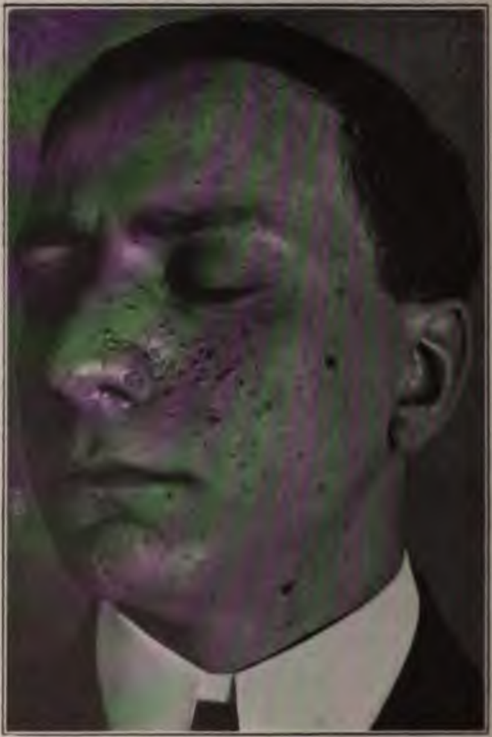
is not uncommon in idiot asylums. In a patient, a girl of fif
studied by the author, the disease had been present in typical d

FIG. 216



Adenoma sebaceum.

FIG. 217



Adenoma sebaceum. (Fred Wise.)

opment on the face for nine years. The intellectual developm
this patient was above the average.

The course of the disease is slow, gradual development occurring for several years until puberty is reached, after which it remains stationary or disappears slowly, leaving temporary atrophic spots. The rule, however, is for the disease to persist.

Etiology.—The lesions appear to be developmental defects of the skin, which may, however, appear for the first time many years after birth. Females are more frequently attacked than males. The condition may be associated with other sebaceous disorders. It may rarely show some sign of heredity. Taylor and Berendt[1] recorded 3 cases in one family, Adamson[2] recorded 2 cases in another and Shelmire[3] recorded 5 cases in one family. The author has had 2 family groups under observation.

Pathology.—The chief change found microscopically is an increase in the sebaceous-gland and vascular elements, this increase fluctuating in different cases. In Pringle's early studies he found an enormous increase in the number and complexity of the sebaceous glands, which suggested a resemblance to the condition found in rhinophyma. McDonagh[4] describes the corium as being filled with sebaceous-gland tissue, either fully formed or partly transformed from epithelial cells, from which the gland-cells take their origin. Sutton[5] found in some sections merely a circumscribed hypertrophy of the sebaceous glands; in others from a larger growth he found true tumor-formation.

Diagnosis.—The history of the disease, which begins in early life and develops gradually; the persistency and permanency of the individual lesions, situated chiefly on the middle of the face and especially in the naso-labial folds; the frequent occurrence of telangiectases with the papules above described; the frequent association of other nevoid growths; and the absence of suppuration or ulceration, will usually suffice for a diagnosis. In colloid milium the lesions are usually few in number, are situated chiefly on the frontal and orbital regions, have a peculiar yellowish, translucent appearance, and are not so much modified by telangiectases. In multiple benign cystic epithelioma the lesions occur on the forehead and also on the trunk. In certain cases a microscopical examination is necessary to settle the diagnosis.

Treatment.—Neither internal remedies nor external applications have any influence upon the lesions. The lesions may be removed by electrolysis, fulguration, curettage, cauterization, scarification or excision. Much improvement followed the use of *x*-rays in several cases treated by the author.

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[1] Brit. Jour. Derm., 1893, v, p. 360.

[2] Ibid., 1911, xxiii, p. 109.

[3] Trans. Sect. on Derm., Amer. Med. Assoc., 1918, p. 141, Adenoma Sebaceum; a report of five cases in one family.

[4] Brit. Jour. Derm., 1912, xxiv, p. 302.

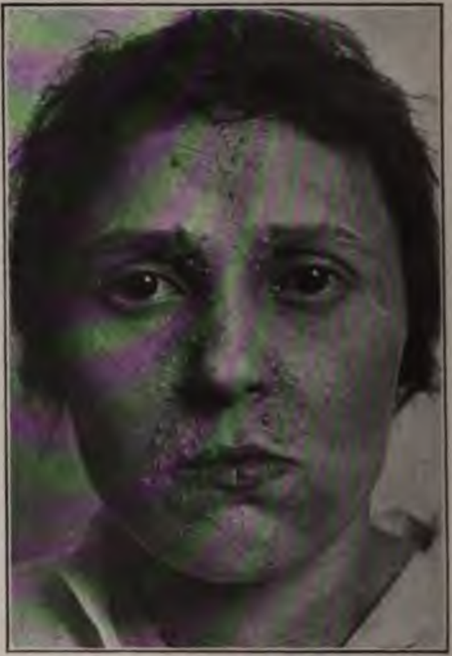
[5] Jour. Cut. Dis., 1911, xxix, p. 480 (A differential study of multiple benign cystic epithelioma and adenoma sebaceum in the negro).

MULTIPLE BENIGN CYSTIC EPITHELIOMA.[1]

Synonyms. — Trichoepithelioma papulosum multiplex (Epithelioma adenoides cysticum (Brooke); Acanthoma a cysticum (Unna). Adenoma of sweat glands (Perry).

This disorder begins, as a rule, at the age of puberty frequently hereditary,[2] several members of two generation sometimes affected. The lesions are conspicuous in their rence about the face. Brooke[3] and Fordyce[4] independentl

FIG. 218



Multiple benign cystic epithelioma.

attention to the origin of these growths being either from th layer of the epidermis or from similar cells of the hair-follicles.

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[1] LITERATURE (some references not given in the text): Perry, Atlas of Rare S 1890, Part 3, Plate IX. Fox, Brit. Jour. Derm., 1897, ix, p. 230 (case report) heil, Jour. Amer. Med. Assoc., 1901, xxxvii, p. 176. Hallopeau, Annales, 1890 p. 872. Heidingsfeld, Jour. Cut. Dis., 1908, xxvi, p. 18 (extensive bibliography gles, ibid., 1910, xxviii, p. 248 (Multiple benign cystic epithelioma and fibrom same patient. Histology by Wende). Sutton, ibid., 1911, xxix, p. 480 (a di study of multiple benign cystic epithelioma and adenoma sebaceum). McDona Jour. Derm., 1912, xxiv, p. 295 (Tricho-epithelioma papulosum in a man ag eight; ten tumors). Ricker und Schwalb, Die Geschwülste der Hautdrüsen, Ber

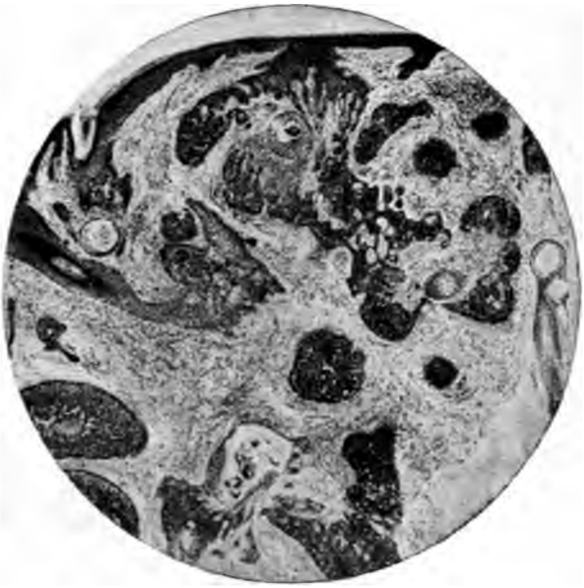
[2] Adamson, Brit. Med. Jour., March 7, 1914 (Four cases of epithelioma cysticum (Brooke)).

[3] Brit. Jour. Derm., 1892, iv, p. 269.

[4] Jour. Cut. Dis., 1892, x, p. 45

zell[1] and others confirm these findings. The lesions occur most often on the face—about the root of the nose, the temples, eyelids, cheeks, forehead, and chin; rarely over the neck, mammary glands, scalp, and upper extremities. There is a tendency for the lesions to be symmetrical, particularly about the face. They are minute, pearly, pale-yellowish or pinkish tumors, sometimes yellowish-red, occasionally having a bluish shade, and vary in size from that of a small pinhead to that of a pea, though much larger lesions occasionally develop. The larger lesions may have associated telangiectatic bloodvessels, and resemble to a degree the early lesion of rodent ulcer. They are firmly imbedded in the skin, project to a variable degree above the surface, are round or oval, solid and painless to the touch. The larger tumors are dense, lucent, and freely movable over the underlying tissue. In some cases the lesions are translucent and suggest in their appearance vesicles; in others, they closely simulate milia; in yet others there is a central depression, sometimes having a blackish point. The lesions are discrete and lie closely placed, in some cases showing none of the characteristic grouping. They develop slowly and continue to increase for a certain length of time and then remain stationary. As a rule, they do not ulcerate. Exceptions to this have been noted by White, J. C.,[2] Jarisch,[3] Stelwagon,[4] Schamberg,[5] and others. They are not, as a rule, the seat of subjective sensations.

FIG. 219



Multiple benign epithelioma (epithelioma adenoides cysticum) of the face, scalp, and upper part of the trunk, showing origin of new growth from surface of the epidermis (Spencer 1 inch. Spencer aplanatic ocular 1½ inch). (Fordyce.)

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[1] Brit. Jour. Derm., 1904, xvi, p. 361.

[2] Jour. Cut. Dis., 1894, xii, p. 477.

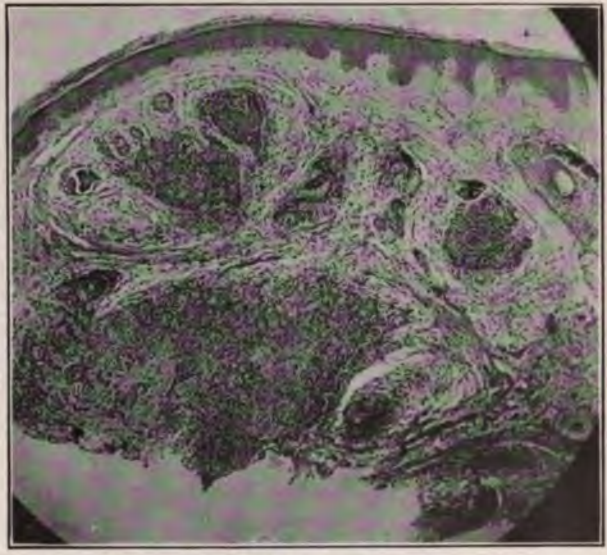
[3] Archiv, 1894, xxviii, p. 163.

[4] Diseases of the Skin, 7th ed., p. 652.

[5] Jour. Cut. Dis., 1909, xxvii, p. 170; ibid., 1911, xxix, p. 500.

Etiology and Pathology.—The disease occurs most often about the period of puberty, and in women more frequently than in men.[1] In some cases there is a distinctly inherited tendency to the disease. Since the studies of Brooke, Fordyce, Hartzell, and others, all observers agree as to the origin of these growths. Adamson[2] regards the tumors as embryonic hair-follicles, which are stimulated to growth at puberty. Earlier observers were of the opinion that they were due to misplaced epithelial cells of indifferent nature. The histological findings agree in the main. Brooke noted rope-like epithelial tracts or finger-like projections extending into the corium, sometimes forming coil-like masses, in which colloid degeneration occurred, producing oval and circular cysts; the masses being surrounded by a condensed connective-tissue capsule devoid of inflammatory reaction. Fordyce recognized irregularly rounded, oval, elongated masses and tracts of epithelial cells corresponding to those in the lowermost layer of the epidermis and the external root-sheath of the hair-follicle. The epithelial masses were distinct or made up of intercommunicating bands or tracts, in some places resembling coil-ducts. "Cell-nests" were met with, as in malignant epithelioma, enclosing horny, granular, and colloid material. In addition, colloid degeneration was detected in individual cells of the cell masses. Adamson[3] classes rodent ulcer with multiple

FIG. 220



Photomicrograph of multiple benign cystic epithelioma.

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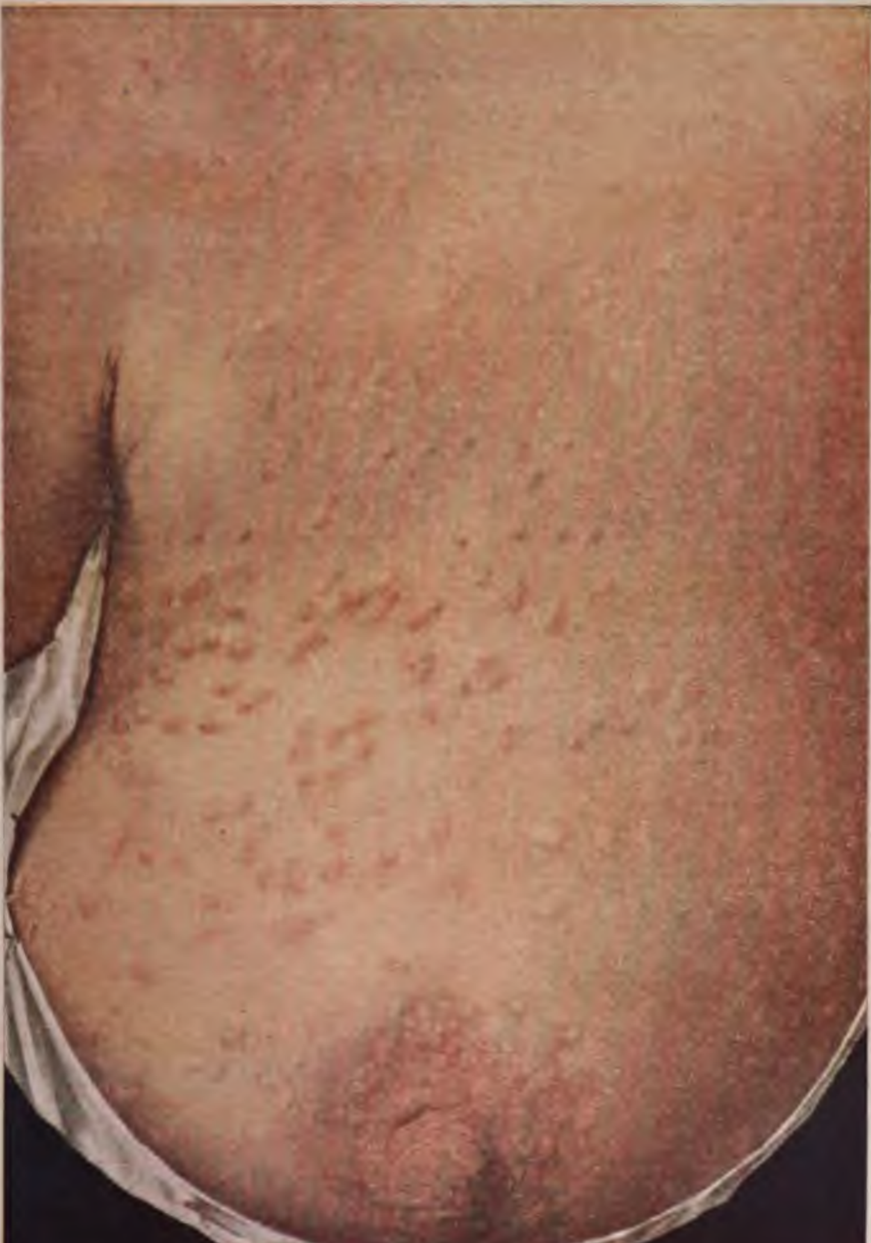
[1] Paul, Medical Jour. of Australia, 1917, iv, p. 8; Hereditary tendency in family. Miller, Jour. Cut. Dis., 1915, xxxiii, p. 462. Three members in one family.

[2] Loc. cit.

[3] Lancet, London, March 21, 1914, p. 810.



Syringocystoma.

benign cystic epithelioma, and considers them both nevoid growths. Cases presenting the clinical association of the two types are recorded.[1]

Diagnosis.—The distinguishing features of the disorder are: its occurrence often in several members of one family, its date of onset usually being in childhood or adolescence; the presence of the major portion of the lesions on the face and head; their persistence, and occasionally their termination in ulceration and development into malignant epitheliomata. These points, taken into consideration with the characteristic lesions above described, will serve to differentiate the disease from adenoma sebaceum and syringocystoma, disorders with which it is most likely to be confused. From rodent ulcer occurring as a single lesion, no difficulty will be encountered, but in cases of multiple rodent ulcer the resemblance is close, the chief difference being the occurrence of rodent ulcer at a later period of life.

Treatment.—Neither internal remedies nor external applications have any influence upon the lesions. The treatment is therefore surgical and calls for the employment of the knife, the curette, or scarification, depending upon the size, number, and location of the lesions. In several cases the latter have been removed successfully by means of electrolysis. *X*-rays may be used in selected cases.

Prognosis.—As a rule, the lesions remain indefinitely without change. Exceptions occur as above noted, when epithelioma of a malignant nature develops, and this is to be remembered in forecasting the future progress.

SYRINGOCYSTOMA.[2]

Synonyms.—Hidradénomes éruptifs (Jacquet and Darier); Syringocystadénome (Török); Epithéliome kystique bénin (Jacquet); Cellulome épithélial éruptif (Quinquaud); Cystadénome épithélial bénin (Besnier); Lymphangioma tuberosum multiplex (Kaposi); Syringocystoma (Neumann); Syringoma (Fiocco). Hemangio-endothelioma tuberosum multiplex (Jarisch). Ger., Schweissdrüsen—Adenom mit cystenbildung.

A group of rare cases has been recorded, the first by Kaposi,[3] under the title *lymphangioma tuberosum multiplex*; others under the titles mentioned above, the type for these being probably best illustrated by those described by Darier and Jacquet.[4] In America studies have been made by C. J. White,[1] Sutton and Dennie,[2] the author,[3] and others.

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[1] Dore, Brit. Jour. Derm., 1912, xxiv, p. 190 (A combination of a number of small typical lesions of multiple benign cystic epithelioma with several larger ulcerating tumors with a pearly edge and other characteristics of rodent ulcer).

[2] Darier, La Pratique Derm., 1900, i, p. 288. Jacquet, Congrès Intern. de Derm., Paris, 1889. Neumann, Archiv, 1900, liv., p. 3. Brauns, ibid., 1903, lxiv, p. 347. Fiocco, Giorn. ital., 1904, p. 3. Besnier, Translation of Kaposi's Treatise, ii, p. 367. McDonagh, Brit. Jour. Derm., 1912, xxiv, p. 228 (Syringoma. Patient aged twelve years; lesions developed at end of first year). Sequeira, ibid., 1913, xxv, p. 140 (Hidradénomes éruptifs (syringoma)). Strümpke, Derm. Wchnschr., 1915, lx, p. 362; Ein Fall von Syringo-cystadenom. Bunch, Proc. Royal Soc. Med., 1916-17, Derm. Sect., p. 162. A Case of Syringocystoma. Fox, H., Jour. Cut. Dis., 1917, xxxv, p. 258; Syringocystoma in a Negress. Discussion. Clark, ibid., 1917, xxxv, p. 607; Syringocystadenoma; case report.

[3] Handbuch, 1872, ii, p. 282.

[4] Annales, 1887, s. ii, viii, p. 317.

Much confusion has existed relative to the relationship of this disease to multiple benign cystic epitheliomata. The latter has been clearly worked out by Brooke, Fordyce, Hartzell, and others, and should be readily differentiated. The clinical picture of the syringocystoma group is fairly well differentiated from the others, although some writers believe the distinctions are not important.

Symptoms.—The patients are for the most part women in early life. The lesions are situated on the trunk, over the clavicles, shoulders, and chest, and about the axillæ, extending from the latter position over the abdomen and occasionally over the extremities; more rarely over the face, eyelids, nose, ears, and scalp. The lesions are pinhead- to split-pea-sized and sometimes larger, round, smooth, globoid nodules, in some cases discrete, in others closely packed, soft in consistency, fawn-colored to yellow in tint in some instances, lighter in others, and in still others of a reddish, brownish, or darker hue, occasionally presenting a somewhat waxy appearance. They are nearly level with the skin or slightly elevated above it.

The course of the disease, when not removed by treatment, is toward persistence of the lesions after attaining a maximum development. There are few, if any, subjective sensations, and the general health of the subject of the disease is unimpaired. The involvement of the sweat-apparatus was indicated clinically in White's case by diminution in the production of sweat. On the other hand, Sutton found the glands capable of functionating.

Pathology.—In the pathogenesis of the disorder it has been suggested that the new-growth is derived from misplaced embryonic coil-elements, a theory corresponding closely in principle to that given in explanation of the production of multiple benign cystic epithelioma which was suggested by Quinquaud and Jacquet, who considered that misplaced epithelial cells of indifferent nature were responsible for the growths. White[4] believes syringocystoma is a hyperplastic and hypertrophic change in previously existing efferent sweat-ducts. Stockmann[5] regards these growths as tardy nevi developing in abnormally placed sweat-glands.

The histology has been studied by Jacquet, Darier, C. J. White, and others. In the case of Jacquet and Darier, pathological changes were noted in the corium, where there occurred epithelial cylindrical ramifying prolongations, which on section showed canals and cysts containing amorphous matter. These newly-formed elements extended from a short distance below the epidermis to the hypoderm and were not encapsulated. There was never traced any direct connection between these tubules and the sweat-apparatus, nor were they traced to other structures of the skin. A double layer of cells about a simple channel and other facts obtained from a study of the sections caused Darier to ascribe their origin to the sweat-ducts.

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[1] Jour. Cut. Dis., 1907, xxv, p. 49 (full bibliography, 4 plates, 6 histological photographs).

[2] Jour. Amer. Med. Assoc., 1912, lviii, p. 333 (A clinical and histological study and comparison of a case of multiple benign cystic epithelioma and syringocystadenoma. Bibliography).

[3] Jour. Cut. Dis., 1910, xxviii, p. 433.

[4] Loc. cit.

[5] Archiv, October, 1908, xcii, p. 145; abstr. Jour. Cut. Dis., 1909, xxvii, p. 138 (report of three cases).

The histological changes in White's case are described as occurring between the subpapillary layer of the corium and a point just above the panniculus adiposus. Within this area clusters of epithelial cells forming bodies of various shapes were noted, and the supporting corium exhibited some degenerative changes. In the center of some of these masses true cysts with atrophic cells were noted, but for the most part an empty lumen was observed.

Sutton found the cells still retained their sweat-secreting function, and concluded that the tumors were true adenomata.

Diagnosis.—The diagnosis is to be established chiefly from multiple benign cystic epithelioma, the softish nodules of syringocystoma, with their general hue, distribution, and lack of pearly elements, rendering the clinical recognition facile. In typical cases, papular syphilodermata are, as a rule, accompanied by other signs of lues. The xanthomata have a yellower tint, but in many respects strongly resemble the lesions of syringocystoma.

Treatment.—Treatment has been of little avail in most cases. Locally, the lesions may be removed by surgical measures, by electrolysis, by liquid air, or by carbon-dioxid snow. Hodara,[1] Max Joseph and Siebert,[2] the author, and others have successfully treated these cases with *x*-rays.

Prognosis.—After developing to a certain extent the lesions remain indefinitely. They do not undergo spontaneous involution, nor do they develop malignant changes, and they have no effect on the general health.

XERODERMA PIGMENTOSUM.[3]

Synonyms.—Angioma pigmentosum et atrophicum; Atrophoderma pigmentosum; Dermatosis Kaposi; Melanosis lenticularis progressiva;

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[1] Derm. Wochenschr., 1913, lvi, No. 15, p. 421; abstr. Jour. Cut. Dis., 1913, xxxi, p. 791 (A case of hidradenoma eruptivum (Darier and Jacquet, syringocystadenoma).

[2] Derm. Wochenschr., 1913, lvi, No. 15, p. 425; abstr. Jour. Cut. Dis., 1913, xxxi, p. 792 (Successful treatment of hydrocystoma tuberosum multiplex).

[3] Kaposi, Wien. med. Wochenschrft., 1885, p. 1334. White, J. C., Jour. Cut. Dis., 1885, iii, p. 353. Bowen, A. H., Jour. Amer. Med. Assoc., April 29, 1889. Pollitzer, Jour. Cut. Dis., 1892, x, p. 133 (histological study in one of Crocker's cases). Brayton, ibid., p. 129. Kaposi, Twentieth Century Practice, v, p. 727. Lukasievicsz, Archiv, 1895, xxxiii, p. 37 (*résumé* of 73 cases and bibliography). Herkheimer u. Hildebrand, Münch. med. Wochenschrft., 1900, xlviii, p. 1099 (abstr. Brit. Jour. Derm., 1901, xiii, p. 66). Kreibich, Archiv, 1901, lvii, p. 123. Monthus, Annales, 1902, s. iv, iii, p. 673. Michael Terterjansz, Inaug. Dissert., Berlin, 1902. Löwenbach, Mraček's Handbuch, Bd. iii, p. 240 (full bibliography). Crocker, Diseases of the Skin, 3d ed., p. 681. Hyde, Jour. Cut. Dis., 1903, xxi, p. 573 (three cases of xeroderma pigmentosum; these illustrate the disease in this text). Förster, Deutsche. med. Zeit., 1904, No. 74, p. 77. Adrian, Centralb., 1904, vii, p. 130. Nicolas and Favre, Annales, 1906, s. iv, vii., pp. 536-549. Klein, Inaug. Dissert., Strassburg, 1906. Terebinski, Russki. Wratsch., 1906, No. 48.

Lioderma essentialis cum melanosi et telangiectasia; Lentigo maligna. Fr., *Epitheliomatose pigmentaire*.

Definition.—Xeroderma pigmentosum is a rare disease which begins in childhood, is characterized by hyperpigmentation, atrophic areas, telangiectases, warty and malignant growths.

The disorder was first described by Kaposi in 1870, and although a rare disease a large number of cases have been recorded in different countries. In America such records have been made by Taylor, Duhring, C. J. White, Bronson, Brayton, and others.

Symptoms.—The disease most often begins early in life, although it has been observed first in adults and even at an advanced age. The early lesions are difficult to determine. In a certain number of cases the first change from the normal has been an erythema or dermatitis. In several instances sunburn was recorded as preceding the characteristic eruption. Following this, or independent of it, pigmented lesions occur and these are usually the first sign of the disorder. These lesions resemble ordinary freckles. They begin in the summer and subsequently are exaggerated during that season, becoming less conspicuous in winter. The lesions occur on exposed surfaces of the body, which include the face, neck, upper chest as far as the third rib, the hands and the forearms. In rare instances other portions of the cutaneous surface become involved, such as the trunk, buttocks, thighs, legs and feet, and Crocker reports these lesions occurring in the subungual space. The pigmentation differs in hue, depending upon the severity of the process and its duration, and presents different shades of yellow and brown. As a rule, these pigmentations are small but by fusion areas several centimeters in diameter may be formed. Interspersed among these lesions are whitish, atrophic spots which may be isolated or coalesce and form cicatriform patches. In these areas the skin is white and shining slightly wrinkled, smooth or covered with fine scales. The ectropion which commonly is present is believed by Crocker to be due to these atrophic areas. Circumscribed atrophic spots not infrequently follow telangiectases. The latter are characteristic of this disease and form a part of the eruption. They may be punctate or stellate and are usually developed in atrophic areas. Not infrequently pinhead-sized angiomata occur. They may be few or numerous and situated in the same areas as the other lesions. Warty growths of

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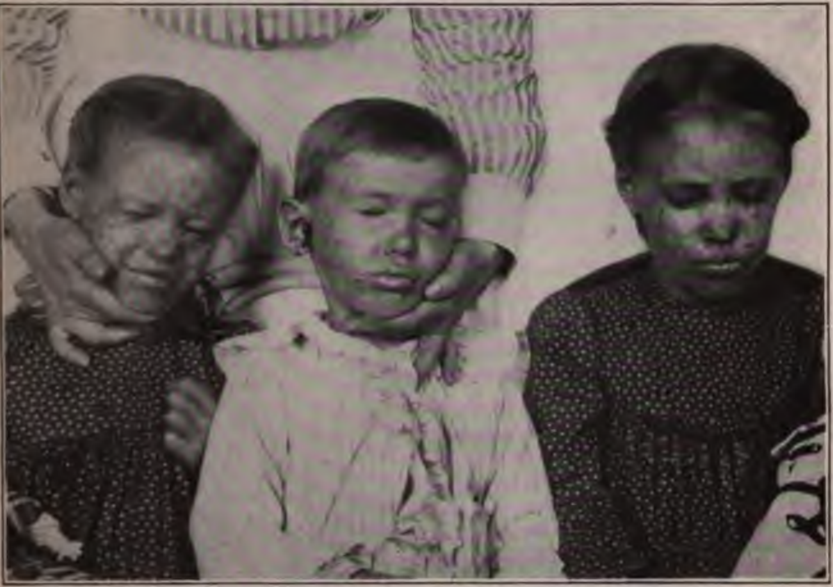
Askura, Japan. Zeits. f. Derm. u. Med., 1906, p. 1 (two cases). Low, Zeitschrift, 1906, xiii, p. 488. Vignolo-Lutati, Monatshefte, 1907, xlv, pp. 21 and 72. Josef Yussman, Centralb., 1907, x, p. 258. Rouvière, Annales, January, 1910, p. 34 (three cases, sisters, five other members of same family not affected; one died soon following glandular enlargement, one cured with x-rays). Fox, G. H., Jour. Cut. Dis., 1910, xxviii, p. 200. Fox, Howard, ibid., 1911, xxix, p. 599 (adult case). Carmichael, ibid., p. 307. White, C. J., ibid., 1912, xxx, p. 290. Toyama, Jap. Zeits. f. Derm. u. Neurol., 1912, xxi, No. 4, p. 24 (this author collects 33 Japanese cases, which he adds to 196 previously recorded in the literature). Gottheil, Jour. Cut. Dis., 1913, xxxi, p. 515. Simpson, C. A., ibid., p. 1020 (three cases). Corlett, ibid., March, 1915, xxxiii (Xeroderma pigmentosum following severe exposure; two cases, one adult). Grindon (three cases shown at the 38th Annual Meeting of the American Dermatological Association, Chicago). Jour. Cut. Dis., 1915, xxxiii, p. 394; Kessler, Trans. Soc. on Derm., Amer. Med. Assoc., 1915, p. 71.

different characters also occur, and sometimes precede the atrophic spots above mentioned. They may be flat or convex, and very much resemble verrucous lesions seen in old age. They may develop from pigmented spots or independently, and terminate by exfoliation or malignant transformation. They may be pea-sized or larger and are irregularly disseminated among the other lesions. At a later date malignant transformation occurs and various types of tumor formation result, the most common of which is carcinoma. Even at an early stage there is marked photophobia and the lacrimation thus resulting produces irritation, which may be followed by ulceration on the face. Ulceration also occurs as the result of epitheliomatous degeneration. Keratitis is common and nodules and opacities occur on the cornea, often obstructing vision to a marked degree. The scalp may be free or the site of moderately marked scaling. Pigmentation in this area is also a matter of record. As the disease progresses thinning of the skin is seen in the affected areas, producing so-called "parchment skin."

FIG. 221



Xeroderma pigmentosum.

The course of the disease is variable, some of the children dying within several months of marasmus. In certain instances its progress appears to be checked and remains stationary over a long period. In many cases malignant transformation occurs after several years, which terminates fatally. A number of patients have survived to adult years. J. C. White reported the case of a patient with xeroderma pigmentosum who lived for forty-five years. Sequeira[1] and several others have recorded cases in which the disease had been of many years' standing. In rare instances pigmentation may be absent. Such a case is reported by Audry.[1] A number of cases are on record in which the symptoms above described have begun in adult life and even at an advanced age. Buermann and Gougerot[2] report the case of a patient sixty-seven years of age so affected. Howard Fox records the case of a patient aged twenty-six years in whom the disease had been present for nine years. Corlett and others describe similar cases and the latter classifies these with the juvenile type. The striking thing about the disorder is its analogy to the group of changes which occurs in the senile skin.

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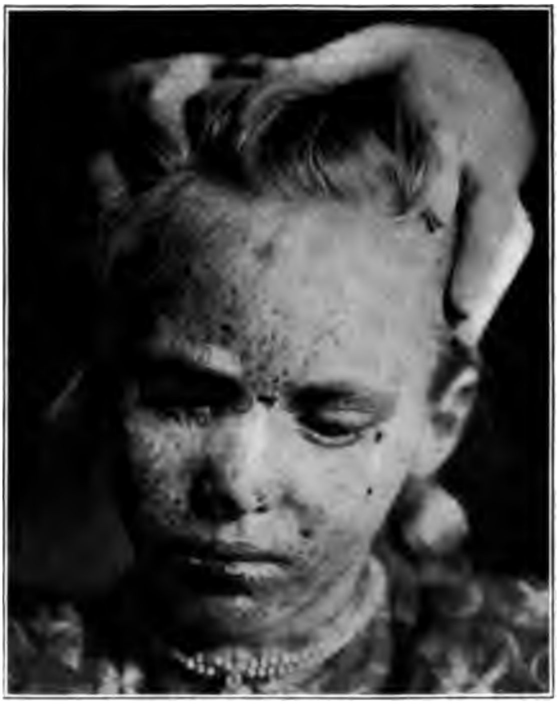
[1] Brit. Jour. Derm., 1906, xviii, p. 253.

FIG. 222



Xeroderma pigmentosum.

Etiology.—Xeroderma pigmentosum commonly begins before the third, usually during the first year of life. It is distinctly a family disease, being present in two or more members, as a rule. The cause of the disease is unknown, but its occurrence in several members of a family suggests some peculiar predisposition of the skin to respond to irritation. Toyama concludes, after a study of thirty-three cases recorded in Japan, that the prevalence of the disease in that country is due to intermarriage. Vignolo-Lutati believes that the disease is produced by the inflammatory reaction seen in a predisposed skin, this predisposition being due to the pathological condition of the cells caused by blood relationship. Corlett concludes that it is highly probably that the disease is dependent primarily and essentially on an inherited susceptibility or predisposition which is called into action by certain rays of the spectrum. The influence of light upon the development of the disease in susceptible individuals has been noted by many observers, and the consensus appears to be that the disease is the result of the action of the sun's rays on a peculiarly sensitive skin.

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[1] Annales, 1907, s. iv, viii, p. 199.

[2] Ibid., 1906, s. iv, vii, p. 391.

Pathology.—According to Kaposi the process appears to begin with proliferation of the connective tissue of the papillæ and of the endothelium of the vessels. This is followed by obliteration of some of the vessels, dilatation of others, and retraction of the papillæ. Secondarily there occurs downgrowth of the rete into the dermis, irregular accumulations of pigment, dilatation of the glands and degeneration of their epithelium.

Crocker considers the disease an atrophic degeneration of the skin dependent upon a primary neurosis to which there is a congenital predisposition. The early changes have not been sufficiently studied, owing to the late observation of most of the cases. As a rule, the disease is well advanced before coming under observation. In a study of various types of lesions Councilman and McGrath[1] found the process to be one of atrophy of both the epidermis and the corium, except in the warty masses and tumors, these lesions being superficial. They found the horny layer thickened everywhere, the granular layer imperfect or absent and the prickle layer atrophic. In warty masses the thickening of the horny layer was exaggerated and showed dense adherent masses of keratinized cells arranged in parallel layers or whorls; occasionally at the bottom epithelial pearls were noted. Beneath these the rete pegs were elongated, the granular layer was well formed, and there were actively proliferating cells in the rete. The papillæ were elongated in proportion to the downgrowth of the rete pegs. At the edge acanthosis was sufficient to suggest superficial carcinoma. In the corium, except in the warty areas, the papillæ were absent or small and atrophic. The connective tissue immediately beneath the epidermis was dense, cicatricial-like and individual fibers were indistinguishable. The elastic tissue was increased.

The vessels were few except beneath the warty growths, where they were abundant. They were normal and the common type was a thin-walled vein. The hair follicles were poorly formed, the coil glands atrophic with cell and nuclear fragmentation. The normal connective-tissue cells were reduced in number and there was a marked cellular infiltration in areas around the vessels. Pigment was present to a greater or less degree. In the atrophic areas only an occasional pigment cell was seen. In the pigmented areas it occurred both in the epidermis and upper corium and was always intracellular. It was usually found in the lower layer of the rete but in well-marked lesions extended to the upper layers. The pigment was granular and bronze or greenish-black. The base of an ulcer was found to be composed of dense cicatricial tissue with few cells or vessels and no evidence of inflammatory reaction. In this location the scar tissue extended deeply.

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[1] Jour. Med. Research, [illegible] p. 331.

The tumor-formations involved only the epidermis and corium and at the base showed small alveoli and small degenerating cells. The vessels in the stroma were large, thin-walled veins and not numerous. As an exception to the findings in other locations, there was in this region a marked leukocytic infiltration.

A variety of new growths have been described by various observers in connection with this disorder. Most observers consider the new growths to be carcinomata of the basal-cell type. Early, Crocker described non-malignant papillomatous tumors. Pollitzer found in a single tumor changes characteristic of sarcoma, carcinoma and myxoma. Kreibich classed the tumors as medullary carcinomata in which the basal layer of the rete is preserved for a long period. The ulcers are sometimes due to infection and sometimes are carcinomatous. In an ulcer taken from in front of the ear in a patient six years of age, the author found the following histological structure: The epidermis showed marked thickening, acanthosis increasing toward the border of the ulcer. The basal layers of the epidermis, however, were intact. The corium exhibited vascular dilatation and perivascular infiltration extending to the hypoderm. The collagen and elastin were largely destroyed and replaced by a dense infiltration of small, round cells reverting to the embryonic type, connective-tissue cells, plasma cells, and polymorphonuclear leukocytes. There were also edema and here and there deposits of pigment. The appendages of the skin, which include the hair follicles and coil glands, were intact. As will be noted, there is nothing characteristic of the disease in this particular ulcer.

All of the histological changes noted in the disease are closely simulated in the condition described by Unna as "sailor's skin," and also in certain cases of senile skin and, finally, in the chronic inflammatory, atrophic, telangiectatic, hypertrophic, pigmented and malignant transformations found following the prolonged use of x-rays.

Diagnosis.—Xeroderma pigmentosum is such a characteristic disorder that an error in diagnosis is hardly possible. The early date of the onset in most cases, the occurrence of lesions chiefly on exposed areas and the combination of pigmented lesions, telangiectases, atrophic spots and areas, and warty growths, associated with the peculiar photophobia and lacrimation that occurs in a well-developed case are all striking features. The clinical picture presented in a chronic radiodermatitis closely resembles that seen in xeroderma pigmentosum, a fact pointed out by Pusey. A somewhat similar picture also is presented in elderly people. In occasional instances scleroderma has to be differentiated, but the board-like areas and peculiar atrophy occurring in this disorder is distinctly different from that seen in xeroderma pigmentosum. The limitation of the lesions to exposed areas in the latter disease is not observed in scleroderma.

Treatment.—No treatment has been advised which has any effect on the disease as a whole. Its symptoms may be relieved by various methods. Tonic treatment is always suggested, this to be given according to the rules of general medicine. The epitheliomatous ulcers and other malignant growths have been treated with good results with radiotherapy and radium. In a group of cases observed with Dr. Hyde much improvement resulted from the general care received at the hospital. In these patients epitheliomatous ulcers responded readily to radiotherapy. Sichel[1] and Balzer and Merle[2] had a similar experience. Jamieson[3], Allen,[4] Rouviere[5] and others report cases cured with radiotherapy. Pringle[6] demonstrated a patient in whom a large carcinoma had been greatly reduced by the use of radium.

Prophylactic treatment is indicated in the prevention of exposure of these patients to the direct rays of the sun and, as Corlett so well expressed it, particularly against sudden or too protracted exposure in people unprotected by an immunizing coat of tan.

Prognosis.—In the major portion of cases the outlook is grave, though many patients live for a number of years.

CARCINOMA OF THE SKIN.

Carcinoma may have its origin in the skin or develop there secondarily from growths situated in other regions. There are several varieties, the most important of which from the dermatological standpoint is epithelioma. Other forms which have to be considered are the lenticular, tuberose, melanotic carcinoma, and Paget's disease.

Carcinoma Lenticulare.[7]—This is rare as a primary cutaneous manifestation but appears generally secondary to a carcinomatous growth of other organs, chiefly of the breast. It may develop in the skin over the region of a carcinomatous breast either before or after the extirpation of the latter. The lesions usually develop slowly, but in occasional instances a shower occurs. They may be few or numerous, isolated or closely packed, producing a diffuse infiltration. They are pea- to bean-size firm, red or bluish-red, shining papules or nodules. After continuing for some time crusted ulcers, papillary and fungus growths, together with large areas of induration, form. When the infiltration is widely diffuse, involving a large portion of the integument of the thorax, the skin becomes densely indurated, producing a leathery covering over which are sprinkled the papules and nodules above mentioned, a condition which Valpeau termed "*cancer en cuirasse.*" The edges of the infiltration are poorly defined except

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[1] Guy's Hosp. Gazette, xxiv, p. 114

[2] Annales, 1906, s. iv, vii, p. 1054.

[3] Lancet, London, 1903, i, p. 105.

[4] Jour. Amer. Med. Assoc., 1903, xl, p. 508.

[5] Loc. cit.

[6] Brit. Jour. Derm., 1916, xxviii, p. 253.

[7] Morrow, Jour. Cut. Dis., 1884, ii, p. 1; Hyde, Amer. Jour. Med. Sci., 1892, ciii, p. 235; two cases fully described.

Fig. 223

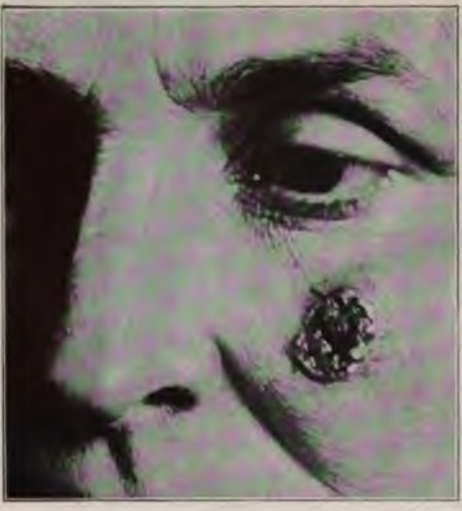


Fig. 224

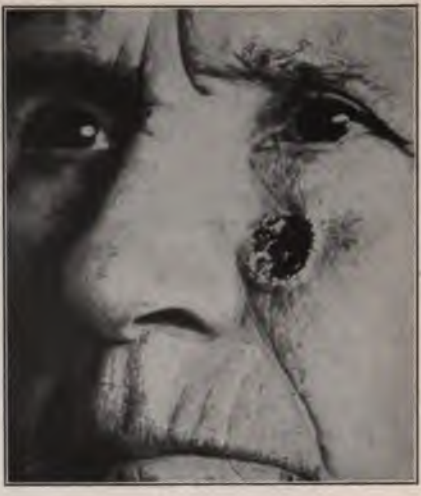


Fig. 225

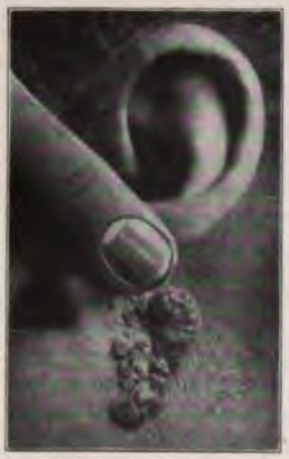


Fig. 226

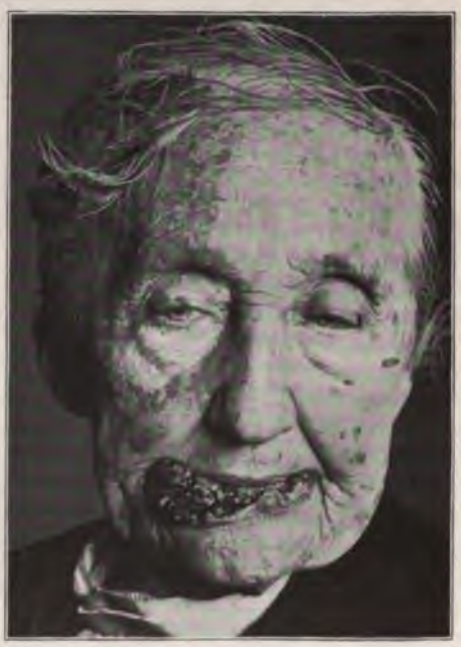
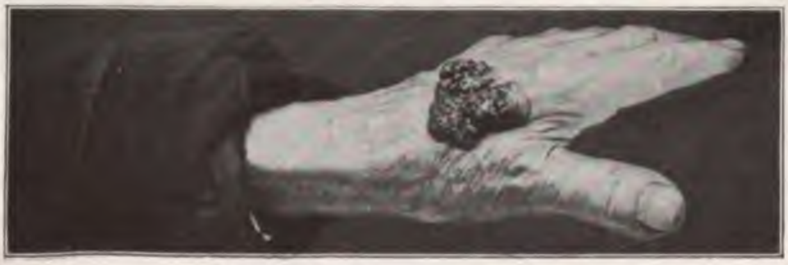


Fig. 227



Clinical varieties of cutaneous carcinoma.

where tongue-like prolongations of dull-red hue extend from the margin, indicating the advance of the carcinomatous process. The lymphatic circulation is obstructed, the regional glands enlarged, and sooner or later the arm and forearm on the affected side become markedly swollen and edematous. In case the disorder occurs before operative removal of the breast the nipple may or may not be retracted. The breast is firmly bound down to the chest wall by the thickened skin, which is hard, smooth or rough, shiny and either reddened or of the normal color, often exhibiting telangiectatic bloodvessels and ulcerating areas. A zoster-like arrangement was recorded by Welsh,[1] and lesions on the lower abdomen and upper thigh by Neustadt.[2]

While commonly occurring in women the disorder is sometimes seen in men. Such a case is recorded by Hartzell[3] and three such examples have been under the observation of the author. In the last case, which was recently seen, the disorder developed following removal of the breast, which was carcinomatous. In this case the nodules developed extensively and resembled the condition usually seen in women. A case with extensive involvement was recorded by Pollitzer[4] in which the skin over the greater part of the trunk, front and back, was red, hardened, dotted over with lichenoid papules and nodules and intensely pruritic.

The course of the disease may be rapid, but ordinarily several years elapse before a fatal termination. In one patient with extensive involvement, the disorder was improved and the patient kept comfortable over a period of five years with radiotherapy.

Carcinoma Tuberosum.—This is a rare manifestation of the disease characterized by larger lesions than those described in the preceding variety. They begin in the deeper portions of the skin, gradually enlarge, and extend toward the surface. They may be few or numerous, discrete or grouped, forming nodular masses. They vary in size from small nodules in the beginning to egg size or larger later. They are firm, oval or round, and as they increase in size the overlying skin becomes tense, reddened, brownish-red or bluish in hue. Their tendency is to break down into ulcers, the latter not infrequently presenting fungating growths. General metastases involving the internal organs may supervene during the course of the disorder, which induces cachexia and a fatal termination.

Melanotic or Pigmented Carcinoma.—This is a rare and malignant form, characterized by the presence of pigment in the lesions. The growths usually begin as hemp-seed to pea-sized, single or numerous, soft or firm, nodules, which may develop into tumors of varying size and which are pigmented to a variable degree. The lesions may be grayish-brown, slate colored or black, and occasionally the pigment is irregularly distributed in streaks or bands over the surface of the growth. They may occur on any portion of the surface, often upon the extremities and about the genitals, and they have their origin in pigmented nevi. In addition to the above-mentioned lesions, papillary and fungoid growths and ulcerations occur. Not infrequently verrucous masses are seen surrounded by grayish or black papules, or by diffuse carcinomatous infiltration. The growth usually has a relatively rapid course and is malignant to a high degree. Occasionally it may remain localized for some time. A melanotic rodent ulcer and superficial melano-epithelioma and an infiltrating melano-epithelioma hav been described by Johnston.[1] Recent investigations[2] indicate tha the majority, if not all, of the malignant pigmented growths whic spring from moles, and which in the past have been considered sarcomatous are pigmented carcinoma.

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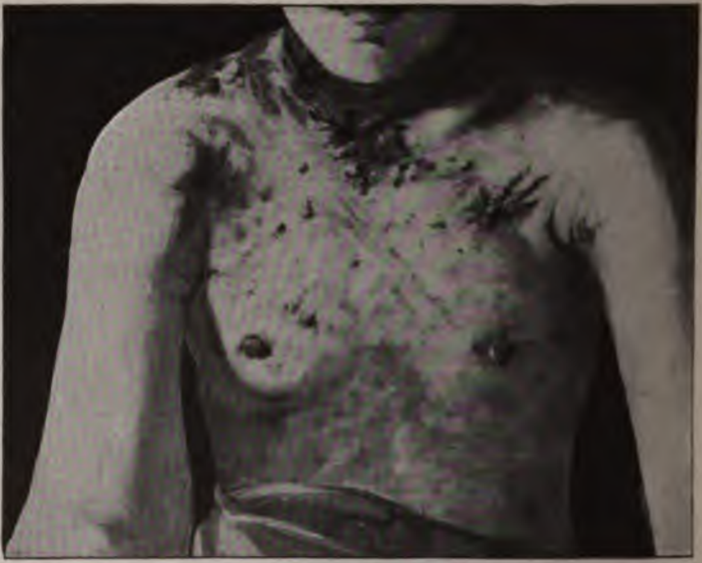
[1] Brit. Jour. Derm., 1908, xx, p. 271.

[2] Zeitschr., 1912, xix, No. 6, p. 487.

[3] Jour. Cut. Dis., 1910, xxviii, p. 97; Case demonstration, a male, greatly improved with x-rays.

[4] Ibid., 1909, xxvii, p. 143.

FIG. 228



Cancer *en cuirasse*, chiefly involving the right side of the chest.

Pathology.—The histological structure of the lenticular and tuber varieties is that of other squamous-cell carcinomas. In the pigmente variety the structure resembles that of the pigmented mole from whic it developed. With malignant transformation a marked proliferati of epithelial cells occurs and the pigment is increased in varyin grades. The epithelial cells have an alveolar arrangement and man are multinucleated. Mitotic figures are seen. The pigment occurs i and between the cells and may be produced in such quantities as t obscure the minute structure of the growth. The pigment is the product of a cell (melanin) and not deposited from the blood. An interalveolar cellular infiltration consisting of lymphocytes and plasma-cells is present in varying degree and the amount of fibrous tissue depends upon the degree of the epithelial invasion.

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[1] Jour. Cut. Dis., 1905, xxiii, p. 64.

[2] *Cf.* melanotic sarcoma.

Treatment.—In single and early lesions of any variety complete surgical removal is demanded. Unless completely excised metastatic dissemination may be augmented, particularly in the pigmented form. In any type when dissemination has occurred radiotherapy may be of service in relieving pain and in removing the tumors or inhibiting their growth.

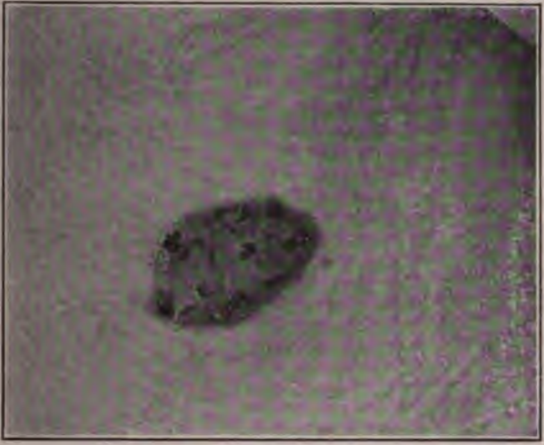
Prognosis.—The disseminated varieties are always grave. In early cases complete removal of the growths when localized results favorably.

Epithelioma.—**Synonyms.**—Epithelial cancer, Cancer of the skin, Rodent ulcer, Carcinoma epitheliale; Fr., *Epithéliome;* Ger., *Epitheliom.*

Definition.—Epithelioma is a destructive new growth arising from the epithelial structures of the skin.

Epitheliomata may be flat and superficial, or deep and nodular, papillomatous or fungoid.

FIG. 229

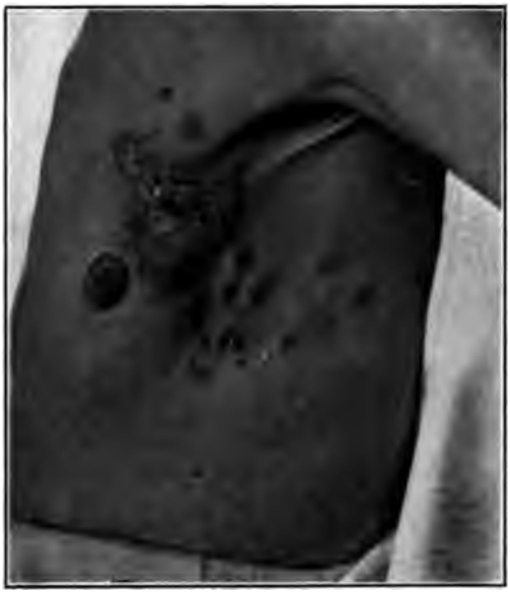


Superficial epitheliomatosis. Numerous other similar areas present.

Superficial Variety.—The superficial flat variety may originate in a senile verrucous or keratotic area on the face, ears, hands and occasionally other areas, or in some other lesion of the skin. In the pea- to bean-sized patches of senile keratosis the first change noted is a thickening of the patch accompanied by slight redness, and the surface beneath the crust presents a superficial granulating ulcer which gradually increases in size, finally developing the ordinary characteristics of the epitheliomatous ulcer.

In other instances a superficial epithelioma may be exhibited as a pinhead- to pea-sized or larger, smooth, hard, waxy, semitranslucent, yellowish or whitish nodule, often with dilated bloodvessels upon its surface. This lesion may remain for years without change except slight increase in size. There may be a single lesion, or several may be present, either widely separated and discrete, or grouped in a small area. The nodules are often largely destroyed through ulceration, which at times begins early, and it is through this process, together with the formation of new nodules, that so many different aspects are presented. In the beginning the nodules become fissured or excoriated and crusts form which fall, either spontaneously, or are rubbed off or otherwise removed. Each successive fall reveals a larger ulcer beneath and in this manner the epithelioma develops. By peripheral enlargement round, oval or irregular patches are formed, which sometimes assume a gyrate configuration not unlike that seen in syphilis. These cases cicatrize as they progress and at times a superficial. scar-like, area results, which may have either a slightly elevated, crusted margin, or a rim of waxy papules or small nodules. In rare instances this form is said to heal spontaneously, and disappear, leaving a superficial scar.

Fig. 230

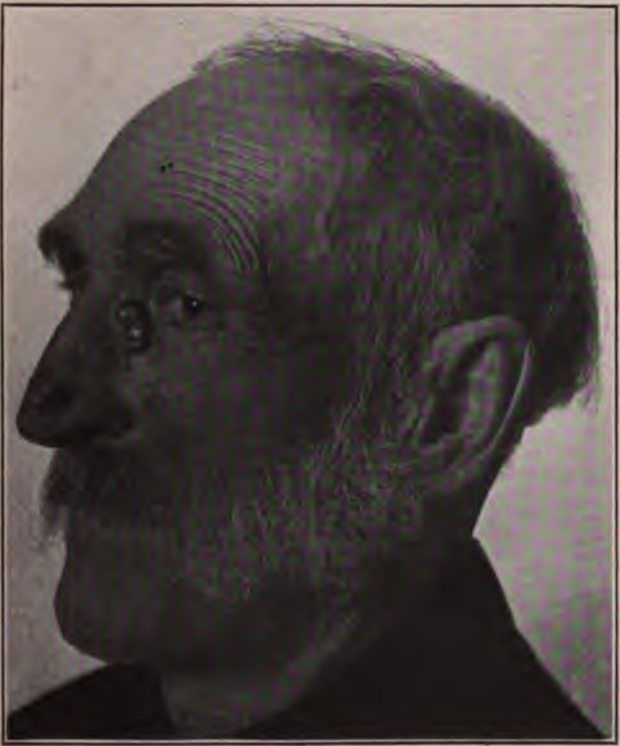


Carcinoma of the breast with lenticular, secondary nodules.

Small patches present a raised, round border, usually smooth. often telangiectatic, with a depressed center and with small, superficial, crust-covered or glazed ulcers. Larger areas present an uneven, ulcerating surface. with an elevated, indurated, irregularly indentated and telangiectatic border. The floor is depressed, uneven, and presents small areas of cicatrization, and reddish, moist elevations which bleed

The ulcers may have reddish-brown comparatively thick r present a varnished or glazed appearance, due to the drying thick, viscid secretions. Occasionally papillomatous growths bout the edges or on other portions of the floor of these ulcers. requently the destructive process is more marked than that d above, in which case the spreading ulceration destroys large f the surface, including at times the cartilage and bone. Great ction occasionally occurs about the nose or orbits, inducing cuous disfigurement. Even in these extreme cases metastases t occur.

FIG. 231



Epithelioma and preëpitheliomatous keratoses.

ent Ulcer[1] (Jacob's ulcer; Ulcus exedens; Chancroid ulcer; *Noli ngere; Jacobsches Karzinom*) was first described by Jacob and sidered by English authorities as distinct from epithelioma, but escription given above of superficial types, beginning as waxy es characterized by spreading ulceration, often with extensive destruction, and unaccompanied by metastases, applies to this

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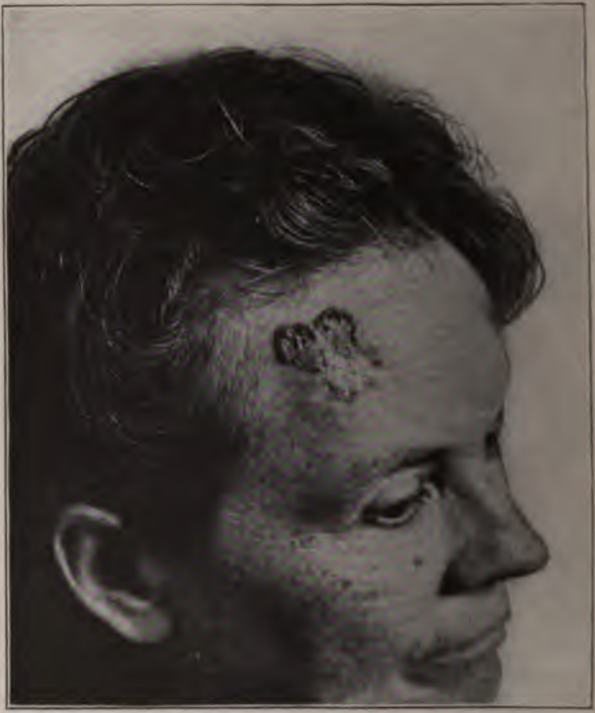
discussion of this type of epithelioma see Dubreuilh et Auché, Annales, 1901, , pp. 705–780; seventeen figures and bibliography. Little, Brit. Jour. Derm., vii, p. 145; "Rodent ulcer;" remarks on 127 personal cases, discussion of clinical tological findings, with references.

Morphea-like Epithelioma.—Examples of this type of epithelioma are recorded by Danlos,[1] Stelwagon,[2] Crocker,[3] Fordyce,[4] Hartzell,[5] who gave it the above title, Heidingsfeld,[6] and others. The lesions are flat, of irregularly rounded contour, slightly depressed, atrophic, or slightly infiltrated, yellowish or pinkish-yellow, and have associated telangiectatic vessels over the surface or at the periphery. Sooner or later superficial ulceration occurs in one or several areas. These are found on microscopic examination to be of the basal-cell type.

Fig. 232



Epithelioma.

Multiple Epitheliomata.—In addition to multiple lesions of the ordinary flat type above described, a form of superficial epithelioma is occasionally seen which occurs in variously sized patches, resembling extramammary Paget's disease, but lacking the eczematous features recognized in that disorder. In a case recorded by Ketron[7] which was apparently of this type, weeping and crusting were described. The patches are light or dark red and scale-covered, and begin as small areas which spread peripherally, often becoming as large as the palm or larger. Some of them are surrounded by a delicate, slightly elevated margin, resembling the rolled edge seen in rodent ulcer. Some of the patches remain flat and comparatively smooth, while in others definite nodules occur, some of which undergo ulceration, producing typical epitheliomatous ulcers. A fungating growth occurred in a case reported by Fordyce.[1] In a group of cases seen by the author the diameter of these patches ranged from one-fourth of an inch to several inches, many being the size of a silver dollar. They occurred on the trunk anteriorly from the clavicle to the pubic region and on various portions of the back, as well as on the arms and neck. In two instances lesions were situated on the face. They differed from senile keratoses in location and lacked the crusting and verrucous elements usually seen in these cases. In a histological examination made by Fordyce, a basal-cell epithelioma was demonstrated.

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[1] Annales, 1899, x, p. 656.

[2] Trans. Amer. Derm. Assoc., 1899, p. 166.

[3] Ibid, p. 1012.

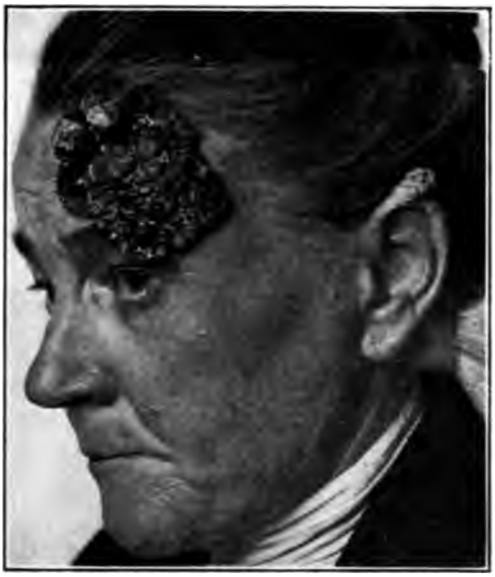
[4] Jour. Amer. Med. Assoc., October 24, 1908, p. 1398.

[5] Trans. Sec. on Derm., Amer. Med. Assoc., 1909, p. 25; Jour. Amer. Med. Assoc., 1909, liii, p. 262.

[6] Jour. Amer. Med. Assoc., 1912, lix, p. 256.

[7] Jour. Cut. Dis., 1919, xxxvii, p. 22; "Unusual forms of epitheliomas of the skin."

FIG. 233



Fungating epithelioma of forehead and ear.

Deep-seated Variety.—This form may originate in the superficial variety or develop independently. In the latter case it begins as a firm, pea-sized nodule in or beneath the skin, which gradually increases in size to that of a nut or small egg. It may be irregularly rounded or appear as a flat plaque with lateral extensions into the surrounding and deeper tissues. Its surface is pinkish or darker red and often covere with dilated bloodvessels. Ulceration occurs sooner or later, producin

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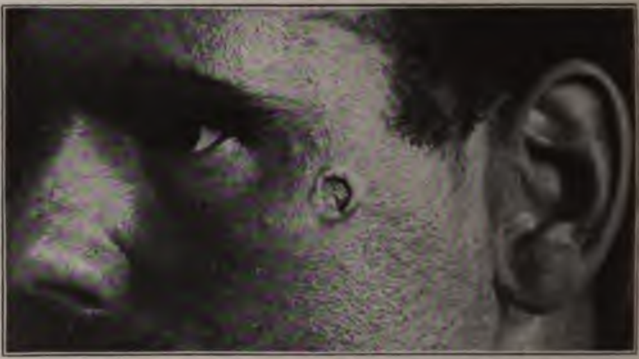
[1] Trans. Sec. on Derm., Amer. Med. Assoc., 1910, p. 119; ibid., 1908, p. 120; "Some of the more unusual forms of epithelial growths of the skin."

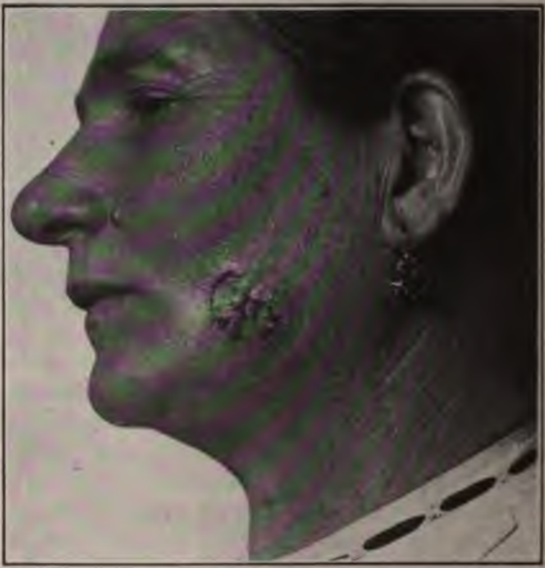
Fig. 234



Epithelioma of the cheek.

an ulcer characterized by marked induration of its base and walls. The floor is irregular and may present papillomatous and fungoid masses, and secretes a viscid, hemorrhagic or purulent fluid. The edge may be everted, undermined, thickened and indurated, and here waxy nodules are often seen. These ulcers often extend rapidly, both peri-

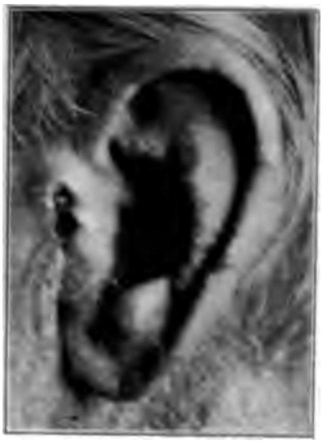
Fig. 235



Flat cicatricial epithelioma of morphea-like character.

pherally and deeply, inducing marked local destruction. It is much more rapid in its progress than the superficial varieties and is followed by involvement of the lymphatic glands, with general metastases, and frequently by a fatal termination.

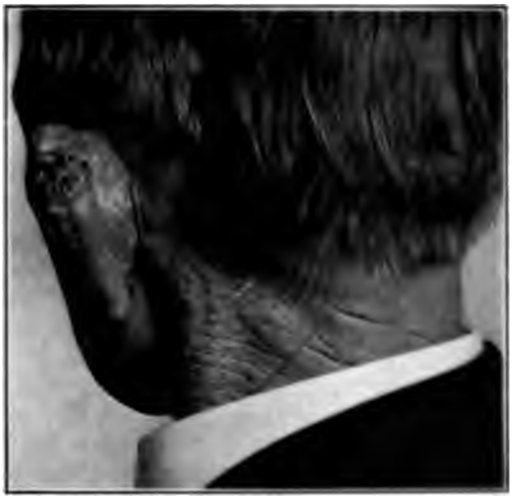
FIG. 236



Epithelioma of the ear.

Papillary Epithelioma. — Papillary growths may occur in connection with the types above described or they may originate as such. When primary a verrucous or papillomatous nodule occurs which presents a moderately infiltrated base and is superficial. Subsequently variously sized lesions occur, which are usually sessile but may be pedunculated. In a fully developed tumor the papillomatous lesions present a cauliflower appearance and secrete a viscid, sanguineous, and not infrequently foul smelling, fluid which may form crusts when produced in moderate amounts. These lesions bleed readily. Fissuring and ulceration follow, the ulcers being either superficial or deep. In time the process infiltrates the deeper tissues and by metastatic extension the glands or internal organs become involved.

FIG. 237

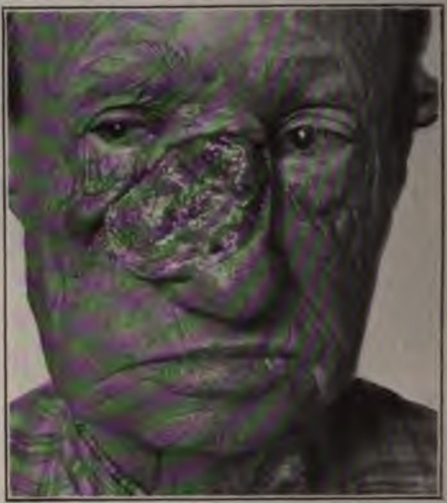


Epithelioma of the ear.

In some cases the epithelioma forms a soft hemispherical nut- to egg-sized tumor which, upon pressure, discharges numerous convoluted plugs composed of epithelial débris, fatty masses and a purulent secretion.

A special type of rodent ulcer was described by Hutchinson under the title *crateriform ulcer*, which is distinguished by its rapid development and by its implication of the glands. It may develop upon the ordinary form, or its onset may be exhibited by the formation of a roundish or conical mass which rapidly exhibits ulceration forming a central crater, with a markedly infiltrated base and walls.

Fig. 238



Rapidly progressing epithelioma of the nose.

Epithelioma of the Lip.[1]—Men are more frequently attacked than women[2] and the lower lip is the common site. The early lesion may be a small nodule, a circumscribed keratosis, a fissure or warty area, or a small abrasion which may be crust-covered. Induration and ulceration following the ulcers have varying characteristics and papillomatous over-growths are of frequent occurrence. In some instances these may produce fungoid masses. Epithelioma of the lip is usually of the spino-cell type and metastases occur early in some instances, followed by the usual consequences of such invasion. Some of these develop slowly and occasionally a benign type occurs.[3]

Carcinoma of the tongue and other mucous membranes of the mouth sometimes develops on an area of leukoplakia, occasionally on a gumma, and is very frequently induced through irritation of a defective tooth. The resulting growths exhibit variously sized and outlined ulcers, which frequently metastasize early and are surgical affections.

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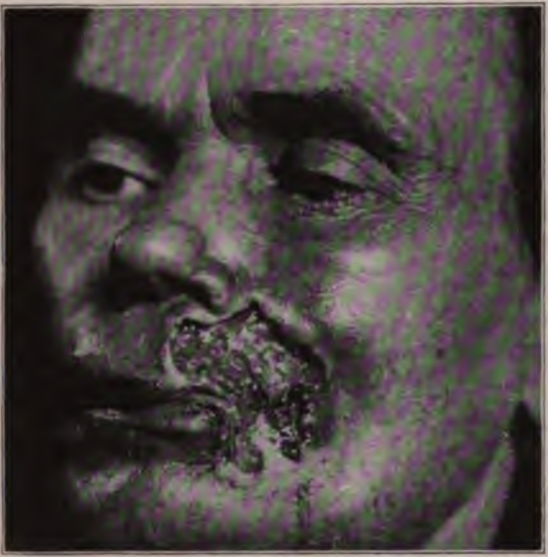
[1] Broders, Jour. Amer. Med. Assoc., 1920, lxxiv, p. 656; Squamous-cell Epithelioma of the Lip. A comprehensive and thorough study of the subject in all its aspects based on 537 cases.

[2] Wise, New York Med. Jour., September 27, 1913; "Epithelioma of the Lower Lip in a Woman;" bibliography. Idem., Jour. Cut. Dis., 1917, xxxv, p. 560; Quinn, ibid., 1915, xxxiii, p. 400; "Epithelioma of the Lip in a Woman."

[3] *Cf.* Pusey, 1913, xxxi, p. 73. Review of rodent ulcer of the lip.

FIG. 239

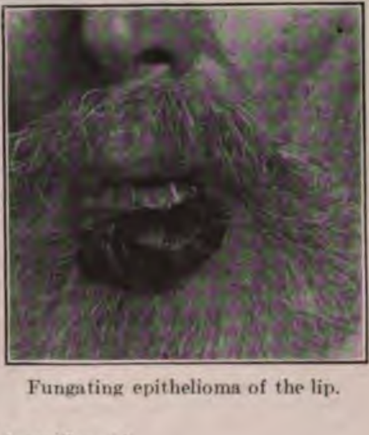


Carcinoma of lip.

On the genitals the papillary variety frequently occurs and begins on the glans, prepuce, or clitoris. While these growths sometimes pursue a mild course for a time, they not infrequently metastasize early and, like the lesions in the mouth, are surgical affections.

On the extremities epithelioma is usually of the papillomatous type. That on the dorsa of the hands may begin in a keratosis, or follow a roentgen-ray dermatitis or an injury. These are usually spino-cell epitheliomata and therefore metastasize and are dangerous. Exceptions occur. Howard Fox,[1] the author[2] Bunch[3] and others have recorded cases following an injury. In Fox's case the epithelioma followed a horse-bite; the lesion exhibited a serpiginous ulcer, a crusting

FIG. 240



Fungating epithelioma of the lip.

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[1] Jour. Cut. Dis., 1914, xxxii, p. 50; ibid., 1915, xxxiii, p. 22.

[2] Diseases of the Skin, 1915, p. 677.

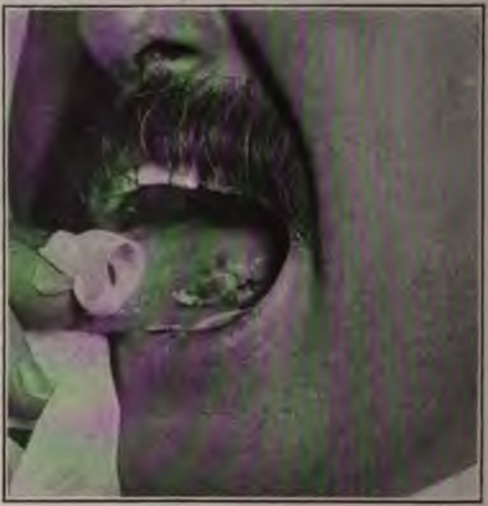
[3] Brit. Jour. Derm., 1917, xxix, p. 114; "Epithelioma of Hand following Traumatism."

surface, and the disorder terminated fatally with metastases. The author's case was similar and resembled blastomycosis in its papillomatous development.

The sites or election of epithelioma are the face, particularly about the eyelids, nose and temples. The ears[1] and scalp are less often involved, and still less frequently the hands, genitals, neck and other areas. The lip is a common site in men.

The course of epithelioma varies with the type. The flat, superficial forms are chronic, lasting for years at times, as comparatively insignificant growths. These lesions may undergo no particular change during a period of ten to twenty years. A bean-sized, pearly growth situated on the nose had been present for fifteen years in one of the author's cases, and was only treated then because of an ulcerative epithelioma

FIG. 241



Epithelioma of the tongue.

occurring on another portion of the face. Eventually, ulceration
occur and produce destructive changes unless properly treated.
infiltrating types and deep forms pursue a more rapid course than
of the basal-cell type, metastasize and are grave.

The form generally seen by the dermatologist is the superfi
comparatively benign basal-cell type epithelioma and is usually situ
on some portion of the face.

Etiology.[2]—The essential cause of carcinoma is unknown;
factors enter as predisposing or contributory causes. Among

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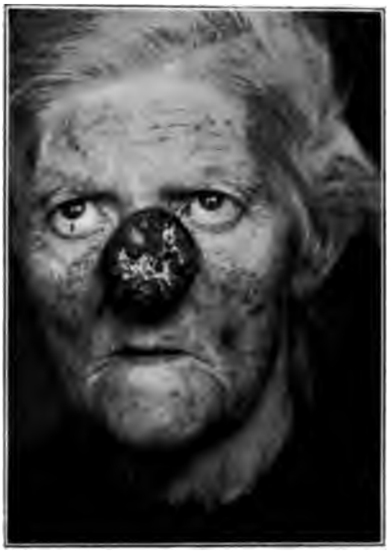
[1] Sutton, Jour. Amer. Med. Assoc., 1920, lxxiv, p. 88. Carcinoma of the externa
In five cases studied microscopically the basal-cell type was found.

[2] Harris, Jour. Cut. Dis., 1918, xxxvi, p. 67; "The etiology and pathology of
cancer." (Read in a symposium on skin cancer before the American Dermatolo
Association at its forty-first annual meeting.) *Cf.* this excellent article for revi
the various theories as to etiology. Extensive bibliography. Williams, Archi
Derm. and Syph., 1920, ii, p. 168; malignant degeneration in benign dermatoses.

trauma, both mechanical and chemical, is important. Any long-standing irritation may lead to epithelioma.[1] This is illustrated in the case of the smoker, the worker in tar[2] and paraffin, the chimney sweep, the sailor exposed to the sun and wind and, occasionally, the roentgen-ray operator. Of 1189 cases of epithelioma occurring in the Kashmir Mission Hospital, reported by Neve,[3] 848 were of the thighs or abdomen and resulted from trauma and burns induced by the kangri, a portable fire-box used by the natives.

Warts and nevi, when irritated or otherwise, may undergo epitheliomatous transformation. Numbers of cases have been recorded of epithelioma following lupus vulgaris. Sequeira[4] found epithelioma as a sequence in lupus vulgaris in 1.5 per cent. of 964 cases. Epithelioma developed in 10 men out of a total of 287, or 2.87 per cent.; while in women only 4 developed this complication out of a total of 677, a percentage of .59 per cent. The average duration of the lupus before the development of the epithelioma was thirty-five years. The lesions were situated chiefly on the cheeks and the neck. In clinical type 8 were nodular and 6 ulcerative, and histologically they were all squamous-celled, with characteristic nests. Others recording examples of epitheliomata following lupus vulgaris are Bargues,[5] Adamson,[6] MacKee[7] and E. P. Zeisler.[8] Epithelioma occurring with lupus erythematosus is reported by Pollitzer,[9] Reyn,[10] Schwarz,[11] and several others.

Fig. 242



Epithelioma.

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[1] Montgomery, Jour. Cut. Dis., 1918, xxxvi, p. 529; "Etiology of Cancer of the Lip."

[2] Zweig, Zeitschrift, 1909, xvi, p. 85; abstr. Brit. Jour. Derm., 1909, xxi, p. 190. (Trade cancers: report of two cases in workers in coal tar, began as warts on the face and scrotum.) Schamberg, Jour. Cut. Dis., 1910, xxviii, p. 644; cancer in tar workers, with good bibliography. O'Donovan, Brit. Jour. Derm. and Syph, 1920, xxxii, pp. 215 and 245, epitheliomatous ulceration among tar workers.

[3] Brit. Med. Jour., September, 1910.

[4] Brit. Jour. Derm., 1908, xx, p. 40; with references.

[5] Annales, January, 1910, p. 3; reporting 6 cases and analyzing the literature of 164 others.

[6] Brit. Jour. Derm., 1911, xxiii, p. 246.

[7] Jour. Cut. Dis., 1914, xxxii, p. 390; ibid., 1914, p. 446.

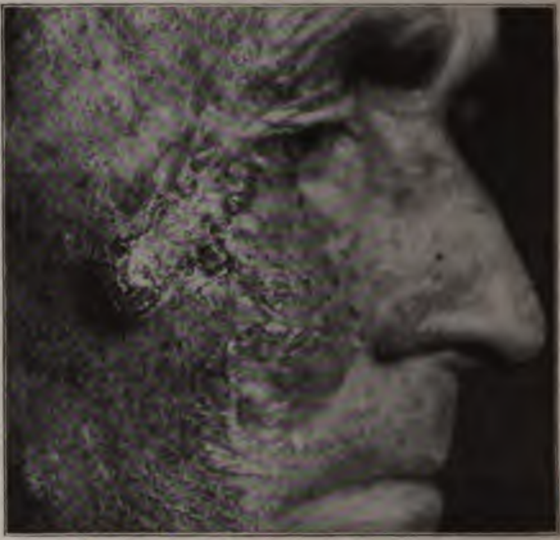
[8] Ibid., 1919, xxxvii, p. 62; ibid., p. 262; same patient showing excellent results of treatment.

[9] Ibid., 1910, xxviii, p. 140.

[10] Nord. Med. Arkiv., 1911, abstr. ii, p. 49; abstr., British Jour. Derm., 1912, xxiv, p. 375.

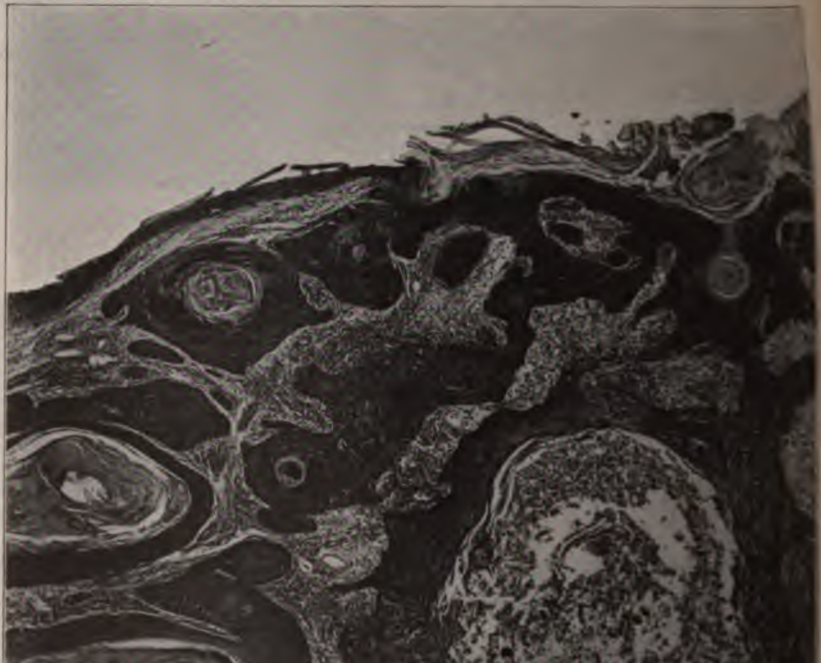
[11] Ibid., 1918, xxxvi, p. 47; "Lupus erythematosus and epithelioma of the lip."

FIG. 243



Multiple carcinomata, with diffuse precancerous hyperkeratosis.

FIG. 244

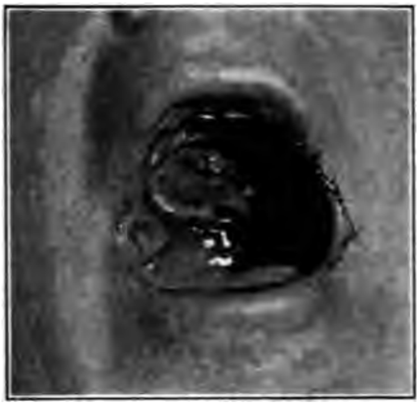


Section from a small tumor from the same patient.

As occurring in psoriasis by Hartzell,[1] Whitfield,[2] Fordyce,[3] Gray,[4] the author, and others.

The disease not infrequently develops in the scars of syphilis and burns. Senile keratoma, which is frequently encountered in persons exposed to the weather, is a common starting point of epithelioma in the aged, and is the usual origin of multiple epitheliomata of the hands and face.[5] Arsenical hyperkeratoses occasionally are followed by epitheliomatous transformations. Such cases have been recorded by Hartzell,[6] Schamberg,[7] Weidenfeld[8] and several others. A group of such cases have been under the observation of the author, in one of which more than a dozen superficial or more deeply situated epitheliomatous ulcers occurred, together with hundreds of hyperkeratotic and hyperpigmented lesions. In this instance arsenic had been taken over a period of twenty years. In another patient plantar and palmar hyperkeratosis, with individual hyperkeratotic areas on the fingers and dorsal surface of the hands, scrotum, and other areas were present. From time to time epitheliomatous growths occurring upon these lesions were excised and their nature demonstrated. The epitheliomata occurred chiefly on the hands, limbs and scrotum.

FIG. 245



Carcinoma of the hard palate.

Heredity as an etiological factor is receiving more attention through the experimental work being done with mouse tumors. Loeb[9] and others have shown that not only are there strains of mice peculiarly susceptible to inoculation with a given tumor, but also there are strains from 30 to 50 per cent. of the offspring of which develop spontaneous tumors. In her remarkable work with mice Maud Slye[10] has not only proved the inheritability of cancer in animals, but also the fact that cancer can by proper breeding be increased or entirely elimi-

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[1] Trans. Amer. Derm. Assoc., 1899, p. 111, a series of cases.

[2] Brit. Jour. Derm., 1906, xviii, p. 40; "Rodent ulcer in a patch of psoriasis."

[3] Loc. cit.

[4] Brit. Jour. Derm., 1912, xxiv, p. 325; "Rodent ulcer on a patch of psoriasis in the gluteal region."

[5] Sutton, Jour. Amer. Med. Assoc., 1915, lxiv, p. 403; "The symptomatology and treatment of seborrheic keratosis."

[6] Amer. Jour. Med. Sci., 1899, cxviii, p. 265.

[7] Jour. Cut. Dis., 1909, xxvii, p. 130.

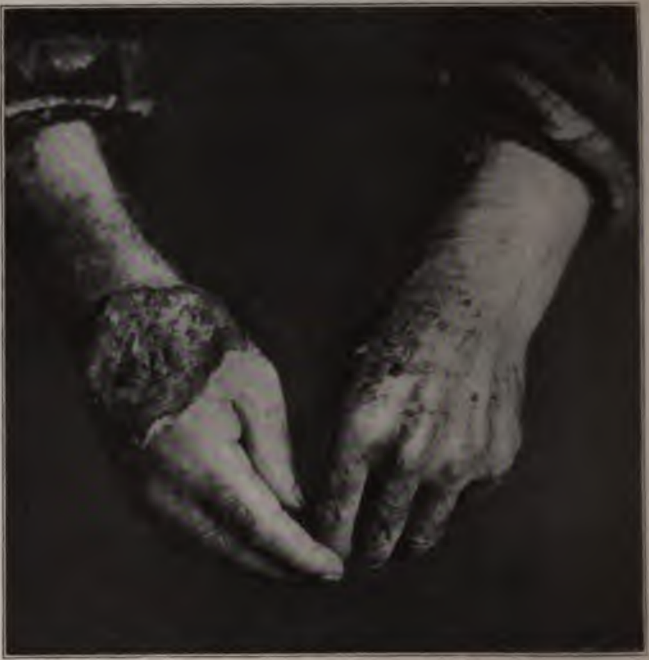
[8] Archiv, 1913, cxi, p. 467; about seventy tumors were present in this case.

[9] Zeitschr. f. Bakt., 1912, lxvii, p. 133.

[10] Jour. Cancer Research, 1920, v, pp. 25 and 53; *Cf.* these articles for a comprehensive report of extensive and carefully conducted experimental work.

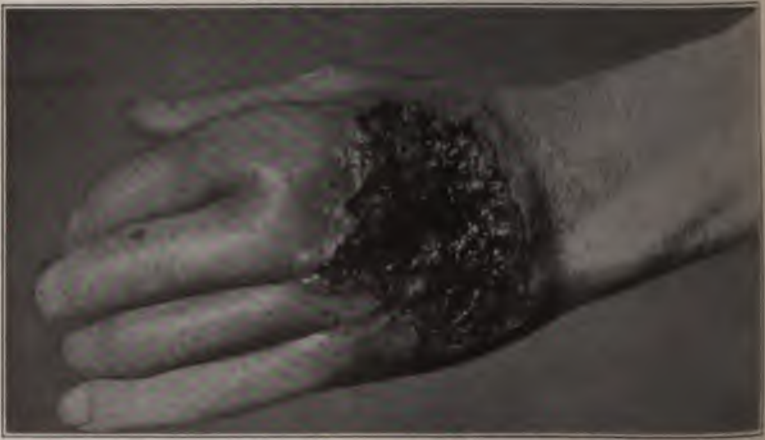
nated in particular strains. Warthin,[1] in an analysis of 1600 ca
found facts pointing to the importance of hereditary influence. P

FIG. 246



Carcinoma and precancerous keratoses.

FIG. 247



Carcinoma following injury, resembling blastomycosis. (Prickle-cell type.)

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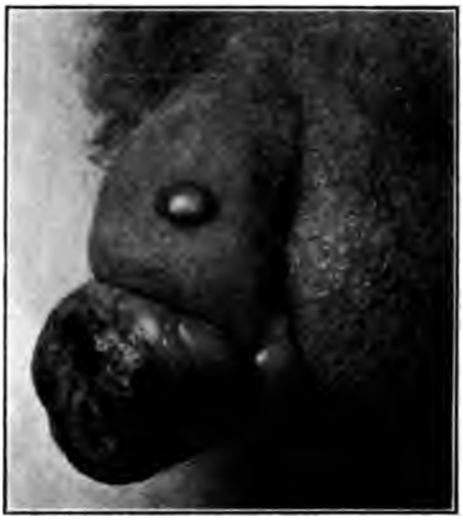
[1] Archives of Int. Med., November, 1912.

[2] Jour. Amer. Med. Assoc., 1912, p. 557.

also found earlier development and greater malignancy in children of cancerous parents.

The disease is one of advanced years, the average age of onset being about fifty-two. It is a pathological curiosity in childhood, but Kaposi has reported a case occurring in the tenth year. Sequeira[1] recorded the case of a boy aged twelve with a rodent ulcer on the back, and quotes 2 others, 1 aged twelve and 1 aged fifteen, also seen by him. Adamson[2] reports an epithelioma occurring in the scar of lupus in a girl aged thirteen. Only about 30 per cent. of all cutaneous cases occur in women, which is possibly due to the fact that women are less exposed to the action of irritants. The disease is more common among northern people than among those of the tropics. It is relatively rare in the black races. Hazen[3] reported the case of a negro suffering with an epithelioma of the lip. The principal cause of this immunity, as maintained by Hyde and others, is the protection afforded by the pigment in the colored skin. Brault,[4] however, observed 28 cases among the natives of Algiers during eight years. He thinks this large number argues against the older opinion in regard to the rarity of the disease in hot countries. The occurrence of cancer in those exposed to the sun's rays without proper protection has been emphasized by Hyde,[5]

FIG. 248



Carcinoma of glans penis with lenticular nodules of shaft.

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[1] Brit. Jour. Derm., 1912, xxiv, p. 391

[2] Royal Soc. Med., 1911.

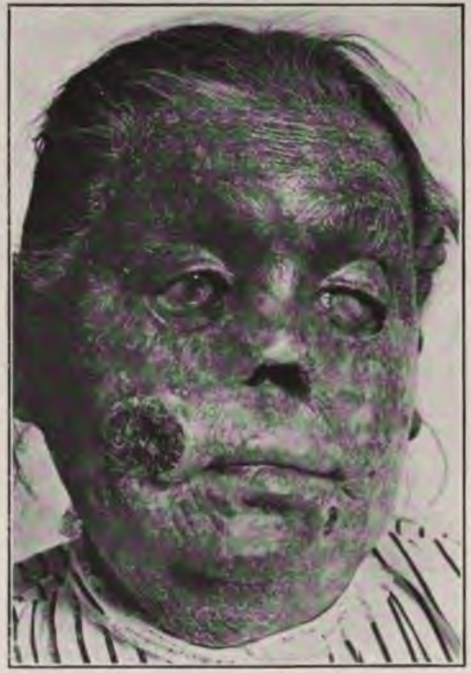
[3] Jour. Cut. Dis., 1911, xxix, p. 321.

[4] Prov. med., 1912; abstr. Derm. Wchnschr., liv, p. 740.

[5] Amer. Jour. Med. Sc., 1906, cxxxi, p. 1.

Dubreuilh,[1] Corlett[2] and McCoy.[3] A review of the above facts indicates that no single causative factor could account for the development of all epitheliomas, a view held by most investigators. None of the various theories concerning cell inclusions, the proper relation of growth and functional activity, nor the congenital tendency of cells, is sufficient. The search for a parasite has been largely superseded by investigative work in other lines.

FIG. 249



Epithelioma occurring in a lupus vulgaris scar.

Pathology.[4]—The pathology of cutaneous carcinoma is not understood. Opinions differ among those who have done the most work concerning not only the starting point of the growths, but the various factors concerned in their future development. Ribbert[5] believes the primary and important change in roentgen and tar carcinoma occurs in the connective tissue.

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[1] Annales, 1907, viii, p. 387; abstr., Brit. Jour. Derm., 1908, xx, p. 62; "Epitheliomatosis of solar origin."

[2] Jour. Cut. Dis., March, 1915, xxxiii; "Xeroderma pigmentosum following severe sun exposure, with report of two cases." Paper read before thirty-eighth annual meeting of the American Dermatological Association, May, 1914.

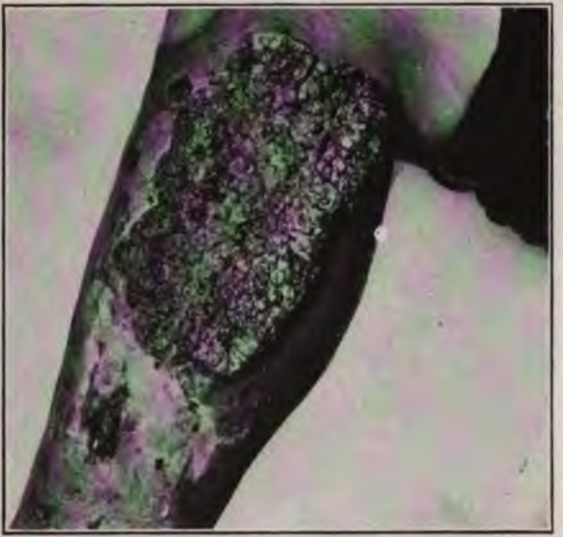
[3] Archives of Dermatology and Syphilis, 1920, xxxviii, n. s. i, p. 175.

[4] Harris, Jour. Cut. Dis., 1918, xxxvi, p. 67; "The etiology and pathology of skin cancer." (An extensive review of the various theories concerning the pathogenesis of cancer.) Fordyce, Trans., Sec. on Derm., Amer. Med. Assoc., 1908, p. 120; ibid., 1910, p. 119.

[5] Fortschr. auf Derm., gibiente Roentgenstrahlen, 1915, xxii, p. 473; abstr., Jour. Cut. Dis., 1916, xxxiv, p. 853; "The histopathogenesis of Roentgen and tar cancers."

The major portion of epitheliomata may be classed in two groups: the squamous or prickle-cell and the basal cell type. In the former the new growth follows trauma and other external irritations, such as that occurring in paraffin and tar workers and chimney sweeps. It develops on senile warts, in scars, in association with lupus vulgaris and lupus erythematosus, and is the common variety found on the lip and mucous membranes. The histology shows a down-growth of epithelium, with branching processes extending in various directions. Whorls of cornified epithelium are seen, constituting the cell nest. In the center of such a nest some hyalin-degenerated cells occur, together with cell débris, around which are concentric rings of cornified cells. Outside of these are variously shaped and sized prickle-cells. In sections, islands of epithelium occur, owing to the branching processes being seen on cross-section. The corium is edematous and presents an infiltration of plasma-cells and lymphoid-cells. The elastic tissue is destroyed in the area of infiltration. In the basal-cell type[1] the new growth may originate from the basal-cells of the epidermis or similar cells of the hair follicle or sweat glands.[2] The growth is composed of cylindrical processes which progress downward

Fig. 250



Squamous-cell epithelioma developing at the seat of an old scar.

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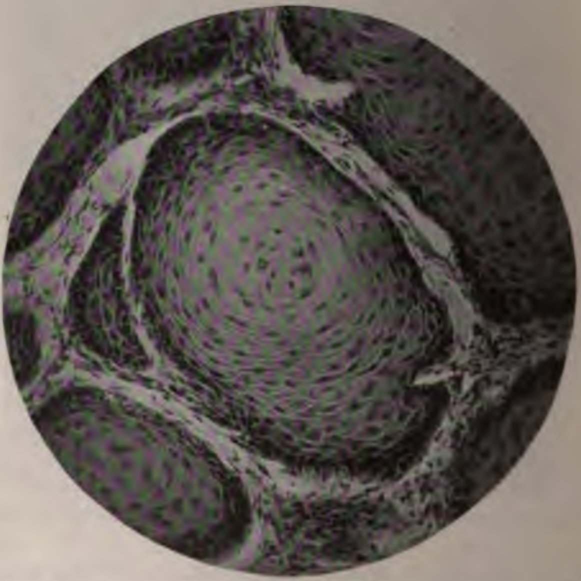
[1] *Cf.* Article of Broders, Jour. Amer. Med. Assoc., 1919, lxxii, p. 856; "Basal-cell epithelioma." An analysis of a large number of cases treated at the Mayo Clinic, giving location of lesions, histology and results of treatment.

[2] Adamson, Brit. Jour. Derm., 1917, xxix, p. 81; "Basal-cell epithelioma with zoniform distribution and the possibility of sweat-gland origin."

and laterally, composed of oval and round cells having a relatively large, deep-staining nucleus and a small amount of protoplasm. These cells are often closely packed, are hard to differentiate, and as a rule prickles cannot be demonstrated. The abnormal cornification process noted in the squamous-cell type is not present here but cystic degeneration sometimes occurs, producing cavities containing fluid. Occasionally this is of sufficient degree to produce clinical manifestations.[1] The tendency is for lateral extension of the infiltration and the connective tissue is preserved to a greater degree than in the former type. The cellular infiltration in the corium is less pronounced and the destruction of elastin is also of a milder degree. Involvement of the neighboring glands does not occur in this variety. Adamson[2] believes this type belongs to the class of nevoid growths.

Fig. 251



A cancer of the leg, of the prickle- or squamous-cell type. Photograph shows cross sections of epithelial processes which have a concentric arrangement, their centers undergoing pearl-formation. (Fordyce.)

Treatment.[3]—The treatment of carcinoma of the skin consists in removal of the growth either by excision, curettage and cauterization, or by the use of x-rays or radium. In the type of cases usually seen by the dermatologist x-rays or radium are most commonly employed. In certain instances curettage followed by cauterization, or cauterization alone, is preferable. Where the growth involves the tissues deeply about the cartilages of the ear it is very resistant to x-rays, and in the majority of such cases excision followed by radiotherapy is probably the best procedure. In all lip cases and those involving the mucous membranes, and the deeply situated prickle-cell type surgical methods should be employed. It is unnecessary to reiterate the necessity of thorough removal of the growth, including the near-by glands, whether they are clinically involved or not. In inoperable cases radiotherapy may also be used for the temporary alleviation of pain and possible checking of the progress of the growth. In 1899, Stenbeck,[1] of Stockholm, exhibited a patient from whom he had removed a rodent ulcer by the use of x-rays, following which the method has been very largely used in the treatment of certain types of epithelioma. The results attained in our experience with x-rays are satisfactory and

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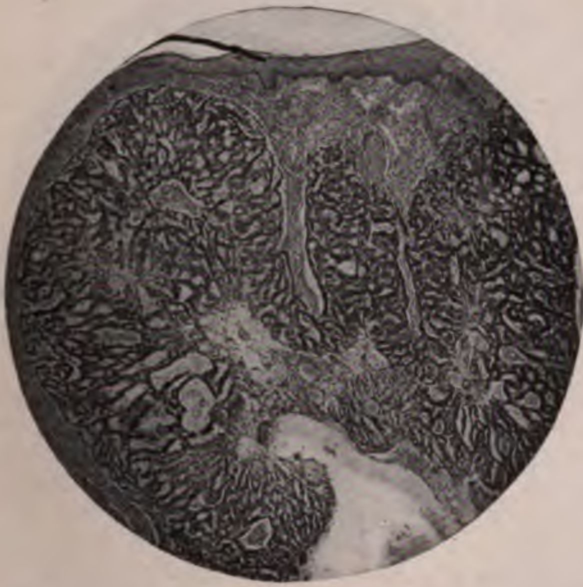
[1] MacLeod, Brit. Jour. Derm., 1909, xxxi, p. 349.

[2] Lancet, London, 1914, clxxxvi, p. 810.

[3] Bloodgood, Trans., Sec. on Derm., Amer. Med. Assoc., 1910, p. 144; The surgical treatment of cutaneous malignant growth. Pusey, Ibid., p. 174; Treatment of malignant growths of the skin from a dermatological standpoint. Idem., Jour. Amer. Med. Assoc., 1913, lxi, p. 552; What can be done in cancer with roentgen rays. MacKee, Jour. Cut. Dis., 1918, xxxvi, p. 92; The treatment of cutaneous cancer. (This paper was read as part of a symposium before the American Dermatological Association at its Forty-first Meeting. An excellent presentation of modern methods of treatment.)

Fig. 252



Cylindroma.

An epithelioma of the basal-cell type removed from the face. Hyalin degeneration of the interior of the tumor has resulted in the lace-like appearance. The tumor cells are small, with chromatic nuclei and very little protoplasm. The connective tissue shows an inflammatory reaction. (Fordyce.)

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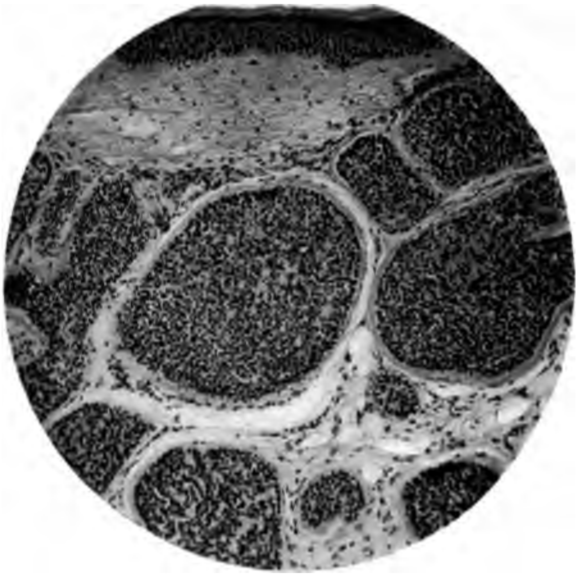
[1] Pusey: The Röntgen rays in therapeutics. Williams: The Röntgen rays in medicine and surgery. Freund, Grundriss der Gesammten Radiotherapie, Vienna, 1903. Allen, Jour. Cut. Dis., 1903, xxi, p. 75. Sequeira, Brit. Med. Jour., June 6, 1903. Hyde, Montgomery and Ormsby, Jour. Amer. Med. Assoc., January 3, 1903. Sir Malcolm Morris, Brit. Jour. Derm., 1912, xxiv, p. 169.

compare favorably with the recorded percentage of recoveries by other methods. The technic depends upon the type of growth and the apparatus employed. The amount of *x*-rays necessary to destroy the growth may be given in a single treatment[1] or in divided doses. The chief advantages of radiotherapy lie in its painless application and excellent cosmetic results. It should be the method of choice in all superficial cases which, owing to location or to the size of the area involved, cannot be treated surgically without conspicuous disfigurement. The treatment is of special value in diffused hyperkeratoses growing in senile skins and showing beginning malignant changes. For practically all superficial lesions the method is satisfactory, but the time required is more than in simple excision. In deep-seated tumors radiotherapy is often successful if the growth be fully exposed to the surface. It is better to treat such lesions surgically and follow this with radiotherapy. Radium acts similarly to the *x*-rays, but has the disadvantage of being difficult to procure in large enough quantity to secure the best results.[2]

FIG. 253



Cylindroma of the scalp.

The cells of this tumor are small and closely resemble those of the basal layer of the epidermis. Inside the cellular aggregations hyalin degeneration has taken place in the form of small cylindrical areas, from which the growth derives its name. Immediately surrounding the cell-masses the connective tissue has likewise undergone degeneration, appearing as a narrow, homogeneous band of hyalin. (Fordyce.)

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[1] Jour. Cut. Dis., 1911, xxix, p. 503.

[2] Wickham and Degrais, Radium-thérapie, Paris, 1900. Simpson, Trans., Sec. on Derm., Amer. Med. Assoc., 1913, p. 30. Clark, Radium treatment of cutaneous epitheliomas by single or massive doses, Jour. Amer. Med. Assoc., May 9, 1914, lxii, p. 1453. Engman and Kimbrough, Urological and Cutaneous Review, January, 1919, p. 1; Radium in the treatment of basal-cell cancer.

While excision, *x*-rays and radium are the methods of choice, certain others have been found of value. These consist in cauterization, or combined curettage and cauterization. Among other caustic substances which may be employed are zinc chlorid, arsenical paste and pyrogallol. The latter is highly recommended by Kaposi, not only because its application is unproductive of pain, but also because it does not attack sound tissue. It is used in an ointment of 10 per cent. strength. Among the formulæ used for caustic purposes is the following:

℞—Opii pulv.,	grs. ij:	133	
Arseni trioxidi,	grs. iv;	266	
Creasoti,	℥ss;	15	M.

Sig.—For employment upon circumscribed surfaces (Kaposi).

Marsden's paste, also employed as a caustic, is made by combining equal parts of gum arabic and arsenic trioxid with water sufficient to make a solid paste. By Robinson[1] it is preferred to others and is applied on rubber plaster.

Cosmé's paste, as modified by Hebra, is prepared as follows:

℞—Arseni troxidi,	grs. vj;	40	
Hydrarg. sulphid. rub.,	ʒss;	2	
Unguent. aq. ros.,	℥ss;	15	M.

Sig.—Arsenical paste for external use, with caution.

All such pastes and ointments should be spread upon cloths and be applied for from three to six days. Opiates may be required in the case of several of these agents to relieve the consequent pain. The method of application of Cosmé's paste is as follows: The paste is spread over a thin sheet of lint to the thickness of a knife-blade, and the lint is then cut to a shape and size corresponding with those of the tumor or ulcer to be destroyed. After their close apposition with the surface to be attacked, the lint and paste should be covered with gutta-percha or other impermeable tissue and a compress laid over the whole. In twenty-four hours the dressing is removed, the parts washed clean, and the same application renewed. By the third or the fourth day the destruction of the cancerous growth is usually complete, and the parts are ready for an emollient poultice, which should be applied for the three or four days during which separation of the slough occurs. The simple ulcer left is to be treated on general principles. The danger of arsenical poisoning is here reduced to a minimum; and the treatment is very effectual where patients consent to the delay and to the severe pain which the treatment occasions.

The thermo- and galvano-cautery may also be often advantageously used for destruction of the growths. The advantages of the thermo-cautery are: The transitory character of the induced pain; the coal-like dressing left upon the attacked surface; and the good cosmetic result. Both measures find their highest value when employed after excision or erasion.

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[1] Internat. Jour. Surg., 1892, p. 179; Treatment of Cutaneous Malignant Epitheliomata.

Not infrequently a combination of two methods will give better results. Sherwell[1] has had remarkably good results with curettage followed by cauterization with the acid nitrate of mercury. Culver[2] used a combined method of curettage, cauterization with chromic acid and *x*-rays with good results. Pfahler[3] recorded excellent results with the use of fulguration followed by radiotherapy. Montgomery and Culver[4] recommend *x*-rays or radium as a preliminary treatment before employing curettage or other methods in certain cases.

Prognosis.—The superficial basal-cell type usually responds to treatment. The deep prickle-cell type is always serious, though many are eradicated by prompt and efficient treatment.

PAGET'S DISEASE.

Synonyms.—Eczematous epitheliomatosis of the nipple, Malignant papillary dermatitis, Cutaneous psorospermosis.

Definition.—Paget's disease is a carcinomatous disorder in the skin with inflammatory symptoms.

This disease was first described in 1874 by Paget[5] and has since been especially studied by a number of English, French and American observers including Thin, Duhring, Malassez, Darier, Wickham, Jackson[6] and others.

Symptoms.—The disorder occurs, as a rule, on the breast in women, but extramammary cases are occasionally seen. The process begins with moderate inflammation, exhibited as redness and scaling, involving the nipple and areola. Later the surface becomes intensely red and granulating exuding a clear, viscid secretion, and produces subjective sensations of heat and burning, with intense or moderate itching. The inflammatory area is well-defined and gradually becomes indurated as the process extends deeply. A prominent feature of the disease is the circumscribed infiltration of the skin and subcutaneous edge which, on palpation, suggests a large sized coin or button in the substance of the areola and surrounding parts. Sooner or later a carcinomatous infiltration of the breast occurs, but this may not be recognized until after extirpation of the latter. The course of the disease is usually slow, but in certain instances the malignant transformation occurs rapidly. In all cases there is no attempt at healing of the apparently eczematous process. Upon the development of carcinoma in the breast the disease runs the ordinary course of such involvement.

Cases of Paget's disease affecting other parts of the body have been reported by a number of observers, including Fordyce and Fox, MacLeod, Hartzell,[1] and others. By these various observers the disease has been seen on the scrotum, the penis, the buttocks, the vulva, and perineum, in the pubic region, about the umbilicus, in the axilla,[2] on the nose, and other areas.

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[1] Jour. Cut. Dis., 1910, xxviii, p. 487.

[2] California State Jour. Med., 1911, ix, No. 8, p. 340.

[3] Jour. Cut. Dis., 1915, xxxiii, p. 225; "Cases of Epithelioma Treated by Fulguration." Six cases clinically cured.

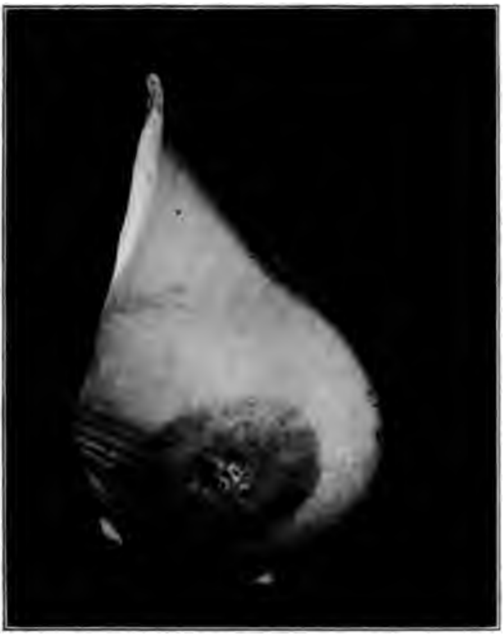
[4] Ibid., 1917, xxxv, p. 837; "The Preoperative Reduction of Epithelioma by Radiotherapy or Radium."

[5] St. Bartholomew's Hospital Reports, 1874, p. 87.

[6] Jour. Cut. Dis., 1896, xiv, p. 428; ibid., 1903, xxi, p. 201.

The disease usually occurs in women past forty years of age, and commonly attacks only one breast, the right being more frequently involved. No definite cause has been discovered. Darier discovered the peculiar cells described below and thinking they were coccidia, ascribed the disease to their presence. The connection between the malignant growth of the breast and the dermatitis which precedes it is not settled. Whitfield[3] and Hartzell[4] consider the disease carcinomatous from the beginning. The pathology of the disease in the mammary region has been studied by Butlin, Unna[5] and others, and in the extra-

FIG. 254



Paget's disease of the breast.

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[1] Jour. Cut. Dis., 1910, xxviii, p. 379; Extra-mammary Paget's Disease, with report of a case occurring on the forearm associated with melanocarcinoma; with analysis of 18 cases in the literature.

[2] Satani, Brit. Jour. Derm. and Syph., 1920, xxxii, p. 117, a case of extra-mammary Paget's disease occurring in the axilla, associated with condylomata acuminata in the external genitalia. References to previously recorded cases.

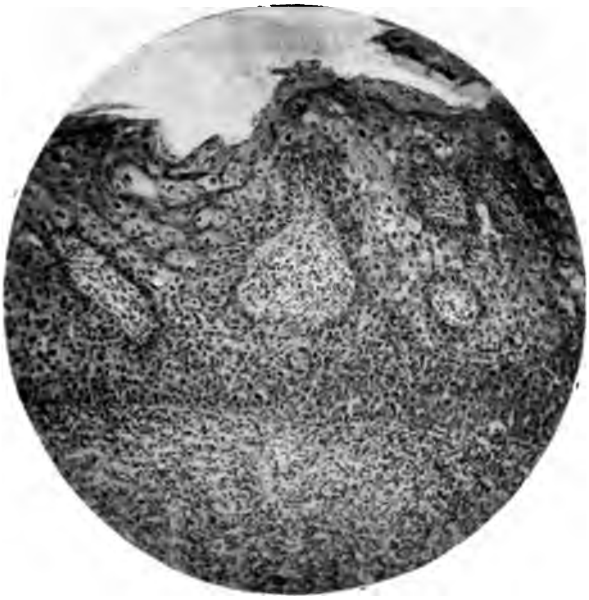
[3] Allbutt's and Rolleston's System, 1911, ix, p. 603.

[4] Diseases of the Skin, 1919, p. 607.

[5] Histopathology, p. 737 An independent description of the pathology of the disease, with review of the work of Butlin, Thin, Duhring, and Wile, Darier, Wickham, Hutchinson, Jr., and others.

mammary regions by Fordyce and Fox,[1] Fox and MacLeod,[2] Hartzell, and others. The characteristic histologic changes consist in edema and hypertrophy of the epidermis, in which peculiar cells are found, together with inflammatory cellular infiltration in the corium. In the epidermis there is edema, producing enlargement and other changes in the epithelial cells. Marked hypertrophy of the rete occurs, producing thickening of varying grades, and a down-growth of the rete pegs into the corium. Peculiar double contoured cells of large size are found in varying numbers. These cells may be round or irregular in shape and often contain two or three nuclei, which show varying changes as to form, size and location. These cells, through their resemblance to coccidia or psorosperms, were formerly considered by Darier to be these organisms and his work was confirmed by Wickham, but all observers now agree that they are cellular degenerations. In the corium a dense cellular infiltration occurs. This usually begins around dilated bloodvessels and occurs as collections of cells as well as in diffuse areas. It is composed of leukocytes and small connective-tissue cells, plasma cells and some mast cells, and in the area of infiltration the collagen and elastin are more or less destroyed. The retraction of the nipple is

FIG. 255



Paget's disease of the buttock, showing the changes which take place in the epidermic cells. They are very much swollen from edema, vascuolated, and have their nuclei pushed to one side. Many of them have lost their prickles. In the corium there is a dense infiltration of lymphocytes and plasma cells. (Fordyce.)

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[1] Jour. Cut. Dis., 1905, xxiii, p. 193.

[2] Brit. Jour. Derm., 1904, xvi, p. 41.

due to a sclerosis occurring around the mammary ducts, the latter being involved in malignant infiltration early in the process in some cases. The subsequent carcinoma may develop either from these ducts or glands beneath or from the surface of the epithelium. The histology of the extramammary cases corresponds to the above in most particulars. In some instances the cellular exudate in the corium is less marked The presence of the Paget cell and other characteristics identify the condition histologically.

Treatment.—In the breast cases operative procedure is indicated and should be done at the earliest possible moment. The usual thorough surgical operation, including extirpation of the glands, is recommended. A deep carcinomatous change may be present when its clinical demonstration is not possible. If, for any reason, operative procedure is impossible *x*-rays may be employed. The local treatment suggested for eczema has no effect upon the inflammatory process occurring in this condition. In the extramammary cases it is agreed by most observers that *x*-rays or radium give the best results.

Prognosis.—If the condition is seen and treated early by prompt removal of the growth, recovery takes place. In several cases so treated with *x*-rays by the author complete recovery has resulted. When the disorder involves the breast proper the prognosis is that of mammary cancer.

PRECANCEROUS DERMATOSES.[1]

Under the above title Bowen, in 1912, described two cases of this unrecognized type of epithelial new growth. Subsequent reports have been made by Darier, Bowen, Morrow and Lee. The disorder is chronic and while described as a precancerous condition in nearly half of the recorded cases cancerous transformation has occurred. The lesions occur in well-defined patches. The early lesion is a firm, pale red papule, covered with a thickened horny layer or a cornified crust. By enlargement of the papule lenticular or rounded nodular lesions are formed which may be discrete or grouped and confluent. Beneath the crust the surface may be red and oozing and granular or slightly papillomatous. The patch increases in size by the peripheral formation of new lesions.

In a histological study Bowen found proliferation of the rete pronounced. The cells were undergoing both direct and indirect division and many presented peculiar clumping of the nuclei and vacuolization of the cell. Hyperkeratosis, parakeratosis, and edema were present. The vessels in the papillary and deeper layers of the corium were dilated and surrounded by masses of plasma cells. Elastic tissue was diminished or absent in the affected regions.

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[1] Bowen, Jour. Cut, Dis., 1912, xxx, p, 241; Precancerous Dermatoses. A study of two cases of chronic atypical epithelial proliferation. Ibid., 1915, xxx, p. 787; Precancerous Dermatoses: A sixth case of a type recently described. (A critical review of all cases recorded). Ibid., Archives of Derm., 1920, xxxviii, n. s., i, p. 22; Precancerous Dermatoses. (The further course of two cases previously reported.) Morrow and Lee, Jour. Cut. Dis., 1918, xxxvi, p. 1; Two cases of the Bowen type of epithelioma.

Treatment.—Destruction of the lesions by freezing caused them to cicatrize in Bowen's cases, but new lesions continued to develop.

ENDOTHELIOMA CUTIS.[1]

A variety of cutaneous lesions have been described under this title, the diagnosis in most instances having been made after microscopic examination of the lesions. A group having uniform symptoms was described by McDonagh[2] under the title "*Nevoxantho-endothelioma.*" In this group the lesions resembled those seen in urticaria pigmentosa with xanthoma-like lesions. One of the two cases recorded by Wise presented similar symptoms. In the other case described by this author the major portion of the lesions were like ordinary soft moles. In other cases several disorders have been simulated, among which are melanoma, and multiple benign cystic epithelioma. Difference of opinion has existed as to whether some of these various cases were endothelioma or basal-cell epithelioma, in the microscopic study made by different observers.

Wise concludes that "tumors of endothelial origin may be indistinguishable from the efflorescence of urticaria pigmentosa, xanthoma, soft moles, syringocystadenoma, multiple benign cystic epithelioma, and perhaps other dermatoses," and states that the inference is justifiable, that a study of sections from cases of this kind would occasionally reveal new growths of endothelial origin.

Endothelioma Capitus.[3]—**Synonyms.**—Sarcoma capitus; Multiple benign epithelioma of the scalp.

Definition.—The disorder is characterized by tumors of variable size situated in the scalp.

According to Crocker, the first case was recorded by Morant Baker under the title "*Withering sarcoma of the scalp.*"

Symptoms.—The disorder is characterized by tumors varying from pea- to orange-size or larger and occurring chiefly in the scalp. Similar but smaller tumors occasionally are present in the face, back and chest. They may present varying shades of red, or be the color of the normal skin. They may be few or numerous and sometimes cover the scalp like a wig. Alopecia may be present over the lesions. They slowly increase in number and enlarge over a period of years and are unaccompanied by subjective symptoms and have no appreciable effect on the general health.

Etiology.—They are reported as occurring in several members of a family in different generations. In several instances tumors have developed following a local injury to the scalp.

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[1] Wise, Amer. Jour. Med. Sci., ebruary, 1919, p. 236. *Cf.* This article for discussion of the subject, with references; report of two cases.

[2] Brit. Jour. Derm., 1912, xxiv, p. 85.

[3] Adamson, Brit. Jour. Derm., 1918, xxx, p. 130; "A case of multiple benign basal-cell epithelioma of the scalp with some remarks on the tumors of the scalp commonly called endothelioma capitus and sarcoma capitus." Report of a case and review of the literature.

Pathology.—The benign character of the disorder led Crocker to believe that the tumors were not true sarcomata. Spiegler classed them with the endotheliomata. Dubreuilh and Aucher demonstrated the growths in their cases to be epithelioma derived from the basal layer of the epidermis and closely resembling rodent ulcer. Adamson reconciles the differences in the recorded cases by assuming that a minority belonged to the group of the sarcomata, but the majority are basal-cell epitheliomata closely resembling multiple benign cystic epithelioma. He found the tumors to be composed of sharply circumscribed alveoli, round, oval, or irregular in shape and surrounded by a clear hyalin strata. These masses are made up of round or oval cells, with large, deeply-staining nuclei. They were densely packed at the outer part of the alveolus and arranged in the form of a pallisade layer.

Treatment.—Surgical treatment has been employed successfully.

RHINOSCLEROMA.[1]

Definition.—Rhinoscleroma is a chronic inflammatory process involving the nose, lip, and upper air-passages, characterized by hardening of the tissues and deformity. The disease was first described by Hebra and Kaposi, in 1870, and has a somewhat restricted distribution, being found largely in southwestern Russia. Cases have been recorded in America by Wende,[2] Pollitzer,[3] Lieberthal,[4] Harmon Smith,[5] Friedberg,[6] Stelwagon,[7] and Alderson,[8] only one of which was indigenous, the remainder being imported.

Symptoms.—The disease commonly begins in the septum or a single ala of the nose, without inflammatory symptoms. The involved parts slowly enlarge, and become finally as dense as ivory. The individual lesions are flat patches, or elevated and circumscribed nodules, papules, and tubercles, painful upon pressure, movable to a certain extent over underlying tissues, and covered either by a normal integument, or by a light- or dark-red, shining, vascular epidermis. Neither hairs nor glands are discernible over the lesions. As the disease progresses the alæ become enlarged, flattened, and so indurated that they cannot be pressed together; while respiration may be impeded by stenosis of the nares. The process may extend to the neighboring parts, involving thus the upper and lower lips, gums, velum, epiglottis, larynx, trachea, and jaws, the teeth meanwhile falling from their sockets and the soft palate becoming in some cases perforated. Involution of the process has not been observed, and the lesions do not degenerate by ulceration. Max Zeissl,[1] however, reports a case in which there was ulcerative destruction of the entire left nostril, as well as of the tip and right ala of the nose. Occasionally, superficial excoriations have occurred, but very rarely a diminution in the consistency of the mass. The disease pursues a chronic course, requiring years for its development; and though the affected parts are painful on pressure, they are otherwise not the seat of subjective sensations.

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[1] For bibliography, see Marschalko, Archiv, 1900, liii, p. 163; ibid., liv, p. 235 (a histological and bacteriological study of two cases, with full review of subject); Castex, La Pratique Derm., t. iv, p. 187.

[2] Jour. Cut. Dis., 1896, xiv, p. 90.

[3] Ibid., 1910, xxviii, p. 388.

[4] Ibid., 1911, xxix, p. 638.

[5] Ibid., 1912, xxx, p. 100.

[6] The Laryngoscope, August, 1910.

[7] Jour. Cut. Dis., 1913, xxxi, p. 427 (case demonstration).

[8] Ibid., 1914, xxxii, p. 308.

Etiology and Pathology.—The disease is observed between the fifteenth and fortieth year, in persons of all social grades and in individuals of both sexes. A microörganism was discovered in the tissue by Frisch,[2] which has been considered etiological. In the early histological study, Kaposi observed a dense infiltration of the corium with small, closely packed cells, which he recognized as a true new-formation. Later investigations have shown the disorder to be a plasma-cell infiltration of the corium, associated with collagenous hypertrophy, and further characterized by the presence of two peculiar types of cells. The characteristic cell originally described by Mikulicz is large and contains clumps of bacilli, supported by a gloea, the latter material being a peculiar mucoid or watery substance. Other cells are seen which have undergone hyalin or colloid degeneration. The microörganisms are found in the lymph-spaces as well as in the characteristic cells. The collagenous hypertrophy is particularly marked at the periphery of the growth, the center being more or less filled with a cellular infiltrate. In the later stages sclerosis occurs throughout the entire area.

The bacillus described by Frisch and Paltauf is short, with rounded ends, and enclosed in a gelatinous capsule measuring 2 microns in length and 0.5 micron in width. It is arranged singly, in pairs, or groups, and lies in the gloea of a Mikulicz cell, or free in lymph-spaces. It presents a close resemblance to the pneumococcus of Friedländer and *Bacillus ozenæ*, with which it is by some considered identical. Perkins,[3] in a study of the organism of rhinoscleroma, classes it as a member of *Bacillus mucosa capsulatus* group, in which are included Friedländer's bacillus and *Bacillus ozenæ*. He considers that the organisms found in the nose in this disorder vary in different cases, though of the same general group, and that the so-called rhinoscleroma bacillus has no etiological relation to the disease, but is rather a secondary infection. Morris and Dore[4] found a short, coccoid, Gram-negative, capsulated bacillus, corresponding to the bacillus of Frisch, and resembling Friedländer's pneumobacillus.

While the organism is pathogenic to animals, the disease has not been reproduced by its inoculation.

Diagnosis.—The disease can hardly be mistaken for another, in consequence of its situation, the disfigurement it occasions, the ivory-like elasticity and induration of the affected parts, and the rarity of ulcerative degeneration. As distinguished from syphilis, it is known to be unaffected by specific medication. Since rhinoscleroma, however, has been by some writers assumed to be a form of syphilis, it is needful to distinguish clearly between the two. But as in the former affection there is rarely softening of the ivory-like induration, much less ulceration, which is common in syphilitic gummata, the distinction is tolerably clear.

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[1] Wien. med. Wochenschr., 1880, p. 621.

[2] Ibid., 1882, xxxii, p. 958.

[3] Jour. Infec. Dis., 1907, iv, p. 51.

[4] Brit. Jour. Derm., 1913, xxv, p. 101.

From the variety of acne rosacea of the nose known as rhinophyma, rhinoscleroma is readily differentiated by the softness and compressibility of the acneform affection and its evident vascular and glandular composition. (*Cf.* section on Gangosa.)

The ulcerations of epithelioma have a more circular outline, a more elevated edge, and occur in persons of a more advanced age. Keloid, if found in the situation of rhinoscleroma, does not ulcerate.

Treatment.—The method of relief thus far employed is a total or partial extirpation of the neoplasm. Kaposi speaks of dilatation of the nares, where there is actual or threatened nasal occlusion, by means of laminaria and compressed sponge. Both excision by the knife and destruction by caustics have been found to secure merely temporary benefit, as the growth is reproduced with rapidity. More recently, *x*-rays have been employed with success. Pollitzer[1] recorded the case of a patient who remained well for three years after treatment with *x*-rays. Friedberg and Lieberthal record improvement from the use of *x*-rays alone or combined with surgical treatment; and Smith reported much improvement in three patients treated with *x*-rays and autogenous vaccine.

Prognosis.—In most instances the future of the patient is grave. The disease may, however, last for many years without producing other than local destructive effects. Interference with respiration, due to obstruction, is to be remembered, and the frequent recurrence after surgical procedure.

TUBERCULOSIS CUTIS.

Tuberculosis of the skin is not a common disorder in comparison with the prevalence of the infection in other organs and tissues of the body. In some of its manifestations it produces great destruction, with consequent disfigurement of the individual. Its lack of response to treatment in many instances, together with the possible destructive effects, makes it a subject of importance. The disease in the skin, as in other situations, is acquired by infection, though a hereditary predisposition may favor its development. In the remote past many phases of tuberculosis of the skin were classified as distinct diseases and in consequence numerous names have been used which now have been eliminated.

The manifestations of tuberculosis in the skin have been ascribed to the local action of the bacillus in the individual lesions, and to the action of its toxin when the bacillus was situated in some organ or tissue distant from the skin. The members of the second group are known as *tuberculides*. In view of recent experimental work many, if not all, of the tuberculides will have to be transferred to the group generally recognized as true tuberculosis of the skin. Some observers make that classification now, but many do not do so and until the matter is definitely settled these several varieties will be individually described. The commonly recognized clinical forms of cutaneous tuberculosis are the following: (1) lupus vulgaris; (2) tuberculosis verrucosa cutis; (3) tuberculosis cutis orificialis, or acute miliary tuberculosis; (4) scrofuloderma. Several subvarieties will be found in the text.[1]

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[1] Loc. cit.

Lupus Vulgaris.—Symptoms.—The symptoms of lupus vulgaris are both numerous and diverse, a fact which may account for the many names which have been applied to these different manifestations, and which with few exceptions are descriptive merely of certain external features.

The primary lesion is a small, soft, reddish, yellowish, or reddish-brown nodule, situated in the corium. It may be elevated moderately or show no elevation, and may or may not be scale covered. The patches are formed by multiplication and coalescence of these nodules. The lesions commonly begin about the nose and spread very slowly over the cheeks. As a rule, little subjective sensation is present, and, except for the gradual enlargement, little change is noted for long periods of time. Under pressure with the diascope, the inherent brown color of the individual nodules of which the patch is composed is revealed. This peculiarity is characteristic and important in diagnosis.

The early lesions above described have been termed *Lupus maculosus*, *Lupus planus*, and, after elevation of the lesion and the production of distinct, visible nodules or tubercles, *Lupus nodosus*, *Lupus tuberculatus*, *elevatus*, *tumidus*, *non-exedens*, and *non-ulcerosus*.

At times there may be extensive edema, thickening, hypertrophy, hyperplasia, pachydermia, and even telangiectasis, and an accompanying lymphangitis or lymphadenitis. To these forms the terms *Lupus hypertrophicus*, *papillosus*, *edematosus*, *elephantiaticus*, *tumidus*, and *exuberans* have been applied. In certain cases marked scaling occurs, and as the patch spreads peripherally cicatrization occurs in the center, producing a depression, and to this form the terms *Lupus exfoliativus*, *Lupus psoriasiforme*, and "*Lupus psoriasis*" have been applied.

In certain cases, as the disease progresses, a fibrous metamorphosis occurs in the early part of the lesion, producing a sclerotic mass. By ntraction of this tissue much deformity may be induced. This pe has been designated *Lupus sclerosus, scléreux, fibrosus.*

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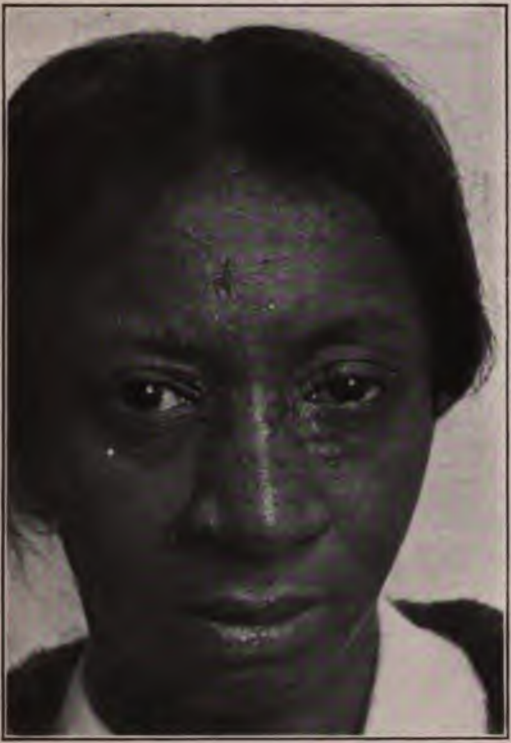
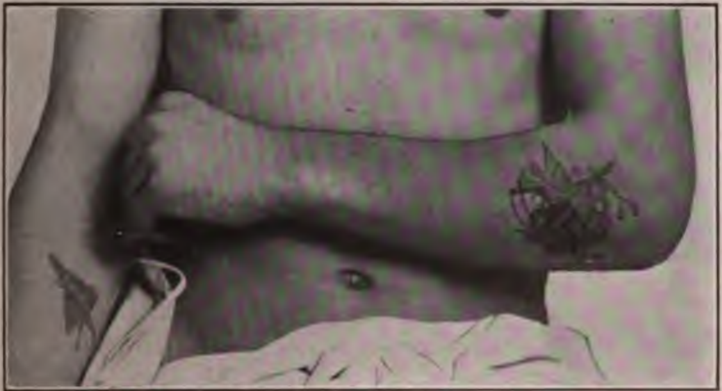
[1] For discussion of classification see recent papers by Olson, Jour. Cut. Dis., 1915, xxxiii, p. 515; "Benign forms of tuberculosis of the skin." Pollitzer, Trans. Sec. Derm., Amer. Med. Assoc., 1918, p. 158; "Tuberculosis of the skin." Wise, ibid., p. 161; Miliary tuberculosis of the skin, lichen scrofulosorum, and the papulonecrotic tuberculides. These papers formed part of a symposium on tuberculosis of the skin read in the Section on Dermatology of the American Medical Association.

FIG. 256



Tuberculosis cutis (Erythematoid lupus vulgaris).

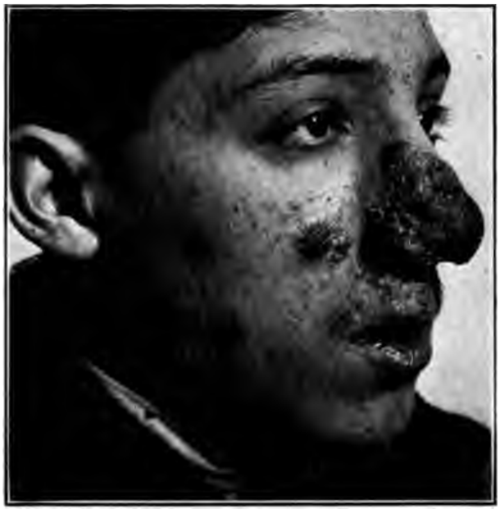
FIG. 257



Tuberculosis cutis occurring in a tatoo. (Phemister.)

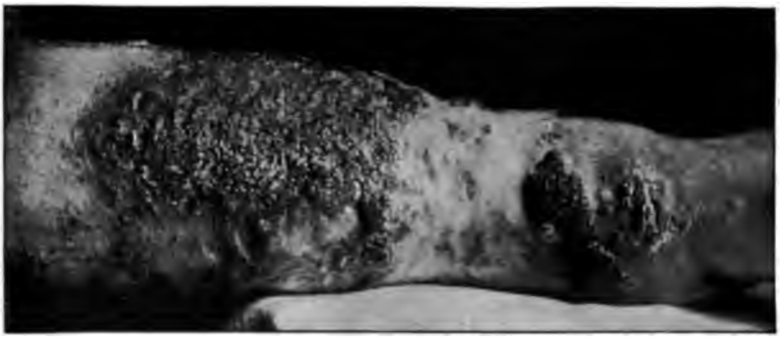
In the ulcerating type of lupus (*Lupus exedens*) oval or circular ulcers are formed, which are well defined, though irregular as to margin, shallow, thin-edged, and flattish. Their floors are dirty-reddish or purplish in color, indolently granulating, furrowed, hemorrhagic, or, when cicatrizing, healthy. When pus is freely formed, whether superficially or deeply, crusting ensues, and the débris of epidermis becomes entangled with the desiccated crusts. These crusts are variously colored and differ in thickness with the severity of the process beneath. When the ulceration advances, it may be superficial, deep, or have other peculiarities, and be subject to the various accidents of the ordinary pro-

Fig. 258



Hypertrophic lupus vulgaris in a young London-born Italian.

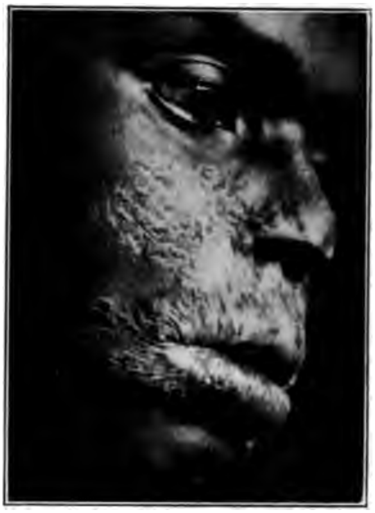
Fig. 259



Lupus vulgaris of the leg.

cess of ulceration, whence the names *Lupus serpiginosus, profundus, superficialis, gangrenosus, exulcerans,* and *rodens.* The names *Lupus crustosus* and *rupioides* are descriptive of the crusting form. When exuberant granulations occur, elevating the floor of the ulcer, the condition has been termed *Lupus fungosus, fungoides,* or *vegetans. Lupus keloides* indicates a cicatricial overgrowth of the scar-tissue left after any one of the several conditions described above.

Fig. 260



Lupus vulgaris in a colored patient.

Lupus vulgaris attacks by predilection the face. After this the extremities and various parts of the body may be invaded. Forchhammer[1] observed in a group of 1200 cases of lupus treated between the years 1896 and 1906 that the primary sites of the disease were as follows: on the face, 81 per cent., of which 50 per cent. began on the nose and 28 per cent. on the cheek; on the neck, 8 per cent.; on the ears, 2 per cent.; on the upper extremities, 8 per cent., and on the lower extremities, 1 per cent.

On the face, the disorder usually begins on the cheek, or about the nose, as a dull-colored maculation or minute nodule, or as a purplish thickening of the skin. Gradual extension of the patch occurs as above indicated, and, as a rule, in this country, not much further change is noted for some time. In certain cases, after absorption of the lupous tissue from the nose, the latter becomes shrunken and retracted to a miniature of its former dimensions, its tip being noticeably reduced to a sharp point, producing thus the characteristic deformity suggesting the beak of a parrot. In other cases, the point becomes bulbous, flattened, livid, and knobbed, with a thickened septum and disordered alæ, an isolated patch or two of the lupous infiltration still remaining in the neighborhood of the cheek on one or both sides.

The subcutaneous tissue, mucous membrane, and cartilages may be destroyed, and in place of the nasal organ itself there may be left eventually two ovoid cavities in the face, separated merely by the posterior flange of the septum.

In severe cases, large portions of the skin of the head (cheeks, lips, nose, eyelids, chin, ears, brow, and neck) become altered, producing a marked and characteristic deformity, reducing the openings of the mouth and lids to narrow slits, interfering with vision, speech, and mastication, and producing a marasmus from these causes alone before there is ulceration at any point.

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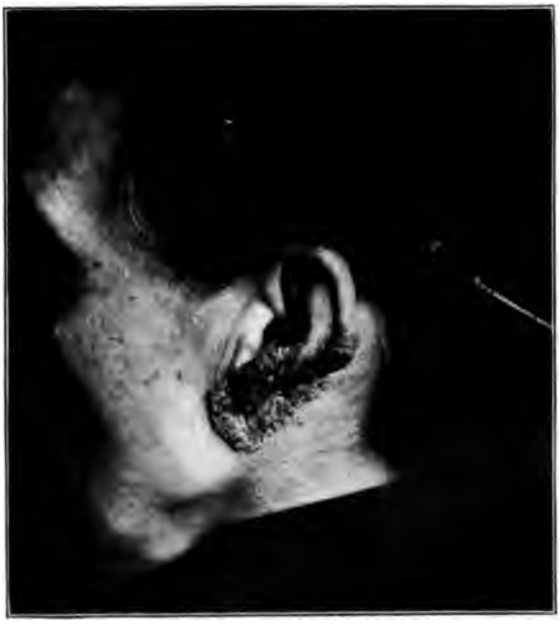
[1] Die Bekämpfung der Lupus Vulgaris in Dänemark, 1896-1911 (Proceedings of the sittings of the Lupus Congress of the German Central Committee—"Bekämpfung der Tuberkulose"—Berlin, April 21, 1911; abstr. Brit. Jour. Derm., 1911, xxiii, p. 338).

The upper lip, when involved, becomes first swollen, fissured, hemorrhagic, and crusted, and the granulating surface indicates extension of the disease to the adjacent mucous membrane. Great deformities are thus produced after healing and scar-formation have occurred.

The lobule of the ear is a favorite site for lupus to attack, the characteristic appearance of the early lesions being present. At a later period, either with or without ulceration, the auricle may disappear or be reduced to a shrunken shell of its former state, the external auditory meatus being occluded by the same process.

FIG. 261



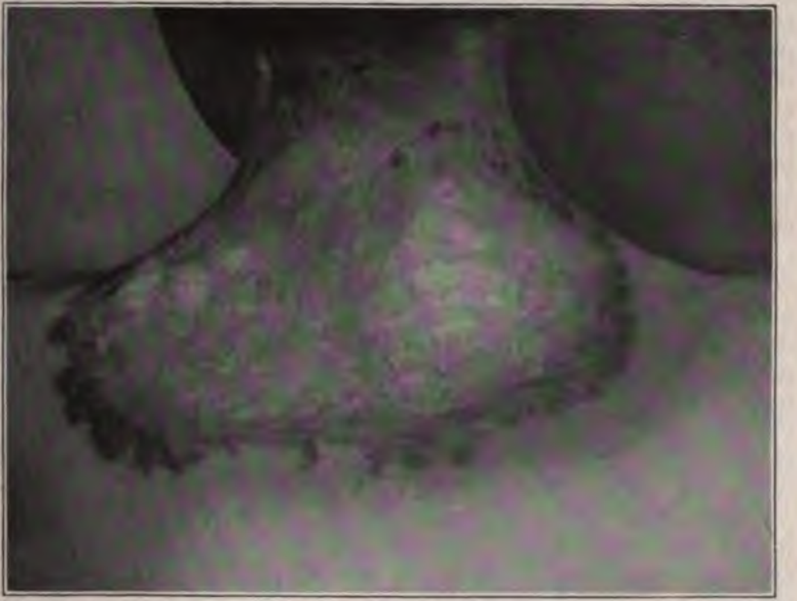
Lupus vulgaris.

On the body, the lesions are usually more extensive but less destructive. Giant areas occur over the loins, the hips, and trunk, and frequently in the form of superficial, serpiginous ulceration, the center healing as the peripheral ring spreads. Gyrate and other figures are induced by the joining of these circles.

On the extremities, the early lesions are similar to those in other areas, but here great deformity occurs later through interference with mobility of the smaller bones of the hands and feet, as the result of rigid cicatrices, and also of the accompanying caries and osseous necrosis. Mutilating effects are thus produced by loss of phalanges, and also by shortening of the hand or foot after the destruction of bone. Elephantiasic enlargement, thickenings, ridges, knobs, nodules, warty excrescences, ulcers, crusts, and callosities are often commingled. The term *Lupus mutilans* has been used to describe this variety. Lupus of the genital region has been described, but is of rare occurrence.

The disease frequently attacks the mucous membranes, particularly that of the nose, and it is thought by some observers that this is the usual starting point of the disorder. The statistics of Forchammer in 800 cases of lupus showed involvement of the mucous membrane in 72 per cent., and in these cases recurrences were chiefly due to reinfection from the mucous membrane. In a large number of cases at the London Hospital with involvement of the mucous membrane, Sequeira[1] found the larger proportion to involve the nasal mucous membrane; then, in the order of frequency, the lips, the buccal mucosa, the gums, the palate, the larynx, and the tongue. In these regions, owing to the warmth and moisture, the lesions appear as moist, papillary growths or granulating patches, which may ulcerate and cicatrize. The borders are well defined, and the surface reddish or pallid, or of a dirty, grayish-white color when the investing epithelium is loosened but not detached. On the tongue the lesions may be papillomatous, or ulcerating, and the gums appear swollen and ulcerated. Frequently, the teeth are exfoliated.

FIG. 262



Lupus vulgaris serpiginosus.

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[1] Allbutt and Rolleston's System, ix, p. 472.

mouth and lids to narrow slits, interfering with vision, speech, and mastication, and producing a marasmus from these causes alone before there is ulceration at any point.

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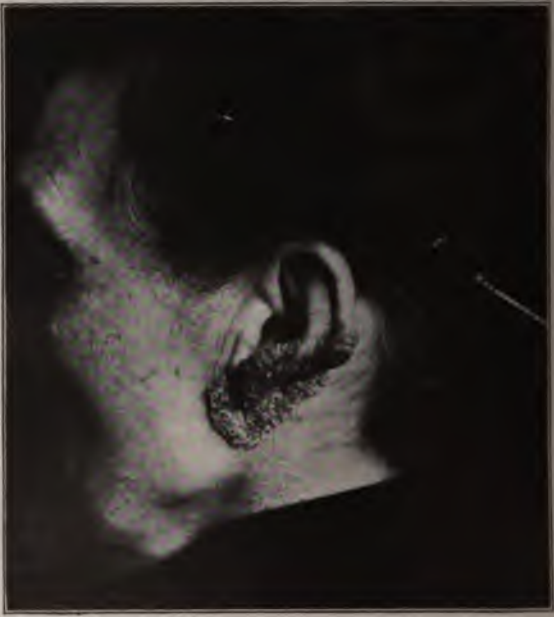
FIG. 261



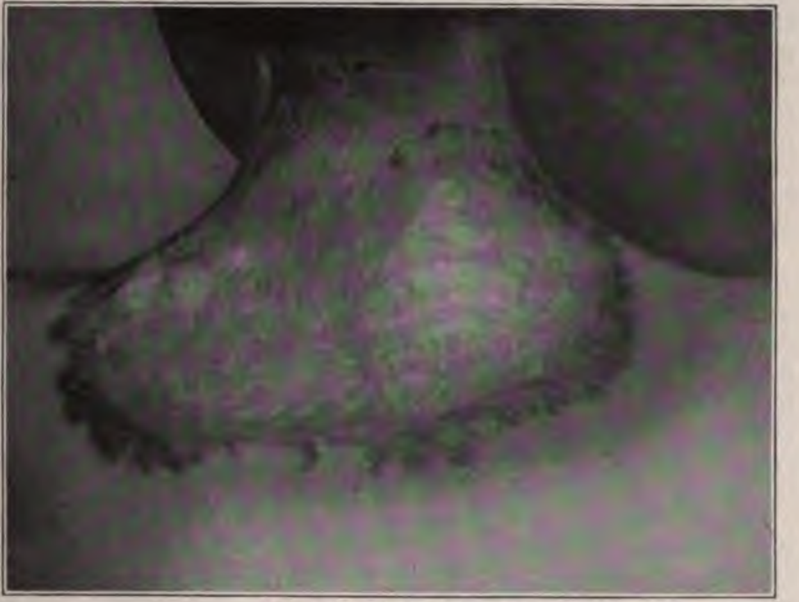
Lupus vulgaris.

On the body, the lesions are usually more extensive but less de tive. Giant areas occur over the loins, the hips, and trunk frequently in the form of superficial, serpiginous ulceration, the healing as the peripheral ring spreads. Gyrate and other figur induced by the joining of these circles.

On the extremities, the early lesions are similar to those in areas, but here great deformity occurs later through interferenc mobility of the smaller bones of the hands and feet, as the re rigid cicatrices, and also of the accompanying caries and necrosis. Mutilating effects are thus produced by loss of phal and also by shortening of the hand or foot after the destruction of bone. Elephantiasic enlargement, thickenings, ridges, knobs, nodules, warty excrescences, ulcers, crusts, and callosities are often commingled. The term *Lupus mutilans* has been used to describe this variety. Lupus of the genital region has been described, but is of rare occurrence.

The disease frequently attacks the mucous membranes, particularly that of the nose, and it is thought by some observers that this is the usual starting point of the disorder. The statistics of Forchammer in 800 cases of lupus showed involvement of the mucous membrane in 72 per cent., and in these cases recurrences were chiefly due to reinfection from the mucous membrane. In a large number of cases at the London Hospital with involvement of the mucous membrane, Sequeira[1] found the larger proportion to involve the nasal mucous membrane; then, in the order of frequency, the lips, the buccal mucosa, the gums, the palate, the larynx, and the tongue. In these regions, owing to the warmth and moisture, the lesions appear as moist, papillary growths or granulating patches, which may ulcerate and cicatrize. The borders are well defined, and the surface reddish or pallid, or of a dirty, grayish-white color when the investing epithelium is loosened but not detached. On the tongue the lesions may be papillomatous, or ulcerating, and the gums appear swollen and ulcerated. Frequently, the teeth are exfoliated.

FIG. 262



Lupus vulgaris serpiginosus.

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[1] Allbutt and Rolleston's System, ix, p. 472.

As a rule, the course of the disease is slow. For a quarter of a century a lupus patch may be limited to a space no larger than the palm of the hand and exhibit some evidence of activity during the greater portion of that period.

While lupus vulgaris usually occurs as a slowly progressing disorder, more acute cases are recorded, usually following measles, and these are recorded under the title *Multiple disseminated lupus vulgaris.* This form usually occurs in children. Adamson[1] collected a series of 28 cases of this type. MacLeod,[2] Sequeira,[3] Dore,[4] Hazen[5] and others have reported cases of similar type. In these, multiple lesions appear suddenly scattered about the body-surface. The lesions consist of small, discrete nodules or patches, varying in size from that of a split-pea to coin-size and larger. The patches are made up of an aggregation of typical lupus nodules, and may be scale covered (resembling psoriasis) or verrucous. In a patient seen by the author, numbers of small brown nodules occurred irregularly disseminated over the trunk, with psoriasiform patches over the extremities. Later the patient developed a tubercular spondylitis, and in the course of some years the scaling patches became verrucous and were then typical of lupus verrucosa. A case representing the above type occurring in an adult, not preceded by measles, is recorded by Little.[6]

The variety of the disease described as *Erythematoid lupus vulgaris*[7] occurs as one or more patches, usually upon the face, and usually asymmetrically. These patches present a raised, scaling border and depressed atrophic center. They are reddish in hue, the color only partially disappearing upon pressure. Some yellowish miliary nodules may be observed under the diascope. The course of the disorder is chronic, and its resemblance to lupus erythematosus is striking. These cases have all been proven, microscopically and culturally, to be unusual forms of lupus vulgaris.

Complications of Lupus Vulgaris.—The disease may be associated in the same patient with other varieties of tuberculosis of the skin, such as verrucous tuberculosis, scrofuloderma, and tuberculous ulcers. Bone and joint tuberculosis may also be present. Visceral and pulmonary tuberculosis may follow, and erysipelas occurs not infrequently, the latter at times having a beneficial effect on the lupus. Carcinoma occurs as a sequel in from 2 to 4 per cent. of the cases. Cutaneous horns occasionally develop on a patch of lupus. A typical horn developed on the upper lip in a case of long standing lupus observed by the author. A similar case with several such lesions is recorded by Sibley.[8]

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[1] Brit. Jour. Derm., 1904, xvi, p. 366.

[2] Ibid., 1907, xix, p. 390.

[3] Ibid., 1913, xxv, p. 99.

[4] Ibid., 1910, xxii, p. 233.

[5] Jour. Cut. Dis., 1919, xxxvii, p. 89. Multiple Disseminated Lupus Vulgaris. Two cases reported. eferences to previously recorded cases.

[6] Brit. Jour. Derm., 1911, xxiii, p. 49.

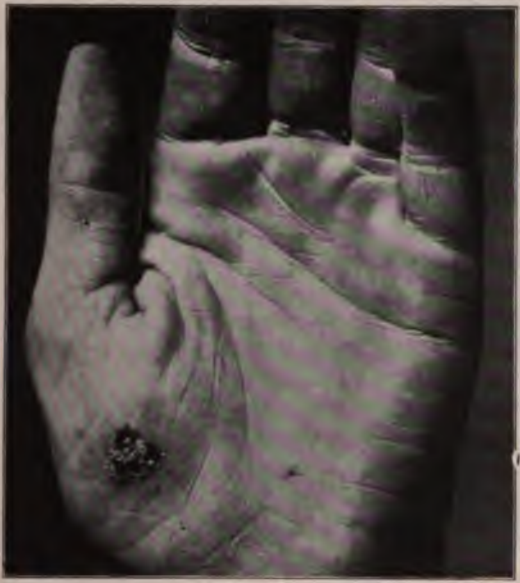
[7] Leloir, Jour. Mal. Cut., May, 1891, p. 241.

[8] Brit. Jour. Derm., 918, xxx, p. 100; Lupus Vulgaris with Cutaneous Horns.

Tuberculosis Cutis Verrucosa.—This form of tuberculosis of the skin is characterized, as its name indicates, by warty lesions. The common type is the anatomical tubercle or post-mortem wart, which in 1884 was proven to be tuberculous by the discovery of the bacillus in the lesions. In 1886, Riehl and Paltauf[1] described a somewhat similar but more extensive variety of this form. These lesions are always produced by direct inoculation, and may therefore be considered the inoculation type of cutaneous tuberculosis.

Verruca Necrogenica (*Post-mortem tubercle, Dissection tubercle, Anatomical tubercle*).—This disorder was first termed verruca necrogenica by Wilks.[2] It occurs usually on the fingers (especially on the dorsum of the thumb and index finger) of those engaged in the handling or dissection of cadavers; or it may be caused by other accidental inoculations with the tubercle bacillus. It begins at the site of an abrasion or wound as a papule, papulo-pustule, or vesico-pustule, with a deep-seated base and reddish or reddish-purple areola. The lesion becomes encrusted and spreads peripherally. Hyperkeratosis follows, and after a time an indurated, horny, pigmented lesion forms, which may become fissured at one or more points. As a rule, the lesion becomes split-pea- to bean-sized, usually remains single, and may thus persist for months or years. At times, dermatitis and suppuration may complicate the process. In a very few cases grave symptoms result,

FIG. 263



Tuberculosis cutis verrucosa.

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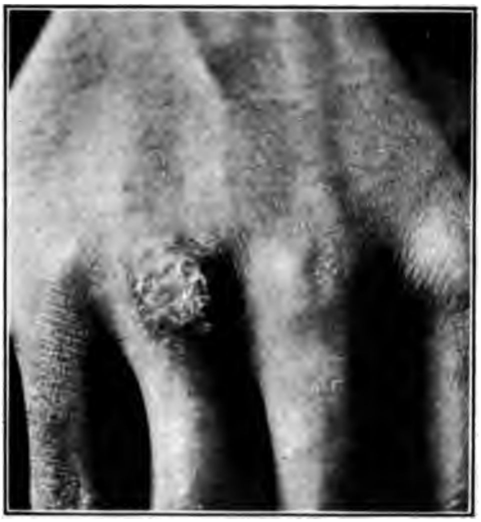
[1] Archiv, 1886, p. 19.

[2] Guy's Hosp. Reports, s. iii, viii, p. 263.

either in the involvement of the deeper tissues (subcutaneous, thecal, tendinous, periosteal), or in the production of erysipelas, pyemia, septicemia, or gangrene. Surgeons divide these cases into mild and acute varieties, according to the symptoms exhibited. In a few instances the local process has been followed by generalized tuberculosis.

Tuberculosis Verrucosa Cutis (Riehl and Paltauf).—This is a more extensive variety of a similar process, also due to inoculation from without. The lesions in this type occur most frequently on the dorsal surface of the hand, whence they may spread up over the forearms, very rarely over the palms. They also occur on other portions of the body, such as the area covering the inner malleolus and various portions of the limbs. The lesions occur as plaques, which are insensitive, variously sized from small-coin to palm or larger; are single or multiple, definitely circumscribed, ovoid or scalloped in outline, brownish-red in color, movable, and are often covered with fine, pointed vegetations, minute pustules, and even crusts. Frequently, pus may be expressed from between the verrucous lesions. When healing occurs, a smooth and scaling scar usually results. In certain instances, particularly over the forearms, the scar exhibits a stippled appearance, similar to that seen following lupus erythematosus.

FIG. 264



Tuberculosis verrucosa cutis. (Gatewood.)

Cases of this type have been seen in miners, the subjects of consumption, who have had the habit of wiping their mouths with the backs of their hands. It is reported frequently among the miners in the district around Dortmund, Prussia. Sequeira[1] records the case of a patient infected through a wound produced by an electric-lighting wire, while he was working in a butcher-shop.

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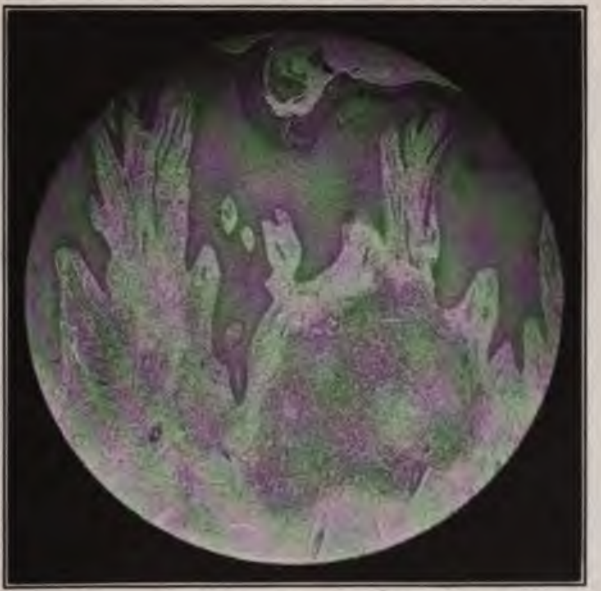
[1] Loc. cit.

This disease very closely resembles cutaneous blastomycosis, from which it is to be distinguished. The glands may become infected early, and visceral complications are not uncommon. Instead of the comparatively dry, warty lesions above described, cases occur in which exuberant, soft, and florid excrescences rise one or two centimeters above the general level, the individual elements being separated by deep fissures, and the whole bathed in purulent mucus and frequently covered with dark crusts. This type was early described by Morrow.[1] Another variant is that described by Riehl as *Fibromatosis tuberculosa cutis*, in which there is not merely a papillomatous, but often a sclerotic, growth found on the lips, nose, cheeks, or about the anus or other mucous outlet of the body, interspersed with verrucous lesions, vegetations, and small, shallow ulcers. The tuberculous masses may be in the form of tumor-like bodies or thickenings of the subcutaneous tissue.

FIG. 265



Photomicrograph of a section of tissue taken from the case of tuberculosis verrucosa cutis shown above. The nodule and the acanthosis are well shown.

Elephantiasis Tuberculosa Cutis is a term applied to gigantic overgrowths of the integument, complicated by lymphatic occlusion. In these cases there has usually been a blocking of the lymphatic channels. It is chiefly important to note in this connection that accidental inoculations with the bacillus of tuberculosis produces in different individuals different clinical results, the common and characteristic train of symptoms being the warty lesions above described.

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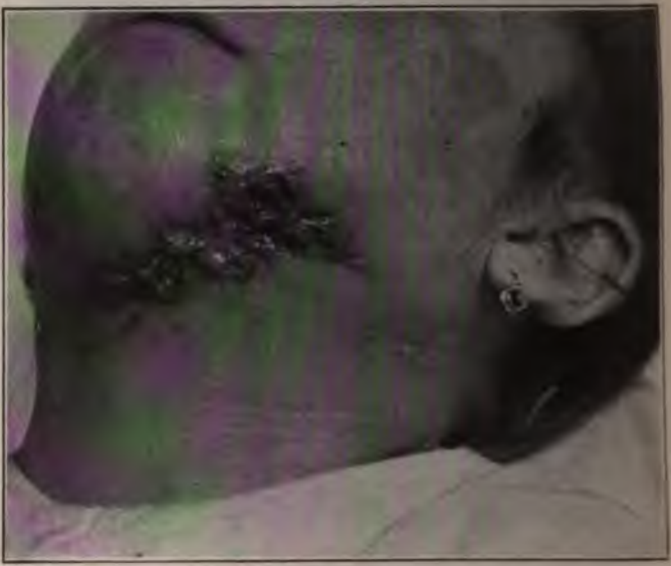
[1] Jour. Cut. Dis., 1888, vi, p. 401.

Scrofuloderma.—The term scrofuloderma should now be limited to those lesions in the skin produced by the local action of the bacillus of tuberculosis by direct extension from some structure beneath the skin. The lesions, therefore, occur most frequently in the neck, from tuberculosis of the glands, and about joints and over bones, which are most frequently attacked with tuberculosis.

The scrofulodermata are characterized by the occurrence of pathological processes in the skin, lymph-glands, or periglandular tissues, which betray evidence of the tuberculous process. They usually begin as firm, well-defined, subcutaneous nodules, similar in type to the syphilitic gummata, which gradually enlarge, become attached to the skin, subsequently degenerate, exhibit typical ulcers, and usually terminate by characteristic cicatrices. The typical and commonest form of scrofuloderma is encountered about the face and neck, where the lymphatic glands have long been tumid, and are either dense or doughy to the touch. Usually this condition is reached very slowly; often months and years are required for its production. The glands may be as small as almonds, or as large as the closed fist. Gradually the characteristic inflammation ensues in the skin which is superimposed. The latter becomes purplish and thinned, and finally yields, giving exit to a sero-purulent fluid mingled with caseous matter and blood. Fistulous tracts and sinuses result, which

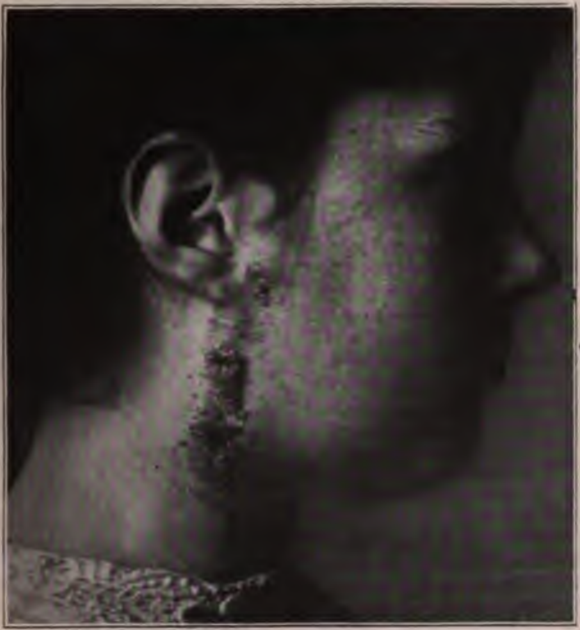
FIG. 266



Scrofuloderma.

undermine and perforate the skin, resulting in the production of a chronic discharge and characteristic ulcers. The ulcers are usually linear, occasionally elongated and oval, rarely circular. Their floors are uneven, covered with pallid granulations, and secrete a watery pus. The edges are undermined, tumid, inverted, and usually not changed in color. The edges of the ulcers are frequently thinned by stretching over fistulous pockets, and may be reddish or purplish in hue. The bases of the ulcers are attached deeply to the subcutaneous tissue, and are firm or soft, and never densely indurated. Fistulæ may be discovered extending to the point of origin of infection. The crusts are thin, tenacious, reddish or brownish, and usually have the shape of the preceding ulcer, which is frequently linear, rarely bulky, or rupioid. The resulting cicatrices are corded, depressed in irregular lines or bands, and often alternate with equally irregular nodules (scrofulous gummata), where the process either has been arrested or is still in activity.

Fig. 267



Scrofuloderma.

Rarely large ulcers are formed by the dissection of areas of subcutaneous and intramuscular tissue in the neck or even over the extremities. The number of lesions in this type of tuberculosis is usually few, being limited, as a rule, to from two to six.

Another type of scrofuloderma is that described as *Scrofulous gummata*, which begins with a subcutaneous nodule on the back or over the extremities, more commonly in children, and in which there is no connection with gland, bone, joint, or other structure. The career of these lesions is practically that outlined above. Scrofulodermata usually occur in children and young adults and in persons of low vitality.

A group of cases described under the title *Tuberculosis fungosa cutis* (*Riehl*) presents tumors resembling those seen in granuloma fungoides and sarcoma, intermingled with other lesions of tuberculosis of the skin. These cases originate, as a rule, in an infected bone, muscle, or other structure beneath the skin, the latter being invaded by direct contiguity; which fact therefore places the condition in the category of the scrofulodermata. Pick[1] reported two cases of this type, and, referring to the literature of others, maintains that it should be kept as an entity. It is essentially an infiltrating form of cutaneous tuberculosis, with excessive granulations, forming fungous tumors, and having in addition ulceration, crusts, and fistulous tracts, in which cheesy degeneration is abundant.

At times, scrofulous gummata, occurring in small-nut- to egg-sized tumors, form along the lymphvessels of the lower extremities particularly. When such a tumor breaks down, it furnishes the typical picture of the scrofulous ulcer, with its cheesy and watery pus, thin, even edge, and indolent career. In these cases tubercle bacilli have been recognized in the secretion. The condition was first described by Bazin as *Tuberculous lymphangitis*. Hallopeau and Goupil described a condition of tubercular lymphangiectasis in which there were swellings of the lymphvessels, from the secretion of which tubercle bacilli were obtained, and in which systemic infection not infrequently followed.

A hitherto undescribed variety of tuberculosis cutis is recorded by Wende,[2] under the title: *Nodular tuberculosis of the hypoderm*. The case presented subcutaneous nodules, varying in size from that of a pea to a pigeon's egg, situated on the chin, cheek, and temple. The lesions would appear, grow slowly, and would finally disappear and new ones form. They were unaccompanied by any redness or scaling of the skin. The nodules were painful to the touch. There was no suppuration or ulceration. The microscopic sections were characteristic of tuberculosis; the histological architecture was that seen in erythema induratum. Tuberculin tests and inoculation experiments in animals were positive. Treatment with injections of tuberculin proved beneficial.

While not conforming to any of the types described above, this condition resembles to a marked degree the scrofulous gummata, but the age of the patient and absence of ulceration are distinguishing features. It is also analogous to certain cases of erythema induratum, but, again, the location of the lesions and the presence of such numbers of tubercle bacilli would be unique for that condition. Its nosological position is therefore not definitely fixed.

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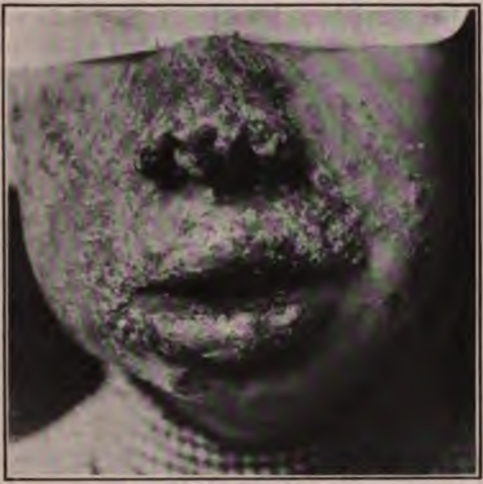
[1] Jour. Cut. Dis., 1904, xxii, p. 305.

[2] Ibid., 1911, xxix, p. 1.

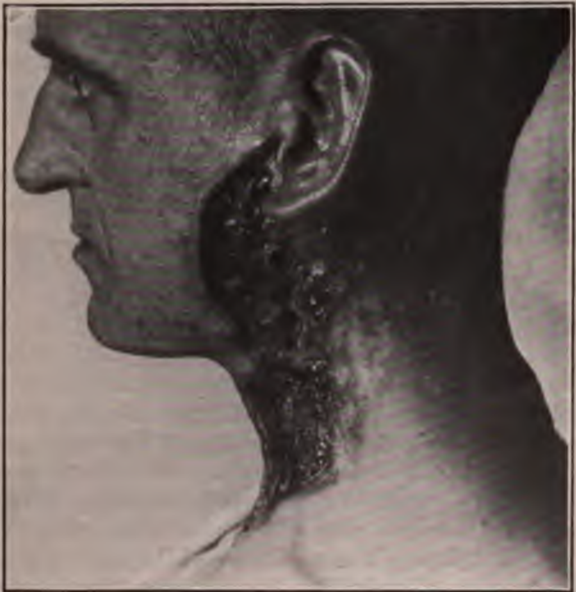
rculosis Cutis Orificialis (*Tuberculous ulcers*).—The clinical ncluded under the above title are those once supposed to be

FIG. 268



Ulcerating tuberculosis.

FIG. 269



Ulcerating tuberculosis secondary to infection of glands of the neck.

manifestations of cutaneous tuberculosis. The title "tuberof the skin" was, in fact, applied exclusively by many writers to the ulcers and their preceding lesions observed and described by Kaposi, Jarisch, and Chiari.[1] These were indolent, oval or circular, shallow, discrete, reddish-yellow, granulating ulcers, often covered with thin crusts, occurring about the mucous orifices of patients affected with internal tuberculosis and with development of miliary tubercles in the adjacent mucous tract. These ulcers occur most commonly about the lips, over the wings of the nose, and occasionally about the ears, in patients suffering with laryngeal, palatal, oral, or pulmonary tuberculosis; about the anal region in patients suffering with intestinal tuberculosis; about the glans in tuberculosis of the bladder; and about other parts of the external genital region in tuberculosis of the internal generative organs. In the case of a patient in advanced pulmonary tuberculosis seen by Dr. Hyde there was a tuberculous ulcer near the anus, and also a well-defined patch of infiltration in near proximity, highly suggestive of nodular lupus.

There may be one or several ulcers, which spread slowly and show no tendency to heal. As a rule, the ulcers are small, but occasionally they attain considerable dimensions.

Acute Tuberculosis has been described under different titles. In the case of children, this form is exhibited as macules, vesicles, bullae, papules, and pustules, terminating in deep, crusted, circinate lesions, accompanied by caseation of the neighboring glands. In all of these lesions tubercle bacilli may be demonstrated, and inoculation with cultures results in distinct tuberculous infection.

Little[2] reported a case of acute disseminated tuberculosis cutis in an infant seven months of age the subject of tuberculous glands of the neck. The eruption developed acutely and was composed of small, deep, bluish, nodular lesions situated on the skin from the buttocks to the feet. He refers to a similar instance recorded by Pelagatti.[3]

A case of universal dermatitis of tuberculous type in an adult was reported by Bruusgaard,[4] in which the dermatitis was accompanied by a general enlargement of the lymphatic glands. In the glands and sections of skin a tuberculous structure, with tubercle bacilli, was demonstrated.

Exanthematous Miliary Tuberculosis of the skin may follow the exanthematous fevers in children. In this variety, the lesions are multiple, indolent, dull brownish-red, acuminate tubercles, situated in or near the cutaneous follicles, and suggesting the lesions of acne papulosa. After degeneration, they present rounded, circular, [illegible] polygonal, sharply-cut ulcers, having a violaceous border, an irregular, granular floor, and a scanty sero-purulent discharge. Miliary nodules are to be seen both on the floor of the ulcerative surfaces and at the periphery of the lesion. The presence of tubercle bacilli may be demonstrated. This disorder occurs, as a rule, in patients exhibiting other unmistakable symptoms of tuberculosis.

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[1] [illegible] 1879, [illegible] p. 200.

[2] [illegible] Derm., 1916, xxviii, p. 243.

[3] Giorn. Ital. delle Malattie della Pelle, 1898, p. 714.

[4] Archiv, 1903, lxvi, p. 227.

In addition to the above described ulcers and also those discussed in connection with lupus vulgaris, occasionally indolent ulcers of fairly large size occur on the limbs and other situations, which are considered to be tuberculous. This latter type of ulcer is to be carefully distinguished from syphilis.

Etiology.—Accidental inoculation of tuberculosis may occur at all ages and in both sexes, the infective material gaining access to the economy, in a large number of instances, by the medium of the lymphatics. There is, however, opportunity for such transmission among the members of any family in which pulmonary tuberculosis exists; hence, the widespread belief in the heredity of the disease. There are certain factors that must be considered as indirect causes. The young, the delicate, and the cachectic furnish a proper soil for the tubercle bacillus. The mode of life among the very poor and the filthy and degraded classes predisposes to infection. In the major number of all cases of lupus vulgaris, the time of its appearance is during the first decade, between the third and the sixth years of life. It rarely begins after the thirtieth year, for the reasons above mentioned.[1] The significant fact in this connection is that in this period of life a child, often deprived of the constant care of the mother by the demands made by a still younger infant, untaught in the simplest rules of cleanliness, picking and scratching the face after miscellaneous contacts of the fingers with all sorts of material, is exceedingly liable to inoculate the skin of the face with tuberculous virus, if there be victims of such disease occupying the same apartment or house. It is significantly first upon the face in these early years, to which the exposed hands have been carried, that the early symptoms of lupus vulgaris are seen. Further, it is noteworthy that well-marked cases are more frequent among the poor, the filthy, and the degraded than among the comfortable and cleanly. The prevalence of the disease in public as contrasted with private practice is conspicuous in all statistics.

Besnier, Little, and others report cases of lupus resulting from tuberculous infection in vaccination; Fournier, an instance in which a young woman was infected during the piercing of the ear for the insertion of ear-rings; Strauss, the case of a student who was wounded by a rapier in a duel and as a result developed lupus in the site of the wound; and Brums, an instance of infection by inoculation with a hypodermic syringe used for injecting morphin. Bernhardt[2] reported a case of cutaneous tuberculosis following ritual circumcision. Ernst[3] recorded a case of tuberculosis verrucosa cutis after tattooing and reports eight cases in the literature of cutaneous tuberculosis following the same operation, seven of which, including his own, were probably infected from tuberculous sputum. Among these eight cases one was tuberculosis verrucosa cutis, five lupus vulgaris, one tuberculosis cutis propria, one a tuberculide, and one unclassified. Dore[1] and others have reported similar instances.

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[1] For reference to cases beginning in old age see Little, Brit. Jour. Derm., 1906, xviii, p. 185, and ibid., 1918, xxx, p. 223. Fox, ibid., 1894, vi, p. 84. Silbey, ibid., 1906, xxx, p. 223.

[2] Archiv, 1900, liv, p. 221.

[3] Derm. Centralb., 1907, xi, p. 66.

In verruca necrogenica and other types of verrucous tuberculosis, it is contact with the bodies of the dead or with tuberculous matter in any form that determines the result. Lassar[2] reported two cases of verrucous tuberculosis in patients working with diseased cattle. He concluded that these were examples of bovine tubercle bacilli inducing cutaneous tuberculosis in man. Fabry and Schulze[3] report large numbers of cases occurring among the miners in the district around Dortmund, Prussia. Lipschütz[4] states that the Riehl-Paltauf variety of tuberculosis verrucosa cutis is due most commonly to inoculation with the bovine type of the tubercle bacillus, while verruca necrogenica is probably due to inoculation with the human type of the bacillus.

Lupus vulgaris also may be produced by inoculation, but frequently the infection is brought to the skin through the lymphatics, and occasionally by way of the blood-stream. In the disseminated cases it is supposed that the bacilli are liberated from a localized focus during the attack of measles which this type usually follows, and spread through the blood-stream to various parts of the cutaneous surface. In scrofuloderma the bacilli find their way to the skin by direct extension from beneath. Wilson,[5] in a careful examination of nine cases of lupus vulgaris, failed to find tubercle bacilli in the feces.

While tuberculosis cutis or orificialis is essentially an example of autoinoculation, it is usually secondary to visceral tuberculosis.

Pathology.—All the varieties of tuberculosis of the skin, which include lupus vulgaris, tuberculosis cutis verrucosa, scrofuloderma, and tuberculosis cutis orificialis, are due to infection with the tubercle bacillus, and are practically identical histologically with tuberculous lesions in other parts of the body. The discovery of bacilli in lupous tissue, first made by Koch, has since been verified by Doutrelepont, Weichselbaum, Meisels, Schuller, Lustig, and others. The striking resemblance, first shown by Virchow, between a caseous miliary tubercle and a lupous nodule had, even before Koch's discovery, pointed to an identity of origin. The tuberculous nature of these disorders has been proven in addition by inoculation experiments. Lenz, Huter, Schuller, ourselves, and others have produced tuberculosis by introducing in rabbits granulations taken from lupus and other varieties of cutaneous tuberculosis.

For our early knowledge of the microscopic character of cutaneous tuberculosis we are indebted largely to the Germans. Virchow, Auspitz, Billroth, Lang, Kaposi, Klebs, Stilling, and Thin have contributed amply to the subject. Recently Fordyce[6] has presented his findings in the several varieties.

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[1] Brit. Jour. Derm., 1913, xxv, p. 257; ibid., 1918, xxx, p. 22.

[2] Zeitschrift, 1903, x, p. 505.

[3] Archiv, 1904, lxx, p. 329.

[4] Ibid., June, 1914, cxx, p. 387.

[5] Brit. Jour. Derm., 1910, xxii, p. 252.

[6] Jour. Cut. Dis., 1914, xxxii, p. 23 (Histological study in some types of tuberculosis, illustrated with eight microphotographs).

The histological structure of the various forms of cutaneous tuberculosis varies in minor particulars, but in essential features sufficient uniformity exists to enable the observer to discern that each is due to a similar exciting cause. Each is produced by the local action of the tubercle bacillus, and presents a cellular new-growth; vascular changes, ranging from slight proliferation in the coats of the vessels to their complete obliteration; and attenuation, hypertrophy, or complete destruction of the collagen. The sebaceous and sweat-glands, hair-follicles, and elastin all suffer alteration, even to destruction. The epidermal changes in all are secondary, and include acanthosis, hyperkeratosis, parakeratosis, scaling, and at times even complete destruction by ulceration. The tuberculous nodule in the skin resembles that found in other organs, and consists essentially of one or more giant-cells immediately surrounded by a number of small round cells, which have vesicular nuclei, and which are either mononuclear leukocytes or daughter plasma-cells (Unna). Interspersed among these may be a few multinuclear cells, and surrounding these is a zone of plasma- and connective-tissue cells. No vessels exist in the nodule, and the fibrous elements are either attenuated or completely absent. The nodule is surrounded in the nodular form of lupus by a collagenous capsule. In another variety no limiting capsule is present, and the cellular hyperplasia spreads along the lymph spaces, producing an even, brownish discoloration of the skin, in which case giant-cells are not numerous.

Degeneration occurs in the nodule, as is shown by the cellular protoplasm becoming homogeneous, and the nuclei becoming incapable of absorbing stains normally. True cheesy degeneration seldom occurs in the skin, which fact might be accounted for by the relative scarcity of bacilli in most of these lesions.

The cellular hyperplasia is composed of giant-cells, which are large, oval, round, or irregularly shaped cells, containing, as a rule, many peripherally placed nuclei, and having a homogeneous center; plasma-cells, which vary in size, are usually oval or oblong in shape, possess a large amount of protoplasm, and present an eccentrically placed vesicular nucleus; and small round cells, usually described as mononuclear leukocytes or daughter plasma-cells (Unna), which contain nuclei similar to the plasma-cells. In addition, some mast-cells are present, and in these the nucleus is surrounded by granules of protoplasm, which are identified by stains having metachromatic properties. Multinuclear cells are also present, and a large number of connective-tissue cells. Tubercle bacilli are found most abundantly in the acute miliary variety of cutaneous tuberculosis; most rarely in lupus vulgaris, in the lesions of which they are often difficult to demonstrate. Giant-cells are most abundant in lupus vulgaris; while cheesy degeneration, common to internal tuberculosis, is more prevalent in the miliary variety. Bacilli may be found between the cells, but are found more often in giant-cells. Animal inoculations may be performed successfully in each variety, and they all react to tuberculin injections.

Lupus Vulgaris occurs in two varieties, the nodular and the diffuse. In the former, the tubercles above described are enclosed in a limiting capsule of collagen, while in the diffuse variety the infiltration spreads evenly along the lymph-spaces without interruption, producing a diffuse infiltration. There is a tendency for the granuloma to be replaced by connective tissue, which at times multiplies to an excessive degree, producing a condition of elephantiasis. As the cellular infiltration progresses, the normal structures of the skin are atrophied or destroyed; collagen, sebaceous and sweat-glands, hair-follicles, and, finally, elastin, all disappear. Proliferative changes may occur in the epidermis, in which marked down-growth (acanthosis) of the rete into the corium results, producing the papillomatous variety of lupus; while with increased cornification verrucous forms occur. Pressure from below may rupture the epidermis, permit pyogenic infection, and result in ulceration. Edema, with accompanying parakeratosis and scaling, may be present. All of these epidermal changes are secondary, however, and are in themselves not tuberculous, the principal and characteristic changes being found in the corium.

Tuberculosis Verrucosa Cutis is distinguished by having the tuberculous plasmoma located chiefly in the papillary layer of the corium. The usual structure of the tuberculous nodule may be demonstrated. Marked acanthosis and hyperkeratosis are also distinguishing features. Miliary abscesses, produced by pus-cocci, may be found both in and beneath the epidermis. Tubercle bacilli are usually more numerous than in lupus vulgaris, and find their entrance from without. Both histologically and clinically, this variety of tuberculosis is nearly identical with some of the forms of lupus vulgaris, and now often is classified as a manifestation of lupus vulgaris.

Tuberculosis Cutis Orificialis.—In this variety, both in the number of bacilli present and in the type of lesion, there is an analogy with the miliary tubercles of other organs. Large numbers of typical, circumscribed nodules are found deep in the corium. Bacilli are numerous and easily demonstrated. The degenerative processes go on rapidly, the tubercles breaking down and coalescing to form masses of softened necrotic tissue, which soon break through the epidermis to form an ulcer. About the borders of such necrotic areas new nodules are constantly forming, and the whole process is rapid, as in acute tuberculosis of other tissues. Histologically, it is composed of the usual tuberculous plasmoma, its distinguishing features being the presence of large numbers of bacilli, and also typical cheesy degeneration, which is not found in the other varieties.

Scrofulodermata.—The scrofulodermata originate in the subcutaneous tissues and involve the skin secondarily. The lymphatic glands or the tissues about the glands or lymphatic vessels become the seat of the tuberculous process, which runs a subacute course. The glands or periglandular structures finally break down into softened, necrotic masses. Such areas of necrosis may remain indolent and superficial, or, in case a gland is involved, may be deep and extend by burrowing prolongations even to the bone. Sooner or later, the skin over these softened masses becomes involved in a subacute inflammatory process and gives way, producing the typical ulcer, with soft, ragged, and often extensively undermined edges. Experimental inoculations and the presence of tubercle bacilli have demonstrated these subcutaneous lesions to be tuberculous. The number of bacilli present varies greatly, being much larger than in lupus, but much smaller than in the orificialis varieties of cutaneous tuberculosis. The relationship of the scrofulodermata to lupus is occasionally shown by the formation of typical lupus nodules near the border of these scrofulous ulcers, the result, no doubt, of inoculation of the skin with a discharge from the ulcer. The granuloma here consists of a diffuse plasma-cell infiltration, with some giant-cells about the edges of the lesions.

Diagnosis of Tuberculosis Cutis.—Epithelioma, though rarely resembling lupus vulgaris, is more often designated by that than by any other false title. Great confusion has arisen over the looseness with which several authors have furnished illustrations of "lupus exedens," which were really pictures of cancer. But the latter is rarely a disease of early life, and when of early occurrence may not persist to adult years; the reverse of which is true in the majority of all cases of lupus. The nodules of lupus are absent in epithelioma, and their evolution in the disease is slower, less painful, and, in certain earlier periods certainly, of deeper situation. The ulcer of epithelioma is more often defined and single, though multiple epitheliomata are not uncommon. The edges of the epitheliomatous ulcer are whitish or pearly, indurated, and everted; its floor uneven and glazed; its secretions scanty, and occasionally fetid; its base a mass of indurated tissue. Lupous ulcers are often ill-defined and multiple; their edges soft and inconspicuous, neither everted nor undermined; their floors granulating and flattened; their secretion relatively profuse and generally odorless; their bases soft and pliable, though occasionally indurated.

Nodular, serpiginous, and ulcerative lesions of syphilis often resemble certain forms of lupus. In any doubtful case, a history of infection, of other types of cutaneous disease, of mucous patches, of adenopathy, of abortions in women, and, finally, recourse to the Wassermann reaction, should aid in the recognition of syphilis. The structure of the lesion should be examined carefully for the purpose of distinguishing characteristic lupus nodules in the patch itself or in the periphery of any exfoliating area. In the case of an adult, a history of long-existing lupus may often be obtained; and it is worthy of note that syphilis with exceeding rarity displays for long periods of time a single exanthematous lesion or aggregation of such lesions exclusively in one part of the body. Their history of appearing in one place, undergoing the usual evolution, healing, and appearing in other areas is characteristic. Lupus ulcers, often multiple and isolated, insensitive, well or ill defined in outline (never reniform or horseshoe-shaped), with supple, low edges and reddish, smooth, hemorrhagic, granulating floor, covered with crusts like soiled parchment, of uniform thickness, do not resemble those of syphilis. The latter are often painful, single, circular, and clean cut in contour, with firm, raised, infiltrated margins, and with offensive, greenish and blackish crusts, resembling oyster-shells. The cicatrices of syphilis are elegant, smooth, delicate, superficial, circular, and, after pigmentation has disappeared, dead-white in color. Those of lupus are irregular, indurated, deforming, yellowish-white and reddish-yellow. Acquired syphilis is a disease of adult life; lupus commonly begins in childhood.

Patches of psoriasis are distinguished from the flat, exfoliating areas of lupus vulgaris by the relatively larger number of the former, by the nacreous luster of the scales, the reddish-hemorrhagic surface beneath, and the sites of election of the patches, usually on the extensor faces of the limbs. Finally, in the lupus patch nodular elements may be determined, and under the diascope the peculiar reddish-brown color of the nodule becomes evident.

Lupus erythematosus is readily distinguished by its characteristics, including the absence of nodules, ulcers, and crusts; the superficial character of the morbid process, the scaliness, and the occasional symmetry of the patches. An intermediate form between lupus erythematosus and lupus vulgaris has been described, but most cases so classed probably belong to the type called by Leloir *Erythematoid lupus vulgaris*, in which nodules are temporarily absent. In all such cases the nodules of lupus vulgaris develop sooner or later and confirm the diagnosis. The two diseases, unfortunately somewhat similar in name, are distinct in character. Here, again, lupus vulgaris begins, as a rule, in childhood, while lupus erythematosus is a disease of middle adult life.

In acne rosacea, with a bulbous condition of the tip of the nose, the redness is vivid; and the telangiectatic complications, with the seborrhoic flux, are conspicuous points of difference from lupus vulgaris. There is, further, no ulceration and little scarring, and the patients have usually suffered from the disease only after arriving at maturity. The mucous surfaces are also spared.

The diagnosis of verrucous growths of tuberculous nature is to be made after an investigation of the history of each case, which often includes a history of contact with cadavers or persons capable of communicating the disorder. The epitheliomatous, warty growths on the dorsum of the hands of elderly persons are not to be confounded with tuberculous lesions. In the former there is commonly a history of longer existence of the wart, and no record of suspicious contacts; while a careful search will usually determine epitheliomatous metamorphoses over the cheeks or temples of the elderly man or woman with epitheliomatous warts on the hands. In the latter, too, the facial lesions are usually multiple, fatty-looking scales, thicker in one part than another, resembling those of a severe seborrhea, but which are removed with difficulty, and which then leave a bleeding surface beneath.

The greatest difficulty in diagnosis is in distinguishing between tuberculosis verrucosa cutis and blastomycosis. In the latter condition the process is more actively inflammatory; it spreads more rapidly; the patches are apt to be greater in number; the periphery of the lesion has a more active, reddish halo, in which numbers of miliary abscesses are seen, and from which the organism of blastomycosis can be obtained for microscopic examination. The patch itself shows more evidence of moisture and suppuration, and in consequence several papillomatous as well as verrucous lesions are usually present. In any case, the demonstration of blastomycetes microscopically determines the diagnosis.

In the orificial cases it must be remembered that tuberculosis of the viscera is a probable coincident disease. The microscope usually is needed for an exact diagnosis.

In obscure cases of any variety, the tuberculin test should be employed (for details, see section on General Diagnosis), and if possible the Antiformin method used to examine for the bacillus. In many instances where the examination of sections has failed to reveal its presence this method has been successfully employed.

Treatment of Tuberculosis Cutis.—The general treatment of the patient is of paramount importance, and the rules laid down for the management of tuberculous patients apply to those suffering with the disease located in the skin. There is no specific internal medication. Several medicinal preparations have been used with benefit, largely on account of their tonic effect. Cod-liver oil, various preparations of iron and arsenic, the hypophosphites, and other preparations of this nature are often indicated. In London, thyroid extract has been given in cases of extensive tuberculous disease of the skin with apparent benefit. Probably the most important measures are those relating to diet and hygiene.

Patients of the tuberculous class manifest in the highest degree the beneficial effects of a change of residence and climate—to the seashore or mountains from the interior valleys or plateau-lands; or the reverse for those who reside by the sea. It is the change which seems to produce the greatest benefit. An abundance of pure air and a life permitting out-of-door exercise are of the highest importance. The thermal and other springs of several countries furnish resorts where the benefit received is proportioned to the salubrity of the climate rather than to the special advantages of the waters furnished. Unfortunately, a large number of the patients affected with lupus or scrofuloderma are impoverished inmates of public charities or applicants to dispensaries, where these aids in the management of their ailments cannot be utilized. But even in these cases, with the modern arrangements, an out-of-door life can be frequently realized.

The most satisfactory results in the treatment of lupus vulgaris are obtained with the Finsen light. Not only is the method successful in removing the disease in a large majority of cases, but the scars produced are much less disfiguring than those left by other methods, except the results obtained sometimes by radiotherapy, or in some of the circumscribed areas treated by Lange's plastic method.

Phototherapy has given the best results of any method yet employed. Forchhammer,[1] in a statistical report based on 1200 cases of lupus vulgaris treated at the Finsen Light Institute between 1896 and 1906, gives the following results of the treatment: cures, 60 per cent.; under treatment, 18 per cent.; treatment discontinued, 11 per cent.; dead, 11 per cent. Of the total cured (721), 33 had been free from recurrence for ten years or more, 289 for from five to ten years, 306 for from two to five years, 93 for less than two years. In a further study, the subjects are divided into initial cases and inveterate cases. In the initial cases 76 per cent. were cured, while in the inveterate cases 51 per cent. were cured.

Phototherapy has also been used extensively by Sequeira, Morris, Leredde, Gastou, Stroebel, Lesser, and Schmidt, and a few in the United States, including ourselves. While this treatment, when properly carried out, stands at the head of the list, it is not a practicable one in this country, on account of the expense involved, and for the reason that it requires hospital facilities.

Radiotherapy has also been used in tuberculosis cutis by Schiff and Freund, Kummel, Hollond, Knox, Pusey, and a number of other observers, including ourselves. The chief disadvantage in the latter method is the telangiectasia which frequently follows in the scar. Much less time is required to relieve the lesions by this method than by that of phototherapy.

With radium the results are very much the same as those achieved with x-rays. After treating eighteen patients with radium Haslund[2] concluded that the results were inferior to those achieved by other methods.

The thorn treatment employed by Unna gives excellent results. The thorns of the gooseberry bush are saturated in the German *liquor stibii compositus*, and one or more thrust firmly and deeply into each lupous nodule which it has been determined to attack. The base of each thorn is then cut off with a pair of fine scissors and the patch covered with a zinc-oxid plaster. When the thorns are cast off, a simple granulating ulcer is left, which in favorable cases heals without delay.

Holländer's hot-air treatment of lupus is accomplished by directing upon the lupoid tissue, through a metal tube of slender diameter, a stream of air at about a temperature of 300° C. The result is for the most part a destructive cauterization requiring complete anesthesia. The resulting scars may be formidable.

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[1] Die Bekämpfung der Lupus Vulgaris in Dänemark, 1896–1911 (Proceedings of the sittings of the Lupus Congress of the German Central Committee—zur "Bekämpfung der Tuberkulose"—Berlin, April 21, 1911 (abstr. Brit. Jour. Derm., 1911, xxiii, p. 338).

[2] Brit. Jour. Derm., 1916, xxviii, p. 294. Radium Treatment of Lupus Vulgaris. Review of literature.

Solidified carbon dioxid may also be used on small patches of lupus vulgaris with good results. Extensive patches are not well handled by this method.

A small galvano-cautery is useful for destroying small nodules of lupus vulgaris.

Boeck[1] recommended the following paste: pyrogalloli, resorcini, acidii salicylici, of each 7 parts; gelanthi and talci pulv., of each 5 parts; this made into a soft paste, which is applied by means of a spatula over the lupous patch and covered with a thin layer of cotton-wool. The paste dries quickly, and with the cotton-wool forms a permanent dressing, which is allowed to remain for a week or longer. White[2] employed this method in a number of cases with favorable temporary results, but recurrence followed. Doutrelepont[3] summarized his fifty years' experience with the treatment of lupus as follows: From a cosmetic point of view, no treatment is as satisfactory as the Finsen light. Surgical treatment has the danger of opening blood- and lymphvessels, thus favoring the spread of the tubercle bacillus, and should be employed only in case of small lesions which can be totally excised. New tuberculin is strongly advocated. He suggests beginning with 1 milligram, gradually increasing the dosage every second day. For local treatment, 1 per cent. sublimate poultices are advocated. Pyrogallic acid has also been useful in his cases. Fibrolysin proved of value in softening lupous scars. Strauss[4] used copper salts in the treatment of lupus by subcutaneous and intramuscular injections and by mouth with success. He often combined treatment with copper salts and iodomethylene blue.

Sequeira[5] describes a method of treatment (Pfannensteil's method) of intranasal lupus by the internal administration of sodium iodid, combined with the local application of hydrogen peroxid, as follows: The patient is directed to take 45 grains of sodium iodid daily, divided into six doses. Each morning the nose is cleansed with a nasal douche, using a solution of sodium chlorid and boric acid, and after drying the cavity is packed with sterilized gauze, which is kept constantly saturated by the patient with a 2 per cent. solution of hydrogen peroxid. This is done by means of a pipette. Excellent results are reported with this method.

For involvement of the mucous membrane, Sibley[6] recommends nascent iodin, both internally and locally, to the inside of the mouth. The patient takes 20 grains of potassium iodid in a tumblerful of water each morning before breakfast, followed four, six and eight hours afterward by one ounce of chlorin-water in a tumblerful of lemonade. The chlorin-water is prepared as follows: One dram of powdered chlorate of potassium is placed in a 24-ounce bottle, and 2 drams of strong hydrochloric acid is added and allowed to stand for twenty to thirty minutes. Then water, 4 ounces at a time, is gradually added, the bottle being well shaken between each addition of water, up to 24 ounces.

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[1] Monatshefte, 1909, xlviii, p. 441.

[2] Jour. Cut. Dis., 1910, xxviii, p. 532.

[3] Archiv, 1910, p. 191.

[4] Deutsch. med. Wochenschrift, March 13, 1913, xxxix, No. 11, p. 503.

[5] Brit. Jour. Derm., 1911, xxiii, p. 327.

[6] Ibid., 1914, xxvi, p. 363.

Locally, a powder consisting of equal parts of potassium iodid and chalk is blown, by means of an insufflator, on to the areas of ulceration in the mouth, after which it is rinsed with chlorin-water, this being held in the mouth as long as possible.

Savatard[1] reports the disappearance of lesions of lupus vulgaris under daily applications of a 5 per cent. tuberculin ointment. The lesions disappeared without apparent reaction.

Bruck and Glück[2] used with success a 1 per cent. aqueous solution of gold and potassium cyanate as an intravenous injection. From 1 to 3 c.c. of this solution in 50 c.c. of salt solution was injected every two to three days until twelve or more had been employed. A local reaction similar to that seen following tuberculin injections was noted.

Bernhardt[3] reports good results in the treatment of six cases of lupus vulgaris by the combined use of salvarsan and tuberculin injections; the former given in doses of 0.3 gramme each month; the tuberculin injections beginning with 0.5 milligramme, gradually increasing to 15 milligrammes, repeating the injections every four or five days.

MacKee,[4] in twelve cases of lupus vulgaris treated with tuberculin, obtained the following results: Three of the hypertrophic variety, without ulceration, recovered after from eighteen to twenty months' treatment; three of the ordinary non-ulcerating cases recovered after from eighteen months' to two years' treatment; a fourth of the same type was greatly improved; and a fifth of a similar type showed no improvement. Four cases of ulcerating lupus vulgaris failed to recover after two months' treatment. In three cases of tuberculosis verrucosa cutis improvement occurred in all. In scrofuloderma the results were disappointing.

In the earlier experience, using large doses of tuberculin, the results were disappointing and at time dangerous, but with the modern small and graduated dosage favorable reports are constantly being recorded.

A saturated solution of trichloracetic acid applied on lupous nodules with a cotton swab has recently been successfully employed by Heidingsfeld.[5]

Sodium phenate in saturated solution, suggested by Leslie Roberts,[6] has been employed by the author with promising results. This preparation induces local reaction, and is applied daily for several successive days until reaction occurs. The applications are renewed after the subsidence of each reaction.

Adamson[7] obtained excellent results in lupus by painting the patches with liquid acid nitrate of mercury.

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[1] Brit. Jour. Derm., 1912, xxiv, p. 81.

[2] Deutsch. med. Wochenschrift, 1913, lx, p. 57.

[3] Archiv, 1912, cxiv, p. 401.

[4] Jour. Cut. Dis., 1914, xxxii, p. 366.

[5] Trans. Amer. Med. Assoc., Sec. on Derm., 1914, p. 52.

[6] Personal communication.

[7] Brit. Jour. Derm. and Syph., 1920, xxxii, p. 383.

Prognosis.—The prognosis of tuberculosis of the skin in all its manifestations is in the highest degree variable. Many patients affected with lupus vulgaris, even after the production of the severest grade of deformity, recover and without further local manifestations gain a degree of facial comeliness that is marvellous. The scrofulodermata in the same way are remarkably improved, in the majority of all cases, by skillful medical and surgical management. In other cases systemic tuberculosis develops after even a single tuberculous infection, and grave results may occur either early in life or after years of tuberculous involvement of the skin and other organs. Other things equal, the prognosis in tuberculosis of the skin, as compared with that of other organs, is relatively favorable, due to the sparsity of tubercle bacilli in most cutaneous lesions, the skin being exposed too largely to external influences to form a good field for development of new colonies of bacilli. Any form of tuberculosis of the skin, however, may result in systemic infection and death.

ERYTHEMA INDURATUM.

Synonym.—*Erythème induré des scrofuleux* (Bazin).

Definition.—Erythema induratum is a chronic recurring disorder, usually involving the skin of the legs of young individuals, characterized by deeply situated nodosities and ulcerations.

It is probable that the disease as reported in the past represents three types: first, the common type described below, which is tuberculous in origin; second, an ulcerative process occurring at a more advanced age and on other regions of the body than the site commonly selected, and which is more painful, more amenable to treatment, and is probably due to vascular disturbances and is not tuberculous;[1] and, third, atypical examples now classed with the sarcoids.

Symptoms.—The beginning of the disorder is marked by one or several deep-seated nodosities located in the hypoderm, which gradually extend to the surface and undergo necrosis, producing ulceration, or, after absorption, atrophy. They are usually bluish-red in color, though they may be a vivid red. They are painless, as a rule, though pain may ensue after ulceration has occurred. The disease commonly attacks the calves of the legs of girls from fourteen to twenty years of age. Crocker has seen it in a woman of over fifty, but she had suffered with the disease earlier in life. It has also been observed, though not often, in boys and men. The front of the leg may be involved occasionally, and also the thigh and even the upper extremities (Crocker and Galloway).

The nodules are hard, and can often be detected by palpation when not visible. They vary in diameter from one-half to one inch or more. Node-like patches may also be present. The lesions are symmetrical, and may be few, but in time a greater number develop. The ulcers are irregular, ill-conditioned, with puriform contents, and tend to heal slowly, leaving scars. At a given time, there may be present nodules, ulcers, atrophic areas, and scars, some of these being relics of former attacks.

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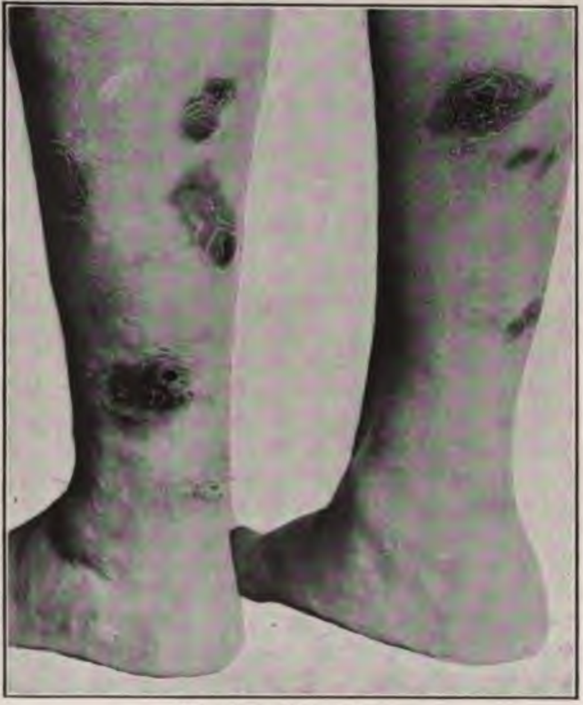
[1] Galloway, Brit. Jour. Derm., 1899, xi, p. 206; Whitfield, ibid., 1901, xiii, p. 386; idem, Amer. Jour. Med. Sci., December, 1901; Galloway, Brit. Jour. Derm., 1902, xiv, p. 199; Whitfield, ibid., 1905, xvii, p. 241; Galloway, ibid., 1913, xxv, p. 217.

The disease occurs, as a rule, in public practice and is comparatively rare. It has been observed in connection with tuberculides of the papulo-necrotic type; in patients having tuberculosis elsewhere in the skin;[1] and also in patients having tuberculosis in situations other than the skin.[2]

FIG. 270



Erythema induratum.

Etiology.—The affection occurs most frequently in the winter, and much more commonly in the female sex in the second decade of life. Washerwomen and shop-girls, who stand much, are liable to it. Its subjects often have a weak peripheral circulation, evidenced by cold, blue hands or a chilblain tendency. The tubercle bacillus, in the light of recent study, plays an important role in the type cases.

Pathology.—From the studies of Thibierge,[3] T. Colcott Fox,[4] Mantegazza,[5] and others, the tuberculous nature of the disease seems well demonstrated, although inoculation experiments are usually negative and the presence of *Bacillus tuberculosis* difficult to demonstrate. A granuloma with giant- and plasma-cells common to tuberculosis has been demonstrated repeatedly. Successful inoculations into guinea-pigs have been made by Gilchrist, Fox and Eyre, Thibierge, and Ravaut, and evidence is constantly accumulating which adds strength to the theory of the production of the common type of this disorder by *Bacillus tuberculosis*. In a case of the non-tuberculous variety, Galloway found a granuloma in the deeper part of the corium and subcutaneous tissue. The cells were mononuclear plasma-cells, a very few polymorphonuclear, and a large number of giant-cells. He differentiates these giant-cells from those occurring in tuberculosis. Whitfield also calls attention to the points of distinction in the histology between these cases and the common type, found even in the presence of giant-cells.

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[1] Fordyce, Jour. Cut. Dis., 1911, xxix, p. 444; MacKee, ibid., 1913, xxxi, p. 568.

[2] Crocker, Dis. of the Skin, 3d ed., p. 812.

[3] Annales, 1899, s. iii, x, 513.

[4] Brit. Jour. Derm., 1900, xii, p. 383 (Report on the Tuberculides, presented to the Fourth International Congress of Dermatology and Syphilis).

[5] Annales, 1901, s. iv, ii, p. 498.

Diagnosis.—Erythema nodosum and syphilis are the two diseases most likely to cause confusion in the diagnosis. From the former, erythema induratum is distinguished by its chronic course, its tendency to ulceration, the absence, as a rule, of pain, lack of fever, and other constitutional symptoms, the presence of scars, and its frequent association with other evidences of tuberculosis. From the gummatous syphiloderm, it is differentiated by the symmetry of the lesions, and by the absence of other evidences of syphilis; finally, it is not benefited by specific treatment.

In the major number of cases in which the tuberculin test has been employed, it has proved positive.

Treatment.—The general tonic treatment indicated in tuberculosis should be used in most cases. Rest in bed, with elevation of the limbs, is recommended. Before ulceration, bandaging should be practised, and local antiseptic dressings after this last has occurred. The disease has responded unusually well to treatment with tuberculin.[1]

LICHEN SCROFULOSORUM.

Synonym.—Lichen scrofulosus.

Definition.—This disorder, first described by Hebra (see his remarks before the German Surgical Society, Fourteenth Congress), is characterized by the occurrence of indolent papules, situated chiefly on the trunk, and occurring in tuberculous subjects. The lesions are millet-seed- to pinhead-sized papules, firm in consistency and flat topped. In color they vary from that of the normal skin to light or vivid red. The papules are occasionally surmounted by minute scales, rarely by an equally small pustule. At the outset the lesions are isolated. Later, they tend to arrange themselves in coin-sized patches. Often there is a crescentic outline formed by the aggregation of individual lesions. When evolution is accomplished, they are closely set together, the surface of the skin then being of a dirty, reddish-brown hue and covered with thin scales, which are readily detached. The course of the eruption is slow. Often the cutaneous symptoms persist for months without apparent change, awakening little or no itching, and are followed by involution, accompanied by slight desquamation and no cicatrices. There may be recurrence.

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[1] Whitfield, Brit. Jour. Derm., 1905, xvii, p. 241; Clark, Jour. Cut. Dis., 1909, xxvii, p. 567; Fordyce, ibid., 1911, xxix, p. 444; MacKee, ibid., 1913, xxxi, p. 568.

Etiology and Pathology.—In 99 per cent. of all cases observed in Austria, there was concomitance of the general symptoms of struma (evidenced by submaxillary, cervical, and axillary adenopathy, periostitis, or ulcerative dermatitis), with frequent complications, such as eczema of the scrotum. The disease was encountered in young tuberculous subjects between the periods of infancy and puberty, rarely after the twentieth year. Crocker[1] has noted its frequent occurrence in children in whom he suspected tuberculous pleurisy. Jadassohn[2] believes that it is a disease of the tuberculous, and not of the cachectic generally, and obtained typical reactions in fourteen out of sixteen cases injected with tuberculin. He has seen the disease disappear after these injections. As to the question of its toxic or bacillary origin, opinion is divided. In favor of the former theory, Schweninger and Buzzi,[3] Fox,[4] Klingmüller,[5] and others have seen a disease apparently identical with it produced by tuberculin injections. It has been suggested that the injections may have stimulated a latent tuberculosis into activity, but in the histological study of a case thus produced Porges[6] found merely changes of an inflammatory character, with no evidence of a tuberculous structure. In a case of lichen scrofulosorum in a negro child, Gilchrist[7] found a granuloma deeply situated, while the folliculitis which produced the clinical symptoms was more superficial.

According to Kaposi, the disease consists of an exudative infiltration of the pilo-sebaceous follicles and perifollicular tissue. Each papule represents, therefore, the orifice of a follicle, with an infiltrated perifollicular annex; and its apical scale or pustule is formed of a mass of epithelial débris or an inflammatory exudate. Porges found areas of tubercular foci composed of round, epithelioid, and giant-cells in the corium. The vessels showed perivascular inflammation, with cellular infiltration about the sweat-ducts. Jacobi,[8] Wolff,[9] and Pellizarri[10] have been successful in finding the bacillus in lesions or in producing inoculation tuberculosis in guinea-pigs.

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[1] Dis. of the Skin, 2d ed., p. 448.

[2] Trans. of the Internat. Cong. of Derm. and Syph., London, 1896, p. 425.

[3] Quoted by Brocq., 20th Century Medicine, iv, p. 359.

[4] Brit. Jour. Derm., 1900, xii, p. 384.

[5] Ibid., 1904, lxix, p. 167. A *résumé* of his work and observations at the clinic of Professor Neisser. In 17 cases of lichen scrofulosorum his findings relative to tubercle bacilli were negative and animal experiments also failed. In a small proportion of the cases a tuberculous structure was present, but in the major part no suggestion of tuberculosis was noted. The result of this work strongly suggests the toxic rather than the local bacillary origin of the disease.

[6] Archiv, 1903, lxvi, p. 401.

[7] Johns Hopkins Hosp. Bull., 1899, x, p. 84.

[8] Verhandl. der Deutsch. derm. Gesell., Third Congress, 1891, p. 69.

[9] Ibid., Sixth Congress, 1899, p. 486.

[10] Trans. Internat. Cong. Derm. and Syph., London, 1896, p. 425.

Diagnosis.—The disease is differentiated readily from papular eczema by the absence of itching. From the miliary papular syphiloderm it differs in that the lesions of the latter, even though grouped, are always individually distinct. The general symptoms, moreover, are strikingly different in the two diseases. Lichen scrofulosorum should not be confounded with lichen planus or pityriasis rubra pilaris.

Treatment.—The malady produces but little inconvenience, and, moreover, yields readily to therapy. Hebra advises cod-liver oil internally and externally. Crocker advises liquor plumbi subacetatis grains 15 (1.), thymol grains 5 (0.33), to vaseline 1 ounce (30.), to be applied externally, with the administration of cod-liver oil internally.

Prognosis.—The prognosis is favorable.

TUBERCULIDES (DARIER).

Synonyms. — Toxituberculides (Hallopeau), Paratuberculoses (Johnston).

Definition.—Under the title Tuberculides, Darier[1] classed a number of cutaneous affections which appeared to have many characteristics in common. These diseases, or rather cutaneous manifestations of disease, have, as a rule, been observed in individuals the subjects of tuberculosis in organs other than the skin, or who have hereditary tuberculous tendencies. Hallopeau and others have suggested that they are due not to the local action of *Bacillus tuberculosis*, but to the toxins floating in the circulation from a distant focus. Fox[2] says that if they are due to the local action of Koch's bacilli, they must be few in number, of little virulence, and readily destroyed. Darier included in this category acne cachecticorum or scrofulosorum, disseminated or agglomerated folliculitis, acnitis, folliclis, hydrosadenitis destruens or suppurativa, granuloma innominatum, and disseminated erythematous lupus (Boeck). Fox, in his report on the tuberculides to the International Congress in Paris, in 1900, included among others in the list acne varioliformis, necrotizing chilblains, lichen scrofulosorum, and erythema induratum scrofulosorum (Bazin). According to Fox, "the essential lesion of the group of tuberculides is a small, extremely indolent granuloma, tending to undergo central softening and necrosis, and thus leaving scars. They are bilateral and symmetrical. The great clinical variation depends upon the depth at which the derma is affected, the implication or freedom of the glandular apparatus, the bulk of the granuloma, the distribution and number of the lesions, and the absence or presence of pustulation or necrosis." The subjects of this disorder often have a feeble peripheral circulation and are usually not robust. Two or more of these various lesions have frequently been noted in the same patient: for instance, lesions of the folliclis type on the upper, with erythema induratum on the lower, extremities, or acneform lesions with lichen scrofulosorum. A patient whose case was reported by Johnston[1] had lesions on the arms which Johnston termed "necrotic granuloma," and others on the limbs which he termed "indurated erythema." Darier reported a case in which tuberculosis was present in the lungs and elsewhere, with tuberculides of the type of acne cachecticorum on the body, folliclis on the knees and extremities, and a tuberculous gumma on the leg. Little[2] showed a case before the London Dermatological Society with acneform and gummatous tuberculous lesions present at the same time. Fox notes that large gumma-like lesions often are associated with acneiform symptoms elsewhere.

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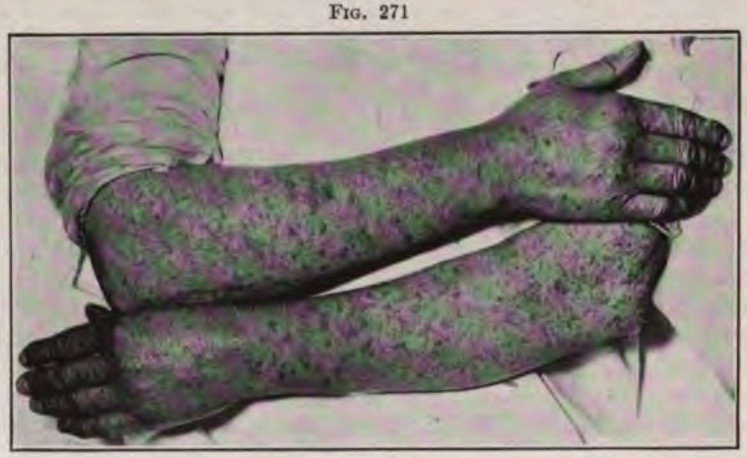
[1] Annales, 1896, s. iii, vii, p. 1431.

[2] Brit. Jour. Derm., 1900, xii, p. 383 (Report on the Tuberculides, presented to the Fourth Internat. Cong. of Derm. and Syph.).

FIG. 271



Tuberculides.

Symptoms.—These vary, as described above, according to the type of lesions present. In general, the disorders are chronic, the lesions deep-seated, beginning usually in the hypoderm or corium, extending into and involving the surface. They are at first colorless, later bluish- or brownish-red or lighter in shade. They may suppurate, forming a pustule in the center of the lesion. The latter dries into a depressed crust, which, when shed, leaves a small cicatrix; or ulceration may occur, leaving a small, depressed scar; or the lesion, nodule, or papule may be absorbed, leaving some atrophy with pigmentation. The lesions often are grouped and appear in successive series. Different types show a predilection for different parts of the cutaneous surface. For example, lesions of the acnitis type select the face, and those of the folliclis type, as a rule, select the extremities. The lesions are generally painless and do not itch, and there may be a large number or only a few exhibited.

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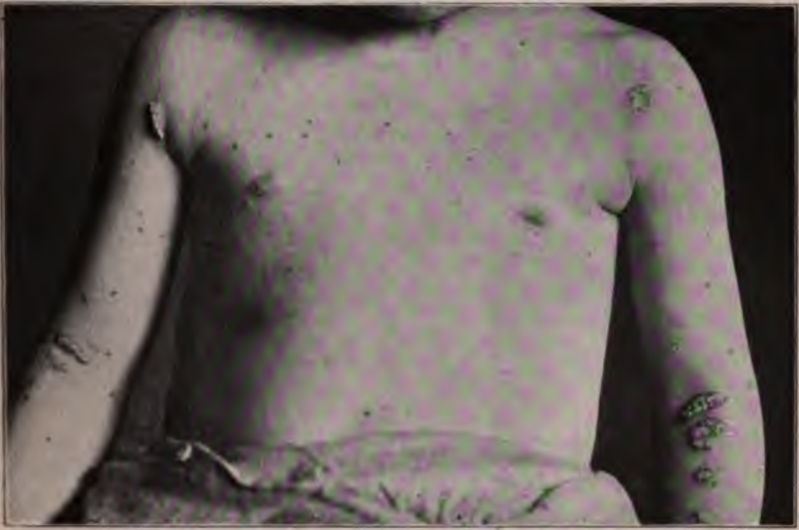
[1] Philadelphia Med. Jour., 1899, iii, p. 443.
[2] Brit. Jour. Derm., 1902, xiv, p. 352.

The above description applies to the group of papular and nodular tuberculides as a whole, while the following description of types is given for their proper identification in this group.

Acnitis.[1]—This variety Crocker[2] describes under the title *Acne agminata*. Here the lesions select chiefly the face. They occur in distinct groups in different regions, especially upon the cheeks below the eyes, the upper lip, the chin, and the forehead. The lesions are usually brownish-red in color, though many appear semitranslucent and almost colorless. They vary in size from that of a pinhead to that of a pea, and are firm to the touch, and occasionally the small papules or nodules are capped with a vesicle or pustule. The lesions are prone to remain for a considerable time, then undergo involution, leaving a small, pigmented scar, which gradually becomes less conspicuous. In some cases involution occurs rapidly when once initiated.

FIG. 272



Generalized tuberculide; small papular and verrucous lesions.

Folliclis.—In this variety the favorite sites are on the hands, forearms, feet, and legs, though the face may be attacked. Lesions on the palms and hands were noted in a case recorded by Wise.[3] The trunk seldom is affected. Here the lesions pursue a more rapid course, commonly completing their cycle in from four to six weeks. They usually are noted first as red spots, which later develop into vesicles or pustule-capped papules or nodules. They are firm to the touch and painless. The pustules dry into crusts, which reveal on exfoliatio small cicatrices. While the lesions are usually discrete, patches may occur. The disease is chronic in its course. Barthélemy[1] reports a case lasting ten years, which had exacerbations, the patient never being entirely free. In the case of a patient examined by us, the affection had lasted for four years and appeared worse in the early autumn. This case illustrated the fact pointed out by Crocker, that the lesions on the fingers are more indolent and firm, and apparently have hard centers surrounded by a rim of pus. The disease occurs, as a rule, in persons having tuberculosis or with tuberculous antecedents.

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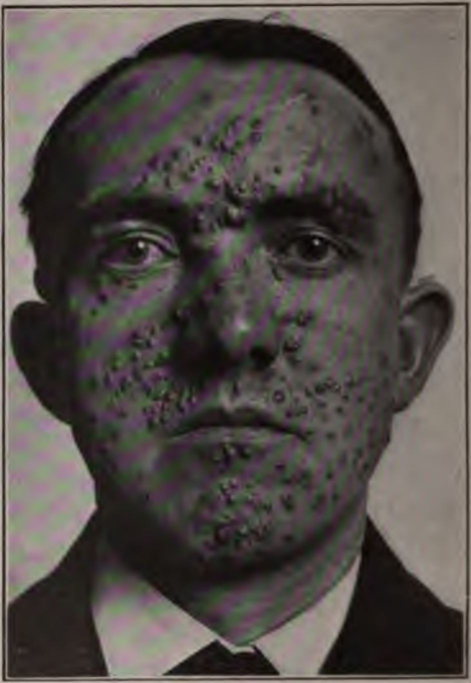
[1] Schamberg, Jour. Cut. Dis., 1909, xxvii, p. 14 (a study of acnitis with report of an extensive case). Oliver, Brit. Jour. Derm., 1914, xxvi, p. 439. A case of Acne Agminata (Crocker); Ketron, Johns Hopkins Hosp. Bull., April, 1915; Knowles, Jour. Cut. Dis., 1917, xxxv, p. 61. Acnitis in the negro.

[2] Dis. of the Skin, 3d ed., p. 1164.

[3] Jour. Cut. Dis., 1914, xxxii, p. 805.

FIG. 273



Acnitis. (Dr. Quinn's case.)

A disorder very similar if not identical with folliclis is describ under the title *Acrodermatitis pustulosa hiemalis* by Crocker.[2] H description, taken from three cases, is as follows:

"The lesions are excited or kept up by the cold, affect the han only, especially about the knuckles and sides of the fingers, and ta the form of indolent, indurated papulo-pustules, isolated and f in number at a time; but the disease as a whole persists by a successi of lesions throughout the winter and early spring.

"They begin as hard, brown, large-pinhead points, but appear la as if there was a 'thorn in the flesh.' If opened early, serum escapes, but later pus forms around the peg and the whole is situated on an elevated, inflammatory, pea-sized base. The center is cast off, leaving a scar. Some of the indurated nodules do not suppurate."

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[1] Quoted by Crocker, Dis. of the Skin, 3d ed., p. 1169.

[2] Dis. of the Skin, 3d ed., p. 350.

A patient exhibiting lesions of this type on the hands and tuberculous glands in the neck was presented before the Chicago Dermatological Society by the author.

Acne Scrofulosorum.[1]—This type corresponds to the small, nodular tuberculide (Darier), and is more commonly seen in London than elsewhere.

This eruption occurs, as a rule, in patients suffering with tuberculosis of the glands or other forms of the disorder. According to Fox, the eruption is an indolent, small, papulo-pustular or acneform one, sparsely disseminated, without grouping, and unaccompanied by subjective symptoms. The lesions occur on the external aspects of the limbs, more particularly the lower, and about the buttocks and the region immediately above. They appear successively or by subacute outbreaks. The papules are inflammatory, are situated about a hair-follicle, are acuminate, and capped with a small pustule, which dries up, and often shells out with a central follicular plug, leaving a cicatrice. On undergoing involution, the lesions flatten and become more irregular in outline, simulating lichen planus, and upon their disappearance leave stains or faint scars.

Acne Cachecticorum of Hebra occurs in the so-called scrofulous individuals and is often combined with lichen scrofulosorum (Kaposi). The lesions occur abundantly upon the trunk and lower limbs; less so upon the face. They are indolent papulo-pustules, pinhead- to bean-sized, livid, purplish- or lurid-red, or of a violaceous tint. The lesions usually are soft, pus-and-blood-containing nodules, having a sluggish career, and leaving minute cicatrices. They closely resemble lesions produced by syphilis, but are distinguished from the latter by the absence of induration and the fact that they do not produce characteristic ulcers.

Etiology.—In a large number of patients presenting these lesions some form of tuberculosis is present. Hempelman[2] found tuberculides of the papulo-necrotic type in 30 infants under two years of age out of a series of 130 affected with pulmonary phthisis. Sometimes only a hereditary tendency is manifested. Occasionally, no evidence of tuberculosis can be demonstrated. While the disease may occur at any period of life, the most common age is, according to Fox, between twenty and forty years. The tuberculides are not infrequently seen in children. Morris[3] has observed that measles is a frequent forerunner of the disease. The patients are prone to have a weak peripheral circulation. Both sexes are attacked. By experimental work, Rist and Rolland[4] proved that in animals already the subject of tuberculosis, the tubercle bacilli introduced from without were rapidly destroyed by some substance present in the tissue. Lewandowsky[1] produced lesions resembling tuberculides by injections of tubercle bacilli into tuberculous animals, and agrees with the conclusions of Rist and Rolland that the tuberculides are caused by the tubercle bacillus reaching the skin by the blood stream and there being destroyed by immune substances.

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[1] Colcott Fox, Brit. Jour. Derm., 1895, vii, p. 341: On Acne Scrofulosorum in Infants.

[2] Archiv of Pediatrics, 1917, p. 362.

[3] Dis. of the Skin, 1903, p. 422.

[4] Sequeira, Brit. Jour. Derm., 1915, xxvii, p. 371; "A new conception of tuberculides; an account of the work of Rist and Rolland."

Pathology.—The symmetry of the lesions and the early involvement of the bloodvessels point to some irritant brought to the cutaneous surface through the general circulation. In different cases different anatomical structures are affected more or less severely, which naturally alters the histological picture, and has in consequence led to confusion in nomenclature. The relationship of the tuberculides to tuberculosis is generally conceded, but opinion is divided as to the exact nature of this relationship. Histologically, a tuberculous architecture has been demonstrated clearly a number of times by different observers. Many cases, however, merely show the changes incident to a simple inflammation. Inoculation experiments have, on the whole, been negative, and the presence of *Bacillus tuberculosis* in the lesions has rarely been demonstrated. The vascular changes in these conditions are evidenced by an endophlebitis, which often appears as the earliest phenomenon. Giant-cells in the tuberculous architecture have been demonstrated in different members of this group by Galloway,[2] MacLeod,[3] Darier, Leredde,[4] Bureau,[5] Little,[6] Adamson,[7] Burnett,[8] and others. Pollitzer,[9] Fordyce,[10] Unna,[11] Dubreuilh,[12] and others have noted special involvement of the coil-glands in some cases, but even this Leredde and Bureau consider secondary to a granuloma of tuberculous origin. In a histological study of two cases, one of the acneform type, the other the so-called scrofulous gummata, the author with MacLeod[13] found in both cases giant-cells and a typical tuberculous structure, with two tubercle bacilli in the latter. In a case of tuberculide of the papulo-necrotic type, Whitfield[14] found one tubercle bacillus microscopically, and he is of the opinion that the tuberculides are rarely due to the presence of bacillus in the situation. Gougerot and Laroche[15] produced tuberculides of the papulo-necrotic type and lichen scrofulosorum in a large number of instances in guinea-pigs by rubbing into an epilated surface of the healthy epidermis a virulent culture of the tubercle bacillus.

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[1] Archiv, 1916, cxxiii, p. 1.

[2] Brit. Jour. Derm., 1901, xiii, p. 17.

[3] Ibid., p. 367.

[4] Quoted by Crocker, Dis. of the Skin, 3d ed., p. 1167.

[5] Ibid., p. 1169.

[6] Brit. Jour. Derm., 1901, xiii, p. 185.

[7] Ibid., 1908, xx, p. 406: Histology of five cases of small, nodular tuberculide (acne scrofulosorum type).

[8] Ibid., 1910, xxii, p. 312.

[9] Quoted by Unna, Histopathology, p. 399.

[10] Jour. Cut. Dis., 1891, ix, p. 128.

[11] Histopathology, p. 399.

[12] Arch. de Méd. expér. et. d'Anat. path., 1893, s. i v, p. 63.

[13] Brit. Jour. Derm., 1901, xiii, p. 367.

[14] Ibid., 1913, xxv, p. 168.

[15] Arch. de Méd. expér. et d'Anat. path., September, 1908, v, p. 581.

Treatment.—The treatment of the general health is important in the majority of these cases. The hygienic surroundings should be regulated properly. Fresh air, sanitation, and good food are of prime importance. Cod-liver oil, iron, and other tonics, according to the special indications, should be used. In folliclis, Crocker advises thiol, grains 5 (0.33), three times daily, with applications of 10 per cent. vasogen iodin. Antiseptic washes, such as boric-acid solution or bichlorid of mercury (1 to 5000), may be necessary. Excision may at times be employed. Mercurial plaster or an ointment containing ammoniated mercury (1 to 30) may be applied. Radiotherapy has been of distinct value in several cases treated by us. Much improvement or entire relief has been reported by Clark[1] following injections of tuberculin. On the other hand, MacKee[2] treated twelve cases of tuberculide of the papulo-neurotic type without benefit.

Arsphenamin was of value in a group of cases treated by Stokes.[3] In 53 per cent. of 17 cases the lesions were entirely cleared up with this treatment.

LUPUS ERYTHEMATOSUS.

Synonyms.—Lupus sebaceus, Lupus superficialis, Seborrhea congestiva, Lupus erythematodes, Ulerythema centrifugum. Fr., *Scrofulide erythémateuse*, *Erythème centrifuge*.

This disease was first described by Biett,[4] under the title *Erythème centrifuge*. Hebra,[5] in 1845, described it under the title *Seborrhea congestiva*. Its present title was given by Cazenave,[6] in 1850.

Definition.—Lupus erythematosus is usually a chronic, sometimes an acute, disease of the skin, characterized by the presence of erythematous, scaling patches, of various sizes and configurations, which induce superficial atrophy and scar-formation. It is capricious as to course, and not infrequently terminates fatally in certain of its forms. The following varieties deserve individual description: The common or discoid variety; the diffuse or disseminated variety, which may be acute at the onset and usually terminates fatally, or which may begin like the chronic form, later becoming disseminated, and then may end fatally; the telangiectatic variety; and, finally, the variety described as lupus pernio.

Symptoms.—**Discoid Variety.**—The early lesion is usually exhibited as one or several rape-seed- to bean-sized, slightly elevated, reddish macules, which do not entirely fade under pressure, and are covered with a grayish or yellowish, and sometimes slightly greasy, adherent scale.

The primary lesion enlarges peripherally, and in the course of months or years may assume the size of a small coin or a large saucer.

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[1] Jour. Cut. Dis., 1909, xxvii, p. 567.

[2] Ibid, 1914, xxxii, p. 366.

[3] Amer. Jour. Med. Sci., 1919, clvii, p. 522, clinical studies in cutaneous aspects of tuberculosis.

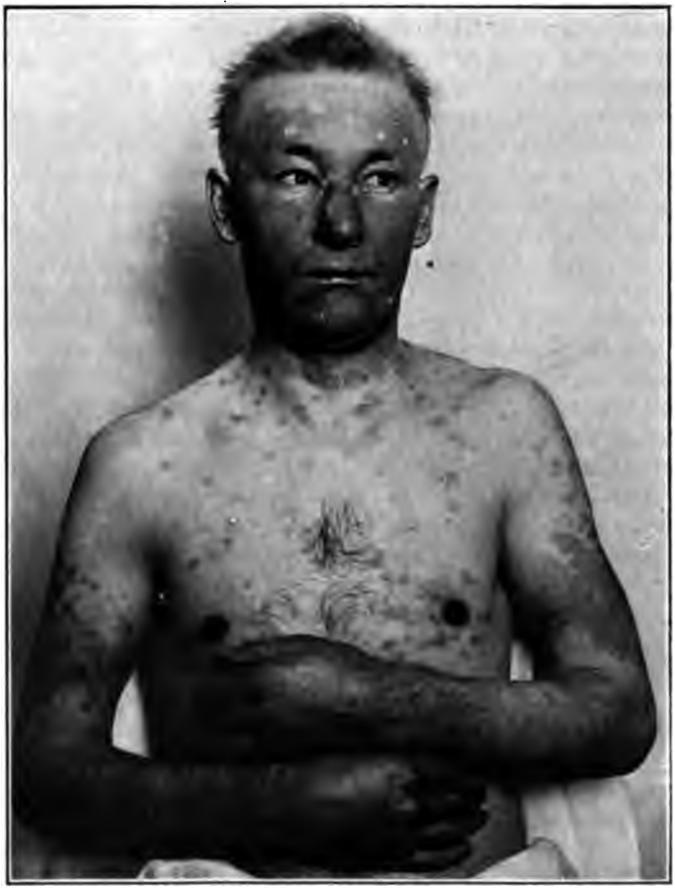
[4] Abrégé Pratique des Maladies de la Peau, Paris, 1828.

[5] Hautkrankheiten, Bd. iii, Thiel i.

[6] Annales des Maladies de la Peau, 1851, iii, p. 298.

The patches are well defined in outline, of a color varying with the complexion of the patient and with the acuteness or type of the disease from a rosy-pinkish to a deep-purplish hue. The shape is usually circular, oval, or in figures representing combinations of these outlines, and it may be irregular from the junction of two or more progressing patches. Its border is red, firm to the touch, and distinctly elevated, and it not infrequently exhibits comedones or light, adherent scales. The center is depressed, paler in color, and shows either adherent, yellowish-gray scales or a glistening, unbroken epidermis. Close examination will disclose in most cases dilated follicular openings, which may be plugged with dry sebaceous matter or horny epithelium. The scales vary in color, being at times of a clear white or whitish-yellow, and again often, from concurrence of comedones, of a reddish or brownish tint. They are usually scanty

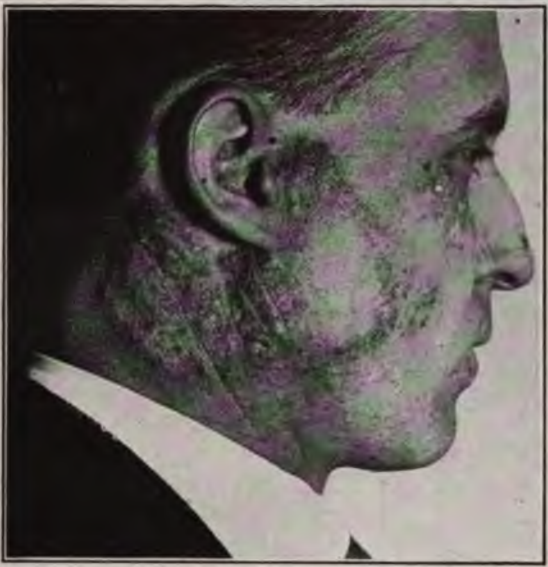
Fig. 274



Generalized lupus erythematosus.

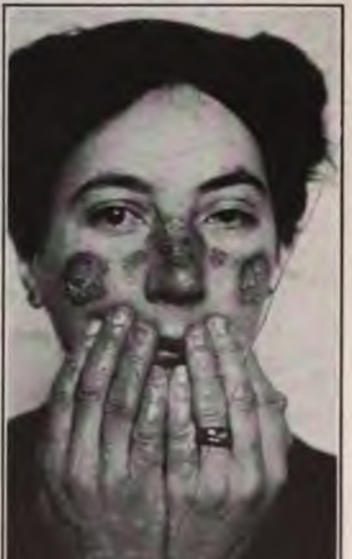
and adherent, but may be abundant, and occasionally may be seen firmly fastened to the orifice of the excretory duct of a sebaceous gland by means of a horny projection from the under surface. In some cases the erythematous redness predominates; in others the crusted, seborrhea-like feature is more pronounced. In the latter there are seen at times patches exhibiting almost a pure type of seborrheic dermatitis of the face.

FIG. 275



Lupus erythematosus.

FIG. 276

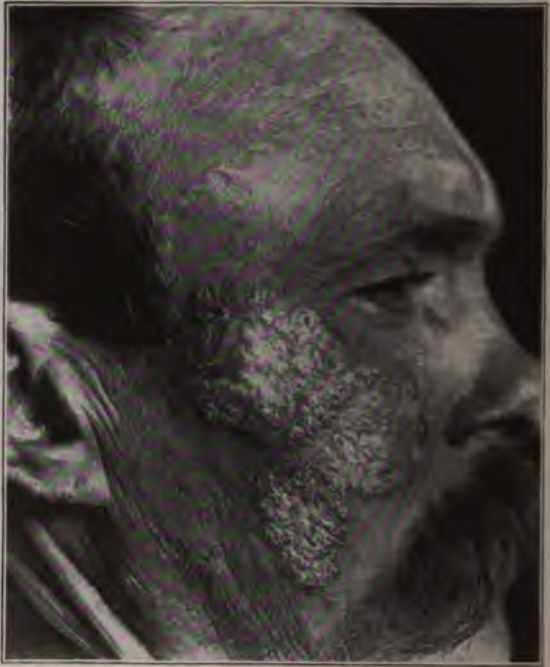


Lupus erythematosus of face and hands.

The disease may spread symmetrically over the nose and cheeks in a form that has been likened by Hebra to the open wings of a butterfly. As the borders advance, the center not infrequently undergoes involution and may show typical scars even while the outer rim is actively progressing. When the disease undergoes general involution, both the center and the border gradually become paler in color and less elevated. Some of the patches resolve without leaving a trace of their existence, but in most instances typical scars are left. These are indelible and characteristic. They are generally uniform and superficial, can be pinched up readily between the thumb and finger, are of a dull, whitish tint, and are rendered punctate in a peculiar manner, suggesting the action of the engraver's tool in what is known as the "stippling" process. They are never pigmented, puckered, radiate, stellate, corded or deeply attached.

The disease is seen most frequently on the different parts of the face, ears, and scalp, but may occur on any part of the body. On the scalp small, irregular patches or larger areas may appear. There is here usually more infiltration and more pronounced scar-formation, but less color and less elevation of the border, than in lesions of the face. The dilated follicles and comedones are often pronounced. Th alopecia which results is permanent. After involution of the lesion a smooth, slightly depressed, delicate and atrophic scar remain At times the scalp alone may be involved. On the hands[1] and fe the disease may occur in the usual form or as a persistent erythem with slight scaling, but it more commonly begins as a lupus pernio.

FIG. 277



Lupus erythematosus (seborrheic type).

The mucous membrane may be involved, presenting redden plaques with minute excoriations, or be partially covered with a whitish exudate or with punctate scars. The early lesion is ill defined, reddish-violet, and slightly infiltrated. Later the lesions present the marbled appearance due to scar-formation. On the tongue the papillæ are lost, and the area becomes reddened without infiltration. When the lips are affected, they have the appearance, as described by Dubreuilh, of having been painted with collodion, which has desquamated. The disease on the mucous membranes is not uncommon. Smith[1] found it present in approximately 28 per cent. of 56 cases.[2]

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[1] *Cf.* paper by Dr. Hyde, with 35 tabulated cases in which the hands were affe (Jour. Cut. Dis., 1884, ii, p. 321).

Subjective sensations are usually slight or absent, but some itching or burning may be present during periods of activity in the lesions. The disease is markedly chronic in its course, lasting in cases for a quarter of a century or longer, and throughout that time not interfering with the general health. Though the disease usually progresses by very slow extension of the border, it may, after remaining comparatively stationary for months or years, rapidly advance for a short period and then again remain stationary. These periods of rapid progression usually follow or are accompanied by a peculiar type of acute dermatitis.

Lupus Erythematosus Disseminatus.—In this variety numerous lesions with different characteristics occur. The early lesions are usually seen on the face, spreading from there over the neck, trunk and extremities. Rarely it may become generalized. The lesions are split-pea to dollar-size, erythematous, slightly infiltrated, scaling patches, some of which persist for some time, while others disappear spontaneously. Moderate atrophy is present in some of the lesions, whereas others disappear without having induced any change in the skin. In another type large areas of the surface are involved, particularly about the face, neck, or trunk, which are more persistent, less well defined, and by their fusion diffuse areas may become involved. Exceptionally the eruption may resemble multiform erythema (See Fig. 274). Mild itching and burning sensations may be present, particularly when new lesions are appearing. Telangiectasia may also be present. Scaling is more marked on the face and hands than elsewhere. Rarely all the lesions are like those of the discoid type; such a case is recorded by Johnston.[3] In rare instances an acute form occurs, accompanied by constitutional disturbances. Pernet[4] recorded such a case and incorporated 9 others from the literature. Roberts,[5] Sequeira and Balean,[6] Barber,[7] and others, have recorded cases of this type. These begin with erythematous patches on the face which blend and cover the cheeks, nose, eyelids and ears. The face is edematous and swollen and in some instances crusting occurs early. In certain of these the face assumes an erysipelatous aspect, which accounts for the description of the disease by Kaposi under the title "*Erysipelas perstans facei.*" The eruption involves the hands and may also extend over other areas. On the hands it may be erythematous, papular or purpuric; on other portions of the body the eruption is multiform. Bullæ and vesicles, scaling patches and crusted areas have all been described. The temperature is elevated, arthritic pains occur, and there may be gastro-intestinal disturbance. In two fatal cases of this type observed by the author the eruption on the body was of the multiform erythema type. In the series collected by Roberts, ten of the eleven cases terminated fatally. Albuminuria is common in this type; Sequeira and Balean found it present in 5 out of 10 cases. MacLeod[1] recorded an instance of a case associated with nephritis, which terminated fatally. The lesions in these were situated on the face and hands. As the toxic symptoms from the nephritis increased the eruption declined.

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[1] Brit. Jour. Derm., 1906, xviii, p. 59.

[2] Kren, Archiv, 1907, lxxxiii, p. 13 (Report of 4 cases, with histology in 2, and review of literature of cases involving mucous membranes).

[3] Jour. Cut. Dis., 1912, xxx, p. 96.

[4] Le Lupus erythemateux aigu d'emblée; Thèse de Paris, 1908.

[5] Brit. Jour. Derm., 1911, xxiii, p. 167; Acute lupus erythematosus (aigu d'emblée).

[6] Ibid., 1902, xiv, p. 366.

[7] Ibid, 1915, xxvii, p. 365.

Telangiectatic Variety. — This form is rare. Here points, spots, plaques, or large disks on the surface, chiefly of the face, usually well defined, present a rosy-reddish or deep-purplish color, which deepens under pressure. When examined with care, the color is seen to be due to dilatation of the cutaneous vessels. The surface may be either slightly edematous or infiltrated and correspondingly elevated. There is an absence of scaling and of dilated follicles, but typical scars not infrequently follow involution of this type of the disorder. The course of the disease in this form is slow and the condition may remain for years without change.

Lupus Pernio.—This is another unusual form, in which the lesions are exhibited on the fingers and toes particularly, but also on other parts of the hands and feet and on the pinna of the ear, beginning as a more or less persistent erythema type of pernio (chilblain). Like the latter disease, this erythema may disappear and reappear with the seasons for years, but eventually may persist and assume the discoid type.

Etiology.—The disease is more common in women than in men, the ratio being two-thirds of the former to one-third of the latter, and usually appears first in the third decade of life, in this particular presenting a contrast to lupus vulgaris. It may, however, first develop in childhood, middle life or old age. We have seen several instances in children. Stowers,[2] Crocker,[3] Galewsky,[4] and others describe the disease occurring in children, and in these analyses girls were in the majority.

Lupus erythematosus may follow dermatitis seborrhoica, acne, variola, erysipelas, vesication with cantharides, undue exposure to the sunlight, or traumatism from any cause. It may be associated with chilblains;[1] with Raynaud's disease;[2] with symmetrical gangrene;[3] with erythema iris;[4] and it is sometimes seen in association with lupus vulgaris.[5] It may appear where the curette has been employed in the case of a patient with a characteristic lesion on the face. It occasionally develops on portions of the face and hands which have been subject to recurrrent attacks of pernio; and it is generally accepted that enfeebled circulation and local irritation are prominent causal factors. By an increasing number of writers the disease is considered a chronic inflammation due to a toxic infection, the exact nature of which is not known. Galloway and MacLeod[6] believe that lupus erythematosus, like erythema multiforme, may be due to toxins arising from various causes. The association of the disease with multiform erythema is frequently observed. Recent observations suggest a focal infection as the probable source of intoxication in some instances. Barber[7] recorded the case of a patient in whom he regarded a streptococcal infection in the tonsils as important etiologically. In a patient observed by Hartzell[8] great improvement followed the removal of an infected tooth. The relation of lupus erythematosus to tuberculosis has been a much disputed question, and although lupus erythematosus has none of the essential characteristics of a local tuberculosis, it occurs not infrequently as a dermatosis of the tuberculous. Besnier was the first to call attention to the fact that lupus erythematosus was in many instances associated with general or local tuberculosis. Cases in which this association occurred have been reported by a number of observers, and Boeck[9] records 42 cases of the common discoid type, in 28 of which he found evidences of present or past tuberculosis. Roth[10] collected records of 250 cases of lupus erythematosus, in 185 of which evidence of local or general tuberculosis could be obtained. Sequeira and Balean[11] found the discoid variety associated with tuberculosis in 18 per cent., and that there was a history of tuberculosis in the family in about 40 per cent. In the disseminated form, the presence of tuberculous disease was noted in 70 per cent. of the patients, and there was a history of tuberculosis in the family in 80 per cent. Pick,[1] after studying the effects of tuberculin injections in 29 cases, concluded that lupus erythematosus discoides is not a manifestation of tuberculosis.[2] Acid-fast bacilli have been found in tissue of lupus erythematosus treated by the antiformin method by Arndt, Hidaka, Spiethoff, and Friedlander. Successful animal inoculations relative to tuberculosis have been made from cases of lupus erythematosus by Gougerot, Ehrmann, Reines, and Block and Fuchs.

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[1] Brit. Jour. Derm., 1908, xx, p. 162.

[2] Brit. Jour. Derm., 1908, xx, p. 236.

[3] Ibid.

[4] Archiv, 1907, lxxxiv, p. 193 (abstr. Brit. Jour. Derm., 1908, xx, p. 26).

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[1] West, Brit. Jour. Derm., 1907, xix, p. 448; Perry, ibid., 1896, viii, p. 223; Adamson; ibid., 1903, xv, p. 287.

[2] Pringle, ibid., 1905, xvii, p. 306; Hartzell, Amer. Jour. Med. Sci., cxliv, p. 793, Engman and Mook, Interstate Med. Jour., April, 1909, p. 267.

[3] Dawson (Brit. Jour. Derm., 1911, xxiii, p. 182) reported a case with lesions on the face, ears, scalp, and backs of the hands and fingers, associated with symmetrical gangrene of the fingers, tip of the nose, and ears.

[4] Sequeira, Brit. Jour. Derm., 1910, xxii, p. 237.

[5] Kyrle, Archiv, 1909, xciv, p. 309; MacKee, Jour. Cut. Dis., 1913, xxxi, p. 568; Roberts, loc. cit.; and others.

[6] Brit. Jour. Derm., 1903, xv, p. 81: "Erythema Multiforme and Lupus Erythematosus: Their Relationship to General Toxemia;" and ibid., 1908, xx, p. 65: "The Relationship of Lupus Erythematosus and Erythema Multiforme; with an illustrative case." (Complete discussion of the subject with literature and conclusions.)

[7] Brit. Jour. Derm. and Syph., 1919, xxxi, p. 186. A case of lupus erythematosus associated with a streptococcal infection of the tonsils.

[8] Archiv of Derm. and Syph., 1920, ii, p. 441. Lupus erythematosus and focal infection.

[9] Archiv, 1898, xlii, p. 71.

[10] Ibid., 1900, li, p. 3.

[11] Brit. Jour. Derm., 1902, xiv, p. 367.

Lupus erythematosus is frequently associated with varieties of tuberculosis of the skin. Roberts[3] quotes the case of a young woman twenty-five years of age suffering with tuberculous glands of the neck, lupus vulgaris of the face, and lupus erythematosus of the fingers. Kyrle[4] reports the occurrence of lupus erythematosus and lupus vulgaris in the same patient. Many other instances could be quoted. The author has in several instances seen lupus erythematosus of the discoid type associated with glandular enlargement of the neck. Similar cases are recorded by Sequeira,[5] and others. Pringle[6] believes that the connection between lupus erythematosus and tuberculosis is very close. Occasionally, disastrous results may follow the injection of tuberculin in cases of lupus erythematosus.[7]

Tuberculosis can thus be accounted an important factor in the etiology of lupus erythematosus, but that it is the whole cause, or even an essential factor in most cases, has not been demonstrated. It is associated more frequently with the disseminated than with the discoid form of the disease.

From the evidence produced, it would appear that the disease is of toxic origin, the nature of which is unknown, but probably from various sources. External irritation and local injury furnish exciting causes for the localization of the process, and circulatory changes play an important part.

Pathology.—Lupus erythematosus has been studied carefully by a number of observers, but, unfortunately, they do not agree either in their histological findings or in their conclusions based upon the latter. In the ordinary discoid variety, it may be said that the chief changes are found in the upper half or third of the corium in the form of a dense infiltration of small round cells of embryonic type, a small proportion of which is probably the result of proliferation of the fixed cells of the part. The infiltration varies greatly in extent and in density in different types of lesions, but is most pronounced along the course of the vessels. It is often found in slight degree in the deeper parts of the corium and subcutaneous tissue; but it nowhere forms nodules as in lupus vulgaris; there are no giant-cells; and there is no degeneration of a mass of cells, as in the latter disease. Individual cells here and there undergo a granular and fatty or colloid degeneration, disappear by absorption, and are replaced by new cells. The connective-tissue fibers are destroyed in the same way. Many of the vessels are seen to be greatly distended and choked with red blood-corpuscles, others show a proliferation of their walls, and in some cases an obliterating endarteritis. Diffuse or localized hemorrhages are found in the upper part of the cutis. The sebaceous glands are at first hypertrophied, affected with hypersecretion, and become filled with cells and abnormal sebaceous matter. Later both they and the ducts of the coil-glands may become infiltrated, undergo degeneration, and disappear, leaving the peculiarly punctate form of scar characteristic of the disease.

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[1] Archiv, 1901, lviii, p. 358 (with bibliography).

[2] For discussion on the relationship of lupus erythematosus to tuberculosis, see article by Freshwater, Brit. Jour. Derm., 1912, xxiv, pp. 57 and 99; Friedlander, Jour. Cut. Dis., 1911, xxix, p. 417 (with extensive bibliography); Arndt, Berlin klin. Wochenschrift, 1910, xxix, p. 1360; Block and Fuchs, Archiv, 1913, cxvi, p. 742 (an extensive review of the literature concerning the etiology of lupus erythematosus).

[3] Loc. cit.

[4] Loc. cit.

[5] Brit. Jour. Derm., 1913, xxv, p. 197.

[6] Ibid.

[7] Pringle, ibid., p. 197; Saville, ibid.; and Ravogli, Jour. Cut. Dis., 1915, xxxiii.

The epidermal layers are involved secondarily. They become atrophied, and the interpapillary depressions of the rete as well as the papillæ are largely obliterated.

Fordyce and Holder[1] investigated a number of cases of the discoid type and describe a peculiar blocking of the capillaries with blood-cells, which they believe to be the primary change. They divide the factors making up the histological complex into the round-cell infiltration, the peculiar degenerated condition of connective tissue, and the secondary atrophy. They find that the commonly described fatty and granular degeneration is not characteristic of the process. Schoonheid,[2] from a histological study of twelve cases, concluded that lupus erythematosus is a chronic inflammatory process, and describes a peculiar degeneration and destruction of the elastin, which he believes to be the immediate cause of the superficial scars. Warde[3] regards the profound stasis in the lymph- and blood-channels as the most important feature in the histology of the disease. Höllender[4] considers the pathologic process to have its origin in the glands of the skin.

Robinson,[5] after examining a number of cases and reviewing the published reports of others, states that the primary lesion, which may be seated in any part of the corium, is focal in character, and when fully developed constitutes a new-growth, which is reticular in structure and closely connected with the lymph-channels. He concludes that "lupus erythematosus is a chronic inflammatory disease of the cutis with special histological characters, as shown by the changes in the bloodvessels, by reticular tissue, and by the presence of mononuclear and the absence of polynuclear cells in the cell-infiltration; and that these changes must depend upon the presence of a poison generated *in loco*. In other words, lupus erythematosus is a local infective process—a granuloma."

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[1] New York Med. Record, 1900, lviii, p. 41.

[2] Archiv, 1900, liv, p. 163.

[3] Brit. Jour. Derm., 1902, xiv, p. 447.

[4] Berlin klin. Wochenschrift, 1903, xxx.

[5] Trans. Amer. Derm. Assoc., 1898, p. 70.

Roberts[1] found in an acute case of the disseminated variety the following histological changes: The vessels and lymphatics of the corium were dilated, and surrounded by perivascular cell-infiltration, consisting of mononuclear leukocytes, connective-tissue cells, and a few small and large plasma-cells. The reticular layer of the corium was rarefied. Elastin was present. There was atrophy of the epidermis.

In mucous membranes, Pautrier and Fage[2] describe the histology as follows: The changes in the region of the lip showed an irregular thickening of the epidermis and cornification of the mucosa. In the corium there occurred a cellular infiltration, consisting of lymphocytes and connective-tissue cells, a considerable number of plasma- and a few mast-cells. There was dilatation of the bloodvessels. The collagen and elastin were normal, except in the areas of densest infiltration, where they were rarefied. The lesions on the tongue showed acanthosis. In the corium the vessels were dilated, and there appeared to be newly-formed capillary loops, and in addition a superficial cellular infiltration, with lymphocytes, connective-tissue cells, and a few plasma-cells.

Diagnosis.—When lupus erythematosus is present on the face the picture presented is usually so characteristic that the disease is there recognized with ease. When the hand and other portions of the body are involved, the diagnosis is somewhat less readily established. In the hand the disease has a predilection for the dorsum, and invades the palm usually only by extension to it from behind.

From lupus vulgaris, erythematous lupus may be recognized by its occurrence originally at a later period of life; by its greater tendency to symmetry; and by the absence of nodules, ulceration, and extension to the deeper portions of the skin or underlying structures. Cases undoubtedly occur in which the diagnosis is difficult, as in the type called by Leloir *lupus vulgaire érythématoïde*. But, as in all cases of lupus vulgaris typical nodules appear sooner or later, the diagnosis can eventually be established.

In eczema there is usually some history of moisture; in erythematous lupus, rarely. In eczema, also, the itching is a more persistent and distressing symptom; but the acuteness of even chronic eczema, as compared with lupus erythematosus, will suffice to distinguish the two diseases. From dermatitis seborrheica, however, the diagnosis may be difficult and may have to depend on a therapeutic test, the latter disease disappearing under appropriate treatment. Psoriasis is rarely, if ever, limited to a single patch on the face; it is also characterized by more lustrous and more readily exfoliating scales. Its patches are, furthermore, uniformly well covered with scales, and are of equal flatness in all parts; while those of lupus erythematosus are irregularly squamous, the scales being often clustered at the orifices of the ducts of the sebaceous glands, while the rim of the patch is elevated and the center depressed. From pernio the diagnosis sometimes can be made only after determining whether the lesions disappear during the warm season, as in pernio, or persist, as in lupus erythematosus.

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[1] Loc. cit.

[2] Annales, 1909, x, p. 673.

In acne rosacea there are marked telangiectases and papulo-pustules or nodules, which are not found in erythematous lupus. In trichophytosis corporis there may be a clearing, but never a cicatriform center of the circular disk. The circular serpiginous syphilodermata of the face occur usually with other manifestations of lues, are characterized by greater infiltration and a more rapidly progressing border, formed by the coalescence of individual papules, or nodules; and in most cases the syphilitic lesions exhibit distinct signs of ulceration. The not infrequent modification or masking of a patch of the disease by an acute or subacute dermatitis (often seborrheic in character) should be borne in mind.

Treatment.[1]—The internal treatment of this affection is not highly satisfactory. Frequently, none is indicated or required. The general health of the individual should be carefully investigated and all defects remedied, if possible. For the local or discoid variety, iodoform in 1 grain (0.06) doses (Whitehouse); ichthyol in from 2 to 5 mms. (0.13 to 0.33); salicin, 10 to 15 grains or more three times daily, and quinin in increasing dosage are all of value. Other preparations, such as potassium iodid, mercuric iodid, starch iodid, arsenic, ammonium carbonate, and sodium salicylate, have their advocates, but are only beneficial in certain instances.

The number of remedies recommended for local use in lupus erythematosus is enormous. White,[2] in reviewing the subject, has enumerated some fifty of those most promising, at the same time calling attention to the fact that lupus erythematosus is no exception to the rule that "the curability of a disease is in inverse ratio to the length of the list of the means recommended for its cure." He admits that our treatment of this disease is wholly empirical and not very hopeful. Unna[3] attempts a rational form of treatment based on his conception of the etiology and pathology of the disease and of the action of certain remedies. He calls attention in particular to the fact that, while the epidermis is exceedingly dry and hyperkeratotic, the cutis is markedly edematous and the seat of dilated lymph-spaces and channels, and emphasizes the dangers of stimulating a dry, indolent process into an active dermatitis.

For convenience, the remedies used may be divided into three classes: the soothing and astringent, the stimulating, and the destructive. The choice of remedies will depend largely upon the type of the disease and on the character of the individual skin. In the acute, inflammatory, or vascular type, soothing remedies alone should be used, and on a skin which reacts readily to stimulation stronger remedies are not allowable. Nor should it be forgotten that the indolent forms of the disease not infrequently under treatment become acutely inflamed, and call for the temporary use of soothing measures. Inasmuch as the affection is one the involution of which occasionally is accomplished under the influence of mild topical applications, and is succeeded very rarely by grave sequels, the simpler measures should always be adopted first. The zinc-oxid and aqua-calcis lotion, also the calamine lotion, are usually beneficial. For continued stimulation and astringent effect, the lotio alba may be employed, the formula being as follows:

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[1] Wise, Jour. Cut. Dis., 1917, xxxv, p. 500; Practical Observations on Lupus Erythematosus and its Management. *Cf.* This article for critical review of present methods of treatment.

[2] Jour. Cut. Dis., 1898, xvi, p. 457.

[3] Ibid., p. 465.

℞—Zinci. sulphat.,				
Potassi sulphuret.,	āā	ʒss;	2	
Alcoholis,		fʒiij;	12	
Aq. rosæ,		f℥iijss;	105	M.

Sig.—To be diluted as required for external use.

In case the lotions are too dry, a mild salicylic acid or sulphur ointment may be employed temporarily. These remedies are employed particularly in the seborrhoic variety of the disease. Frequently, much can be accomplished through protection and compression of the surface by the application of collodion, the glyco-gelatins, celluloid cream, or tragacanth jelly. Unna recommends especially for irritable cases:

℞—Ichthyol. (vel ichthyol. sulfon.),	ʒss;	2	
Collodii,	ʒv;	20	M.

For more indolent cases:

℞—Saponis mollis,	ʒss–ij;	2–4	
Collodii,	ʒv;	20	M.

To the latter may be added one or two parts of salicylic acid.

Unna recommends also gelanthum as a substitute for collodion in the above formulæ, for though it does not produce as much compression as the latter it is more convenient, in that it may be washed off at any moment with warm water. A favorite formula with him is potass. hydroxid., 1; gelanthum, 1000.

Stimulation of the areas must always be produced with great care, on account of the possibility of causing spread of the disease. In indolent cases, this may be done by the use of sulphur, in the strength of from 2 to 20 per cent.; salicylic acid in the strength of 1 to 5 per cent., or ammoniated mercury, resorcin, ichthyol, or tar. The mild salicylated soap-plasters or plaster-mulls containing the above remedies in small quantity may be used where a moderate amount of stimulation is desired. Good results follow the use of green soap applied as a paste or in the form of tincture. It not only cleanses the patches of scales, but also stimulates the surface, often to the extent of producing a reparative process. When decided irritation of the parts is induced, the soap should be discontinued and soothing treatment alone employed. The application of hot water for a few minutes daily is frequently of value. After drying, the surface should be dusted with powder.

A combined internal and external treatment has been devised by Höllender,[1] the medicaments used being quinin and tincture of iodin. His method is as follows: Seven and one-half grains (0.5) of quinin or quinin sulphate are given three times a day. Coïncidently, each area of the disease is thoroughly painted with tincture of iodin. After five or six days a rest from treatment is taken until the scale from the application has peeled off. Several courses of treatment as above may be needed, though sometimes one suffices. Höllender regards his method as specific and of value in differential diagnosis. Excellent results have been reported by many observers. Before applying the treatment the patient should be tested for any idiosyncrasy against quinin.

Phenol and lactic acid have been employed successfully by Small[2] as follows: The part to be treated is washed with ether, then a few drops of a mixture of phenol one part and acid lactic fort. four parts are vigorously rubbed on by means of a glass rod for two to three minutes. An inflammatory reaction follows. Treatment is repeated after a week or more.

The treatment of lupus erythematosus with phototherapy, radiotherapy, radium,[3] carbon-dioxid snow, and liquid air has been successful in a number of cases, but with all of these methods great care is necessary to avoid producing injurious results in this erratic disorder. After the employment of all the methods above mentioned recurrences happen, even when good results have apparently been achieved. In properly selected cases they may all be used with benefit. *X*-rays have given best results in those cases having a marked seborrheic development; radium is very valuable in indolent cases. The quartz lamp is also of much value in this type; and carbon-dioxid snow and liquid air in those cases of discoid type that have become stationary and are indolent in character. The high-frequency current, also, has many supporters, but, as a rule, it is, like the above mentioned measures, of value only in certain cases.

In indolent patches, where decided stimulation or a superficial destruction of tissue is desired, the carbon-dioxid snow is of great value; or, in the place of that, the stronger salicylated soap-plasters and plaster-mulls are recommended, or creosote, phenol, chrysarobin, pyrogallol, silver nitrate, lactic acid, or Fowler's solution may be used. Two drachms (8.) each of iodin and potassium iodid, mixed with 4 drachms (16.) of glycerin; or equal parts of chloral, tincture of iodin, and phenol, are recommended highly. These stronger remedies, however, are to be used with great caution and only in indolent cases, and then only after milder measures have failed to produce good results.

The treatment by ionization has been successful in the hands of many.[4]

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[1] Berlin. klin. Wochenschr., July, 1902.

[2] Brit. Jour. Derm., 1919, xxxi, p. 136. Lupus Erythematosus. A simple method of treatment.

[3] Simpson, Trans. Amer. Med. Assoc., Derm. Sect., 1914, p. 289.

[4] Hartigan, Brit. Jour. Derm., 1908, xx, p. 81; Davis, ibid., 1909, xxi, p. 325; Crocker, ibid., p. 293; and others.

Many authors report results of treatment with tuberculin. This method is not recommended, and dangerous results have followed its employment in this disease, whether from the standpoint of diagnosis or treatment. The local application of tuberculin in the form of inunction has also been followed with negative results.

Prognosis.—A favorable opinion with respect to the future of the disease can never be given safely, on account of the capricious nature of the disease and the constant recurrences and relapses which characterize it. In the fixed or discoid type, the general health and comfort of the patient suffer rarely. The affection is capricious in its course, and may on occasions, after long periods of persistence, rapidly improve under the simplest treatment. Spontaneous involution, with disappearance of all symptoms, is reported in some cases. Its tendency to the production of persistent scars should always be remembered in formulating a prognosis. Numerous instances of the development of carcinoma upon the scar of lupus erythematosus have been reported.

MULTIPLE BENIGN SARCOID (BOECK).

Synonyms.—Benign miliary lupoid (Boeck); *Lupoïdes miliares dissemineés; Sarcoïdes noueuses et nodulaires des membres* (Darier).

Definition.—A disorder characterized by the formation of nodules and plaques, both cutaneous and subcutaneous, having a benign course and terminating after resolution of the lesions in atrophic, scar-like areas.

The disorder described under the above title was first recorded by Boeck.[1] Many examples have since been recorded, several of which have been in America, by Gottheil,[2] Pollitzer, G. H. Fox and Wile, Howard Fox, Sweitzer,[3] Sweitzer and Michelson,[4] the author and others.

Many cases of similar nature were recorded under different titles and their nosological position was undetermined. Darier[5] collected these and after an extended study subdivides the reported cases into four groups, as follows: (1) multiple, benign sarcoid or miliary lupoid (Boeck); (2) subcutaneous sarcoid (Darier-Roussy); (3) sarcoid nodularis of the extremities, the type resembling erythema induratum (Bazin); (4) sarcoid of Speigler-Fendt. For the present the group will be considered together, though the fourth member belongs elsewhere. The first three should be classed together and are probably the result of infection by the tubercle bacillus.

Symptoms.—The type originally described by Boeck occurs as nodules, papules, and infiltrating plaques. The nodules may be few or numerous; may be firm and elastic; are round, oval, or irregular in shape; and are of a reddish or purplish color. At times they show delicate peripheral telangiectases, and occasionally slight scaling. The papules may be few or very numerous (Hallopeau and Zych). The plaques are similar to the nodules, and occupy the entire thickness of the skin, being movable over the underlying structures. As a rule, they are much larger than is apparent from inspection. The lesions usually occur upon the face, back of the shoulders, and extensor aspects of the arms.

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[1] Jour. Cut. Dis., 1889, xvii, p. 543.

[2] Ibid., 1902, xx, p. 400.

[3] Trans. Sec. on Derm., Amer. Med. Assoc., 1914, p. 262; "Sarcoid of Boeck," with references.

[4] Jour. Cut. Dis., 1919, xxxvii, p. 98; "Sarcoid of Boeck and Erythema Induratum."

[5] Monatshefte, 1910, i, p. 419.

The color of the early nodules is bright red, later becoming darker, and finally a yellowish-brown. Under pressure the nodules are seen to be composed of grayish-yellow foci, which characteristic suggested the name of "miliary lupoid." On the face the lesions have a peculiar appearance, with a blue center and a yellowish border. The nodules finally disappear, leaving, as a rule, atrophy and distinct scar formation, which may be white on the face, yellow on the back, and darker at the periphery of the lesions on the legs. Occasionally, the regional glands are swollen, but, as a rule, they are not involved. Exudation and ulceration never take place. The disease runs a benign course and responds to treatment with arsenic.

A case demonstrated by the author at the thirty-eighth annual meeting of the American Dermatological Association presented sarcoid tumors on the face and neck of a female patient, who had suffered with lupus erythematosus for twenty-three years before the beginning of the sarcoid. The lesions in this case were plaques and nodules involving the entire skin, movable, indurated, of irregular contour, varying in size from that of a coffee-bean to a large nut, bluish- or brownish-red in color, and painless. On undergoing involution, which occurred spontaneously, distinct atrophy was left. New lesions appeared as the older ones cleared up. The duration of the sarcoid at the time of presentation was three years.

The association of sarcoid and lupus erythematosus was noted in cases reported by Kingsbury,[1] Trimble[2] and others.

In the second group, described by Darier and Roussy[3] as subcutaneous sarcoid, the lesions are described as developing slowly, and usually without subjective sensations. The nodules are deeply situated, rounded or oval in contour, and sometimes occur in chains following the bloodvessels. As a rule, they are limited to various areas on the trunk and are imperfectly symmetrical. They vary in size from that of a hemp-seed to a large nut, and in number from two to twenty or more. There is no disturbance of the general health. The lesions commonly develop to a certain size and remain stationary.

The third group described by Darier includes those cases which resemble clinically erythema induratum of Bazin. The cases of this group are more common than those of either of the two first mentioned types. They occur chiefly in adult women, and present hazelnut-sized, reddish or purplish tumors, the infiltrations on the arms and legs occupying largely the extensor surfaces. In these cases the disease runs a chronic course. Occasionally, ulceration supervenes.

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[1] Jour. Cut. Dis., 1918, xxxvi, p. 533.

[2] Ibid., p. 592.

[3] Archiv de Méd., 1906, xviii, p. 1.

In the fourth group (Spiegler-Fendt type) the lesions occur as multiple tumors situated in the skin, usually on the trunk, which are of a lilac or deep red color. They are painless and at times may be of large size Their course may be rapid or slow, and they frequently respond to arsenical medication. Spiegler's case resulted in death after metastases.

Mixed cases occur in which lesions belonging to one or more of the above types may be seen in a single patient. It appears to be a fact that certain cases of atypical erythema induratum have in reality been sarcoid tumors. A case under the observation of the author[1] was an example of the mixed type and study of the case showed marked evidence of tuberculosis.

Etiology.—The chief factors in these cases have been to determine the role played by the tubercle bacillus. Darier considers the disease to be a tuberculide. In a few instances tuberculosis has been reported in inoculated animals, and tubercle bacilli have been demonstrated in one case. Most of these have belonged to the group described as resembling erythema induratum. Tuberculin reactions have been positive in a few instances. Strumpke[2] recorded a case of sarcoid in extensive generalized tuberculosis. Pautrier[3] suggests that certain cases of sarcoid, particularly of the Darier-Roussy type, owe their origin to syphilis. The major portion of cases have occurred in females.

Pathology.—In the Boeck type, sharply circumscribed, deeply situated nodules occur, which are separated from one another by connective-tissue septa. The cellular infiltration is composed chiefly of epithelioid connective-tissue cells. The nuclei are large and vesicular, less deeply stained, and show distinct nucleoli. In some instances true giant-cells of a sarcomatous type are seen. In early stages a few mast-cells are present, and occasionally leukocytes are detected. The infiltration apparently develops from the perivascular lymph spaces, but in certain foci appears to be derived from the corium itself, independent of the vessels. A marked tendency throughout the entire corium to connective-tissue-cell proliferation is noted. The elastic fibers are absent throughout the new growth. In the surrounding tissue they are normal. Dilatation of the capillary loops of the papillæ occurs, which accounts for the bluish color in the network in the center of the older patches. Changes in the epidermis are purely secondary. Immediately over the nodules, the interpapillary prolongations are obliterated from pressure.

In the Darier-Roussy type, the histological picture resembles that described above, but presents a greater number of giant-cells and a greater resemblance to the picture seen in tuberculosis. The infiltration is more diffuse, and the nodules are composed of epithelioid cells, lymphocytes and giant-cells.

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[1] Jour. Cut. Dis., 1919, xxxvii, p. 626.

[2] Derm. Zeit., 1913, xx, p. 199; abstr., Jour. Cut. Dis., 1914, xxxii, p. 160,

[3] Annales, 1914, v, p. 344; "Sarcoids and Syphilis,"

The histopathology of the third group conforms closely to that found in tuberculosis, though tubercle bacilli have been demonstrated in only one case.

In the fourth type, aggregations of round-cells enclosed in a capsule of connective tissue are seen. Giant-cells and epithelioid cells occur in small numbers. In this type the condition closely resembles the lymphogranulomata and, as stated earlier, does not properly belong with the sarcoids.

Diagnosis.—The various sarcoids are to be distinguished from leukemia cutis, nodular lupus erythematosus, and erythema induratum of Bazin. In view of the probable identity of the third type with the latter disorder a distinction is not necessary. By reference to the symptoms characteristic of these diseases it will be seen that a differential diagnosis is usually possible, although a microscopic examination is frequently necessary. The history of the cases of the various types of sarcoid and their characteristic histopathology distinguish them from other disorders.

Treatment.—In the Boeck type arsenic is frequently of service. Under this medication 2 patients under the observation of the author have entirely recovered. With this therapeutic agent the lesions are gradually absorbed, leaving atrophy and pigmentation. The drug should be given in moderate-sized doses over a long period of time to get the best results. The deeper types commonly do not respond to this treatment. Not infrequently the Spiegler-Fendt type, which is an entirely different disorder, responds to arsenic.

Radiotherapy is of some service. Darier reports successful treatment with *x*-rays, calomel and tuberculin. In Unna's case[1] lesions treated locally with ichthyol underwent involution more rapidly than those untreated.

Prognosis.—As a rule, the prognosis is good as regards life, but should be guarded relative to the disappearance of the lesions. In many cases they disappear, leaving only moderate disfigurement, while others persist. In rare instances a fatal termination has resulted, but in these other factors were interposed.

SYPHILIS.

Synonyms.—Lues venerea, Morbus gallicus, Pox, "Bad disorder." Fr., *Syphilis;* Ital., *Sifilide;* Ger., *Lustseuche, Krankheit der Franzosen.*

Definition.—Syphilis is a chronic, constitutional, infectious and contagious disorder, hereditary or acquired, which may attack any tissue or organ of the body, is characterized by symptoms referable to the part attacked, and is produced by *Spirocheta pallida* or *Treponema pallidum.*

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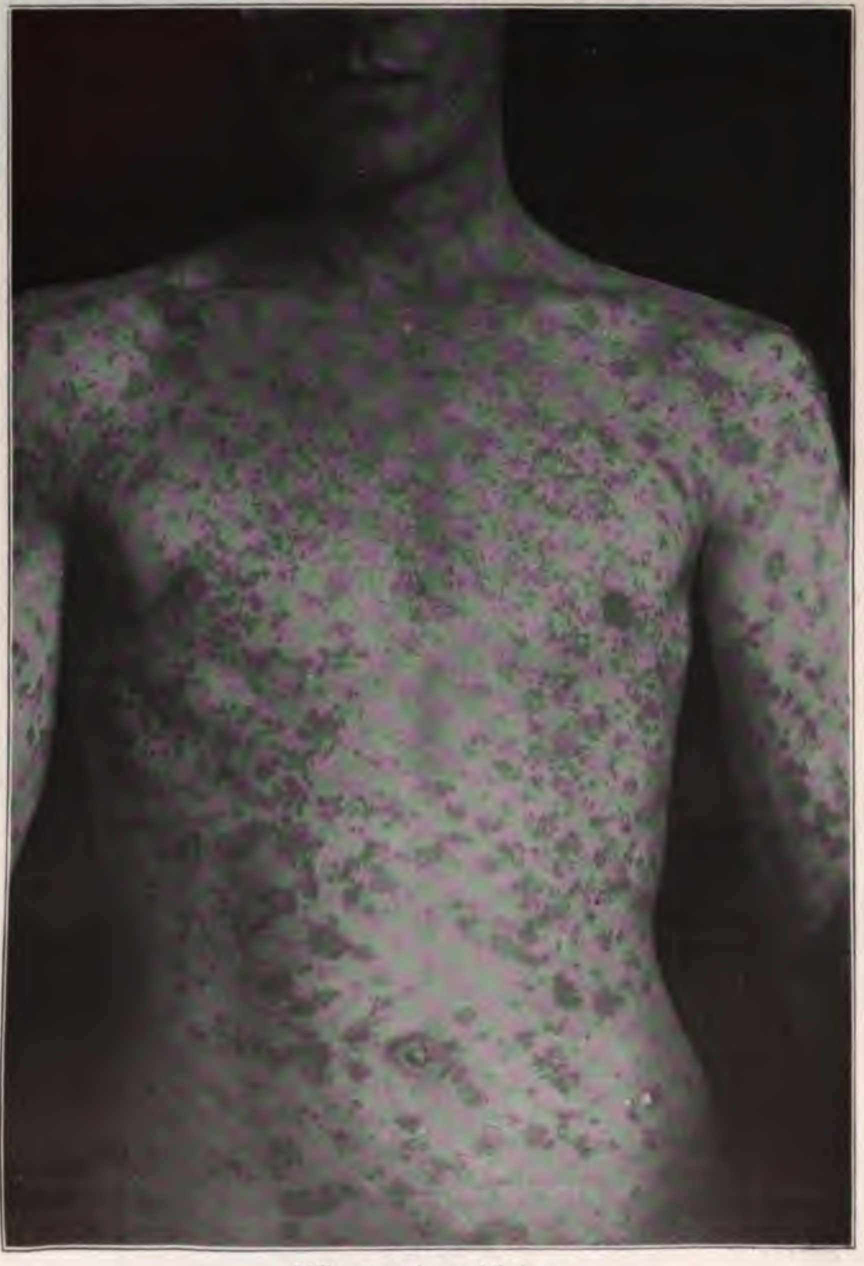
[1] Derm. Wchnschr., 1912, lv, p. 1203.

In acquired syphilis, the first evidence of its existence is the initial sclerosis or chancre, which appears about twenty-one days after

Fig. 278



Miliary papular syphiloderm.

infection. This interval, ordinarily described as the first incubation period, may extend over a period varying from ten to thirty days. In about six weeks after the beginning of the primary lesion, more or less generalized cutaneous manifestations appear. This last-mentioned interval, usually described as the second incubation period, may extend over a period of from four to twelve weeks. During this period there is gradual enlargement of the superficial lymphatic glands.[1] Those nearest the early lesion enlarge first; later others are involved. Chief among these are the inguinal, the epitrochlear, the anterior and posterior cervical, post-auricular, occipital, submaxillary, and axillary groups. Their enlargement varies, but usually they are from pea- to nut-sized, hard, painless, indolent, freely movable, and with no tendency to break down or suppurate. The regional adenopathy in association with extragenital chancre may be marked; the glands, especially the submaxillary, when the primary lesion is on the face, become as large as a small egg. They remain enlarged during the early stages of the disease and finally undergo a fatty metamorphosis, leaving small fusiform enlargements. In the late stages, adenopathy is not important from a diagnostic view-point.

During this period the general condition of the patient may exhibit both subjective and objective phenomena—anemia, and in certain cases even chloroanemia; wandering pains, substernal or about the articulations; a cachectic appearance; a special irritability of the skin and mucous membranes; and occasionally a well-marked febrile process, the so-called syphilitic fever. Careful examination at this time may also reveal changes in the tonsils and thyroid gland. The skin may exhibit icteric symptoms as the result of hepatic disturbances; albumin may temporarily appear in the urine; pains in the head, limbs, and other parts of the body may produce distress even of a severe grade; the joints may become painful and swollen; and the blood examination reveals a certain grade of anemia with leukocytosis;[2] all of which point to the fact that a general intoxication is in more or less rapid evolution. In many patients the major portion of the above described symptoms pass unnoticed or are absent. A moderate elevation of temperature, accompanied by cephalalgia and slight indisposition, not infrequently occurs.

Chancre[3] (*Initial sclerosis, Indurated chancre, Primary sore, Hunterian chancre*).—Acquired syphilis is always initiated in a primary lesion. This is usually single, though multiple lesions are not infrequently seen. The clinical appearance of the chancre varies with its location, age and accidental features added by infection or irritation. It may originate in a scratch, a superficial abrasion, fissure or a herpetic lesion. In form it may be papular, nodular or occur as a plaque of varying dimensions. It may be level with the surface or moderately or markedly elevated. The surface may be scaly, eroded or ulcerated.

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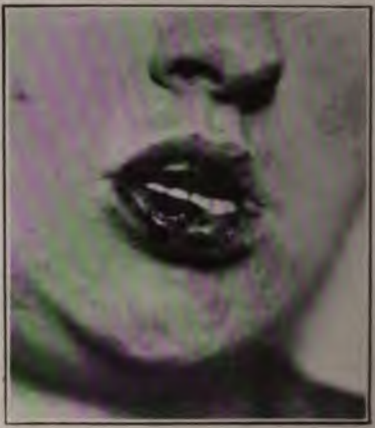
[1] Friedländer, Jour. Cut. Dis., 1912, xxx, p. 14: The value of Lymphatic Gland Examination as a Factor in the Diagnosis of Syphilis.

[2] Hazen, Jour. Cut. Dis., 1913, xxxi, pp. 618-739: The Leukocytes in Syphilis (bibliography).

[3] Klauder, Jour. Amer. Med. Assoc., 1919, lxxii, p. 693; The early diagnosis of syphilis, a correlation of clinical and laboratory methods. A good description of primary lesions.

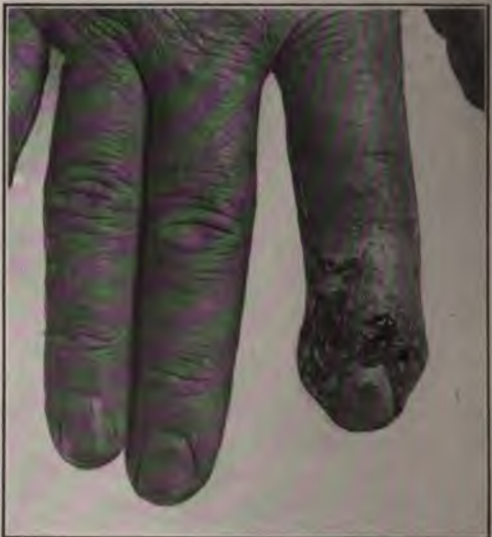
FIG. 279



Chancre of lip with neighboring lymphatic glandular involvement. (Morrow.)

Induration, which is characteristic of most lesions after a short time may be thin and parchment-like or involve the deeper tissues. Durin

FIG. 280

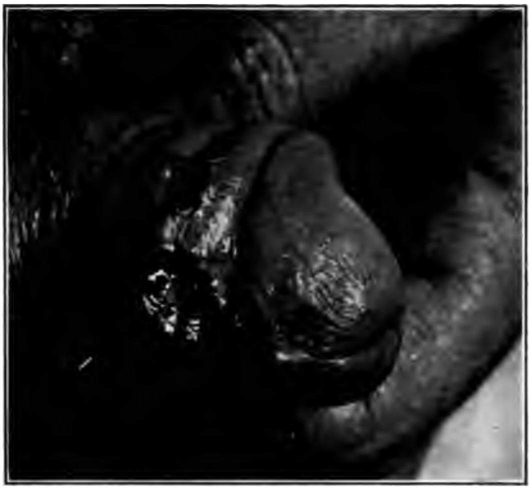


Chancre resembling paronychia.

the first few days when the diagnosis is most important the clinic appearance may be merely a superficial abrasion with little if an sclerosis. A microscopic diagnosis is essential in these early cases. The common type is the chancrous erosion. This begins as a small abrasion which slowly enlarges, becomes oval in contour and is surrounded by a dark-red areola. Its surface, which is at the onset smooth and glossy, later becomes covered with a grayish pellicle and exudes serum, which may be occasionally tinged with blood. Induration, which begins after a few days, increases for some time.

Less common than the above are the ulcerative types. In the superficial form ulceration occurs on a nodule or indurated plaque and produces a shallow ulcer with sloping edges and a floor which exudes serum and blood and which frequently is covered with a membranous pellicle. The deeper ulcers have abrupt edges, are markedly indurated, and the destructive process is more marked. In rare instances all the induration is removed by the ulcerative process. The so-called Hunterian chancre is merely a markedly indurated deep ulcerative lesion. Occasionally the lesion may be a dry, scaling, indurated, dark-red elevation without erosion or ulceration. This type is termed the indurated papule. A type of lesion described by Taylor as the silvery spot occurs on the glans, and at first presents the appearance of a superficial burn with phenol or silver nitrate. This later becomes dark-yellowish and induration develops around and beneath the lesion.

Fig. 281



Multiple penile chancres.

Other types occur and are named according to the clinical form assumed by the lesion. Occasionally a chancre develops in a chancroidal ulcer, in which case the latter becomes indurated instead of remaining a soft lesion and the resulting lesion may assume a variety of forms.

Genital chancres in men are usually situated on the mucous membrane of the prepuce or behind the corona, or on the glans; less often at the orifice of the meatus or they are entirely intra-urethral. Occasionally they are seen on the shaft of the penis, in the pubic, inguinal or perianal regions or on the scrotum.

In women the major portion occur upon the labia majora and minora, the fourchette, about the urinary meatus and upon the cervix.

Relapsing Chancre (*Chancre redux*).—Occasionally after the chancre has disappeared under treatment it recurs within a few weeks, and assumes its original form. This relapse is attributed to insufficient treatment (Lambkin).[1] The term "chancre redux" has also been applied to a lesion simulating chancre which is in reality a superficial, non-ulcerative, sluggish gummatous infiltration.

Extragenital Chancres[2] occur most frequently upon the lips, fingers, female breasts, tongue, tonsils, eyelids, cheeks and various other situations. On the finger the lesion assumes a variety of forms. It may be a dry papule which attracts little attention, a nodular, eroded or ulcerated mass, or its surface may be papillomatous or vegetating. Not uncommonly the lesion begins like a whitlow. In these the area is swollen, red, painful and the surrounding tissue is indurated. The surface becomes irregularly ulcerated and secretes a clear fluid. The nail is frequently destroyed. When untreated these are indolent and chronic. On the lip and other surfaces chancres are often large, deeply ulcerating lesions accompanied by marked induration. Frequently the chief characteristic is an indolent obstinate ulcer.

Diagnosis.—Every genital lesion not clearly placed in some other category should be examined for *Spirocheta pallida*. The chancre often is not characteristic in its earliest stages when its recognition is of paramount importance and the nature of the lesion is demonstrated only on microscopic examination. In well developed cases the important features are the long period of incubation, indolent nature, induration, mild inflammatory symptoms and lack of subjective sensations and the persistence of the lesion. Not infrequently herpes labialis continues unusually long, becomes indurated at its base and develops into a typical chancre. In any indolent lesion on the lip, finger or elsewhere that shows no tendency to heal, a chancre is to be considered. Chancres on the tongue and tonsil present difficulties in their recognition, but the induration and accompanying adenopathy, which are usually marked, together with a microscopic examination, will decide their nature.

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[1] Power and Murphy, A System of Syphilis, 1908, vol. i, p. 180.

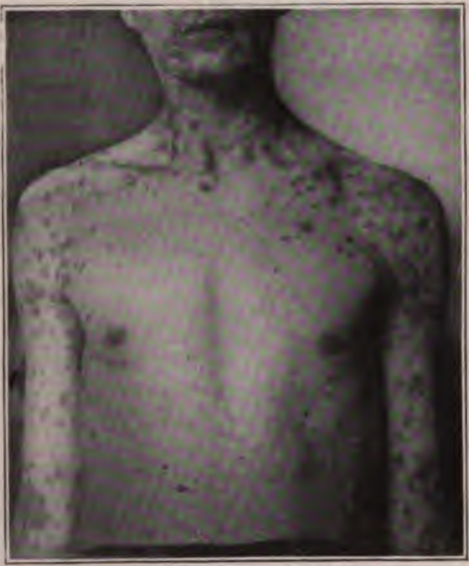
[2] Montgomery, Jour. Cut. Dis., 1905, xxiii, p. 342; The Location of Extragenital Chancres (bibliography). Smith Morton, ibid., 1915, xxxiii, p. 684; Primary Syphilis of the Tonsil. Report of three cases. Literature. Cole, Trans. Sect. Derm., Amer. Med. Assoc., 1916, p. 133; Report of a series of sixty-one extragenital chancres. Zeisler, E. P., Jour. Amer. Med. Assoc., 1917, lxxiii, p. 1546; Multiple chancres. Mitchell, J. H., Surgical Clinics of Chicago, 1918, ii, No. 2, p. 341; The extragenital chancre as a complication in minor surgery. Porter, Archiv. of Derm. and Syph., 1920, n. s. i, p. 15; Statistical study of extragenital chancres. Montgomery and Culver, Jour. Amer. Med. Assoc., 1919, lxxii, p. 180; Chancre of the fingers.

At times the accompanying secondary infection, which is most likely to occur in the chancres on the fingers of surgeons and nurses, obscures the nature of chancre for a time and the latter should always be suspected in such inflammations that persist beyond the usual period of acute infections.

Genital chancres are readily distinguished from herpes progenitalis when fully developed. In the early stages each may be represented by superficial abrasions. The important findings in herpes consist of a group of several lesions, either vesicles or superficial erosions, situated upon an inflammatory base accompanied by burning and smarting sensations. The early chancre is usually single, less inflammatory and unaccompanied by subjective symptoms. In doubtful cases a microscopic examination is essential.

Chancroid begins as an acute infection, is inflammatory, and the ulceration may be deep and irregular, but not indurated, presents a purulent discharge and is often accompanied by inguinal adenitis.

FIG. 282



Small, flat, papular syphiloderma.

Syphilodermata (*Syphilides*).—The skin manifestations of syphilis are of common occurrence, numerous as to their forms, and of great importance from a diagnostic point of view. Lesions of the skin appear in syphilitic individuals of both sexes, at all periods of life, and in all stages of the disease. These symptoms are, however, much more frequent during the first two years after infection, subsequent to which period the manifestations of the disease are betrayed more commonly in subcutaneous lesions, or lesions which affect the viscera and the osseous, nervous, muscular, and vascular systems.

General Characteristics of the Syphilodermata.—Syphilis may be benign or malignant. Ordinarily, it is only moderately severe. The cutaneous manifestations of the malady are protean in character. As early stated by Hutchinson, it is the great imitator of other diseases in its cutaneous manifestations.

Distribution.—The earlier lesions of syphilis are usually symmetrical, superficial, and more or less generalized. The late lesions are asymmetrical, deep seated, grouped, and more or less localized. In some cases there are exceptions of importance to the above rule. The eruptive phenomena of syphilis may be general or few, and either conspicuous and formidable, both as to their extent and persistence, or short lived and insignificant. It is said that as a rule the more general and profuse the earlier eruptions of syphilis, the more favorable the issue with respect to the prognosis.

Color.—The shades of color in syphilitic lesions are usually less brilliant than those seen in other skin diseases. Dull-red or an admixture of red, yellow and brown in varying proportions, frequently with a preponderance of the brown, is characteristic. The early macular eruption (roseola) is of a lighter hue. These color shades have been compared with that of raw ham and with copper hues; these, unfortunately, having been so associated with the syphilodermata that the non-recognition of such tints has led to error in diagnosis.

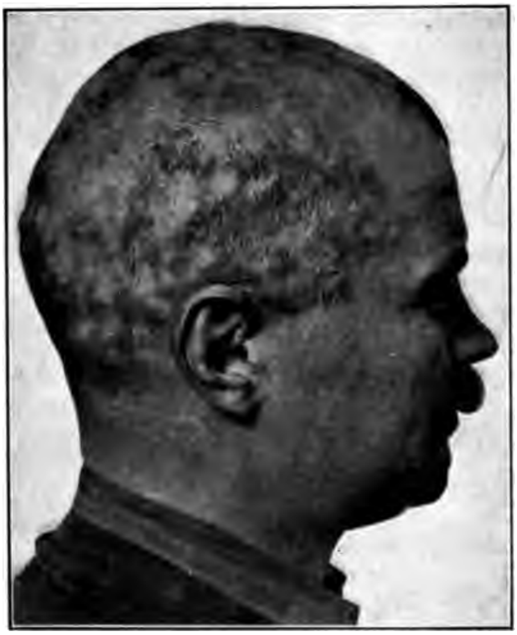
Configuration.—In the early lesions there is no characteristic arrangement except that assumed by annular papules and the deeper pustular syphiloderm. Relapsing early lesions may have a circinate arrangement. The late lesions are characterized in their arrangement by figures resembling a horse-shoe, the kidney, a half-moon, the letter *S*, and a dumb-bell, and it is of common occurrence to find outlines of patches, ulcers, and scars observing the curve of a segment of a circle, the coalescence of several such lesions tending to produce a serpiginous aspect.

Subjective Sensations.—The eruptions produced by syphilis are rarely attended by itching, burning, or painful sensations of any sort. This absence is often a positive aid in establishing the diagnosis, and as a rule is the more valuable the graver the lesion. Great difference, however, will be noted in this respect between different individuals. Occasionally, considerable itching will be induced by condylomata situated in the anal region, and syphilitic ulcers, especially of the leg, may be productive of more or less pain. As a rule, however, even with extensive destructive processes going on, practically no subjective sensations are present.

Career.—The early lesions of syphilis appear rather rapidly and usually attain their development in from one to two weeks, though new lesions continue to appear occasionally for some weeks, particularly the papular variety. After a few months the lesions gradually disappear, an occasional exception being those which occur on the palms and soles, which may be more or less persistent. Syphilis has been described as a chronic disease, and yet its lesions, compared with those of some other diseases, are of short duration. A characteristic feature of the late syphilodermata is their spontaneous involution with development in new areas. In this particular, syphilis is strikingly different from lupus and carcinoma, in which diseases lesions tend to persist in a single location for a long period of time.

Site.—No portion of the skin is free from the possibility of invasion by syphilis. The disease may involve at once almost the entire integument, or it may rapidly spread from point to point, having covered finally a large area; or it may appear conspicuously at distant and isolated points of limited extent; or, finally, it may be manifested in an insignificant lesion or group of lesions ephemeral in course and limited to one part of the body. Different lesions of the syphilodermata have sites of predilection, though lesions may be induced in any area by local irritation. The mouth and throat thus frequently show marked lesions as the result of local irritation from smoking. Predominant sites of syphilodermata are the hairy scalp, particularly at its borders, the vicinity of the mucous outlets, angles of the lips, the eyelids, the anal and genital regions, the palms and soles, the alæ and the root of the nose, the forehead, the interdigital spaces of the feet, the umbilicus, the axillæ, and the groins. Special lesions occur only in certain areas, as, for example, the squamous syphiloderm of the palms and soles, the condylomata and mucous patches in moist

FIG. 283



Patchy syphilitic alopecia.

regions, and the papules of the forehead (constituting the so-called *corona veneris*).

Polymorphism.—Often several varieties of lesions are present, especially during the early stages of the disease. Viewing the cutaneous and other symptoms of syphilis as a whole, this feature is significant, as it is possible to observe at one and the same time upon the body of a single affected individual symptoms indicative of pathological changes in the skin, mucous membranes, hair, nails, lymphatic glands, and periosteum. To a less marked degree this is true of the syphilodermata, several of which may coexist.

Concomitant Symptoms.—Along with the cutaneous manifestations of the early stages other symptoms may occur. The initial sclerosis may still be present or its scar be plainly visible. There may be adenopathy, usually general and characteristic; sore throat, mucous patches, alopecia syphilitica, iritis, and bone-pains. The skin is sallow and dingy, the hair lusterless and lifeless. Onychia and paronychia may occur. Alopecia occurs during the active stages and is exhibited as a general falling of the hair; or it occurs as ill-defined and incomplete patches. The coalescence of these patches gives the scalp a moth-eaten or mangy appearance. Alopecia also occurs in the scars following ulcerating lesions occurring in the scalp late in the disease. Onychia and paronychia usually occur during the early stages.[1] With the late manifestations concomitant symptoms are usually absent, but there may be bone-pains and cephalalgia.

Classification.—The cutaneous manifestations of acquired syphilis have recently been classified by George Henry Fox,[2] as follows:

FORMS.	VARIETIES.	DESCRIPTIVE ADJECTIVES.
	EARLY.	
MACULAR.	Roseolar.	
	Annular.	
	Vitiligoid.	
MACULO-PAPULAR.		
PAPULAR.	Miliary	Disseminate, corymbose, annular.
	Lenticular	Disseminate, corymbose, hypertrophic, confluent, squamous.
	Discoid	Moist, annular, confluent, squamous.
PAPULO-PUSTULAR.		
PUSTULAR.	Acuminate	Crustaceous.
	Obtuse	Crustaceous.
	Ecthymoid	Crustaceous, rupial, ulcerative.
	LATE.	
NODULAR.	Agminate	Confluent, squamous, cicatricial.
	Circinate	Squamous, crustaceous, ulcerative.
	Serpiginous	Crustaceous, ulcerative, cicatricial.
SQUAMOUS.	Diffuse.	
	Circinate.	
GUMMOUS.	Diffuse	Verrucous, crustaceous, rupial, ulcerative.
	Tuberous	Ulcerative, cicatricial.

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[1] *Cf.* section on Diseases of the Nails Associated with Syphilis.

[2] Jour. Cut. Dis., 1913, xxxi, p. 224. (This classification was adopted by the American Dermatological Association at its Thirty-eighth Annual Meeting, held in May, 1914.)

It is usual to classify the cutaneous symptoms of syphilis as early and late. The former are exhibited as macules, papules, and pustules; the latter as nodular, squamous (Fox), and gummatous.

Syphiloderma Maculosum (*Erythematous syphilide, Macular syphilide, Syphilitic roseola*).—This form of macular syphiloderm is the earliest expression of systemic cutaneous syphilis, and is more or less constant in occurrence, differing in this respect from several of the other syphilides. Often it is unnoticed by the patient, whose attention may first be called to it after its recognition by the skilled eye of another. It occurs in kidney-bean- to filbert-sized macules, roundish, oval, or of irregular contour, and varies in color from a light-rosy to a dull-mulberry hue. In some cases these markings of the skin-surface are very indistinct, requiring for their recognition close scrutiny in a clear light, and occasionally even then leaving uncertainty in the mind. With a lens tinted in cobalt-blue they may be recognized at an earlier date than if viewed with the unaided eye. At times they constitute an irregular "marbling" of the surface, of a kind which renders it difficult to define with the eye the individual lesions composing the eruption, while the general visual effect of the exanthem is distinct. The spots are not elevated above the general level of the integument, but may change in type, a papular lesion developing later in the same site.

The eruption usually appears between the sixth and eighth week after the appearance of the initial sclerosis, and when untreated develops for about one week more. It may be gradual or sudden in evolution and persist for a variable period of time, depending upon the severity of the constitutional disorder and the treatment to which the patient is subjected. During the early part of its career, the hue of the lesion is lighter and may be made to disappear under diascopic pressure. Later the lesions become more deeply stained, exudation having occurred, and the color does not disappear under pressure. When involution is in progress, there is a slow disappearance of the eruption, which gradually fades and disappears in the course of from one to three months.

The eruption may be limited to the skin of the abdomen; it may extend sparsely over the chest, the loins, the ano-genital region, and the thighs; or it may develop over the palms, soles, forearms, and legs; and, in exceptional cases, may profusely cover the entire surface of the body. Local irritation, such as that induced by the cuff about the wrist, or pressure of the belt, may increase the number of the lesions in certain situations; and they may be made temporarily more conspicuous by exertion, stimulants, and hot baths, or other causes of cutaneous hyperemia. They disappear leaving either no trace of their former existence, or slight pigmentation, or they may develop into papules. Papules and pustules may be coincident. The lesions may recur at any time up to the end of the first year, and if so they are more localized, the individual macules larger, and they frequently assume an annular configuration.

Fournier,[1] Nielsen,[2] Crocker,[3] and others[4] have described erythematous lesions occurring in circinate arrangement several years after infection. The titles *neuro-syphilide* (*Unna*), *circinate syphilitic erythema*, and *tertiary circinate erythema* have been used to designate this condition, which consists of erythematous lesions occurring as circular patches or narrow rings, with normal or pigmented centers, and segments of circles, which by coalescence form gyrate figures. Their color is reddish or reddish-yellow. They remain of the size of their original development, and present no infiltration, but at times exhibit moderate scaling. The lesions are usually few in number and are situated chiefly on the forearms, the thighs, the nates, and the sacrum. They are resistant to treatment, have a tendency to recur, and, as a rule, are unaccompanied by other symptoms of the disease, though in some cases syphilis of the central nervous system has been concurrent.

Diagnosis.—The recognition of the macular syphiloderm is readily established when concomitant symptoms, such as the primary lesion or its relics, mucous patches, or alopecia are present. From scarlatina, measles, and rötheln it differs in the indolence of the rash; the absence, in most cases, of decided elevation of body-temperature; and the order of its appearance in different portions of the body, as it rarely occurs first upon the face. Urticaria and the rashes induced by the ingestion of copaiba and other medicaments are distinguished by the marked itching of the affected surface and by their very general diffusion over the entire body, a condition less often observed in the syphiloderm. Tinea versicolor, usually limited to the anterior surface of the trunk, is characterized by a fawn-colored to a chocolate-colored tint; by the furfuraceous desquamation, which the patient usually describes as most noticeable after a hot bath; and by the demonstration of the vegetable parasite (*Microsporon furfur*) among the scales scraped from the affected surface. Tinea versicolor is, moreover, of much longer duration than a syphiloderm, and almost never extends to the exposed parts of the body—the face and the hands. Ringworm of the skin of the body is not symmetrical, and is a parasitic disease. Pityriasis rosea occurs in well-defined, saffron tinted, small-egg-sized scaling patches, usually on the trunk only, the long axis of the patch at right angles to the vertical axis of the body. The disease is never accompanied by adenopathy.

Pigmentary Syphiloderm (*Vitiligoid* (*Fox, G. H.*); *Leukoderma syphiliticum, Vitiligo acquisita syphilitica*).—This is a rare form of syphilis, usually occurring on the sides of the neck in women, and first described by Hardy,[5] in 1853. Subsequent descriptions have been made by Fournier,[1] G. H. Fox,[2] Atkinson,[3] Taylor,[4] Maireau,[5] and others.[6] Much difference of opinion has existed concerning the disorder, some viewing it as a hyperpigmentation and others as a depigmentation of the cutaneous surface. According to the observation of different observers, both forms exist.

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[1] Annales, 1896, vii, p. 1141.

[2] Monatshefte, 1896, Bd. xxii, No. 10, p. 500, and No. 11, p. 555.

[3] Page 848.

[4] Fox, Howard, Jour. Cut. Dis., 1917, xxxv, p. 215. The Annular Macular Syphilide or so-called Neurosyphilide. Report of a case. Literature.

[5] Maladies de la Peau, Paris, 1853, p. 154.

The disorder occurs most commonly from the sixth to the twelfth month after infection, but its occurrence may be of later date. It may be the only manifestation of the disorder, or it may occur in connection with other cutaneous symptoms. It appears usually under the age of thirty-five, and generally in women.[7] The lesions are rounded, slightly oval, or irregularly shaped, split-pea- to dime-sized or larger, discrete or confluent macules, either well defined or having irregular margins, and of a *café au lait* color, which does not disappear on pressure. They may be conspicuous or faint, in the latter case being difficult of detection, appearing like patches of dirty skin. The intervening skin seems whiter than normal. The patches may coalesce, forming a delicate network. In another type the discoloration is more or less uniform, and in it there later appear white spots, which gradually increase in size, and which after a time have oval, rounded, or irregular lines of pigmentation between the whitish areas. At this time vitiligo is strongly simulated, the surface of the lesions being smooth and non-elevated, and the condition being unaccompanied by subjective symptoms. The evolution of the lesions is slow, and they may last for a few months to two or three years, being resistant to the usual treatment for syphilis. In a study of the lesions in a patient, Taylor watched the development of a single pigmented spot and concluded that there is a pigmentary lesion *sui generis*. He further stated that in certain cases syphilis causes an abnormal distribution of pigment (Neisser's *leukoderma syphiliticum*), and his conclusions were, first, that syphilis produces pigmented macules without change in the intervening skin; second, that it causes hyperpigmentation, with coexisting vitiligo; and, third, that it also causes an abnormal destruction of the amount of pigment normally present in a given portion of the skin in which there is a preponderance of the vitiliginous appearance coincident with the pigment spots. German authors (quoted by Crocker) regard the disorder as a simple leukoderma of syphilitic origin on the site of a previous roseola. Fox regards it as of syphilitic origin, but not as a direct manifestation of syphilis. Duhring[8] states that in nature it is a simple pigmentary formation, probably differing in no way from chloasma. The lesions are found chiefly on the neck, particularly at the sides, but are also occasionally found on the face (chiefly on the forehead), the chest, flanks, and rarely on the limbs.

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[1] Lecons sur la Syphilis, Paris, 1873, p. 422.

[2] Amer. Jour. Med. Sci., April, 1878.

[3] Chicago Med. Jour. and Exam., October, 1878.

[4] Jour. Cut. Dis., 1885, iii, p. 97.

[5] Thèse de Paris, 1884; abstr. Jour. Cut. Dis., 1885, iii, p. 218.

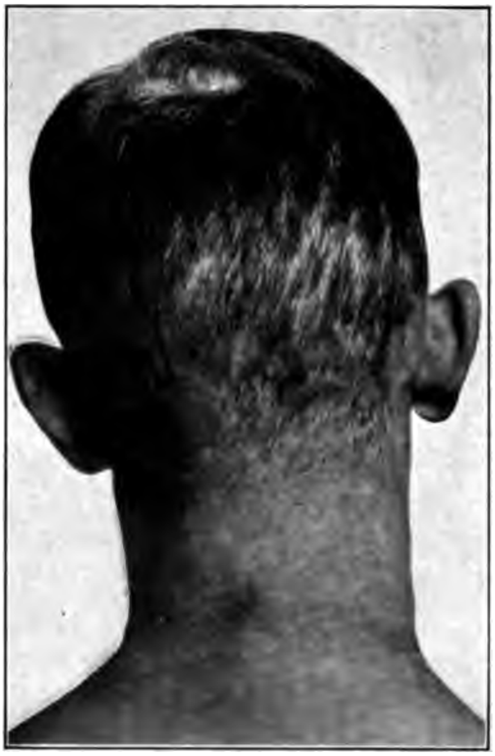
[6] Sutton, Trans. Sect. Derm. Amer. Med. Assoc., 1916, p. 178. The Dyschromias of Syphilis. Histological study.

[7] Little, Brit. Jour. Derm., 1911, xxiii, p. 184: Leukoderma Syphiliticum (case demonstration, male patient).

[8] Diseases of the Skin, 2d ed., 1876, p. 485.

The disorder is to be distinguished from vitiligo, which is asymmetrical, selects as its site the face and the dorsum of the hands and forearm, and the patches of which are of larger size; from chloasma, which occurs in large patches, usually on the forehead and sides of the face, with possibly associated systemic conditions; and from tinea versicolor by the presence in the latter of scaling, the occurrence of the lesions chiefly over the chest and back, and by the microscopic demonstration of *Microsporon furfur*.

FIG. 284



Patchy alopecia with vitiligoid eruption in secondary syphilis.

Hyperemic patches differing from the macular lesions above described were first recorded by Ehrmann.[1] These lesions occur in association with other manifestations of the disease and on various parts of the body. The patches are slightly elevated, of livid-red color, and present a tree-like form, consisting of bands joining a common trunk, or a peculiar network arrangement. In sections there were noted new vessel-formation and an obliterating endarteritis, and *Spirocheta pallida* was found in the vessel-walls.

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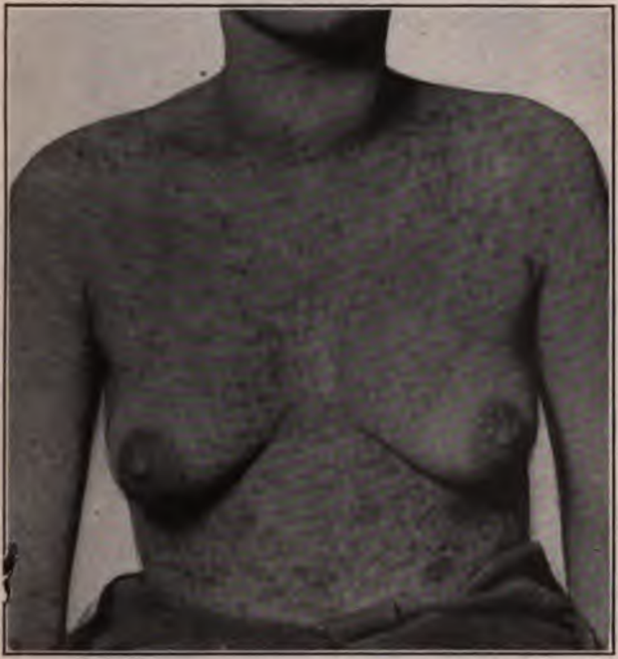
[1] Trans. VI. Internat. Derm. Cong., 1907, ii, p. 763.

Syphiloderma Papulosum.—The type of all cutaneous lesions produced by syphilis is to be recognized in the papule. Papules occurring in syphilis may appear as the first cutaneous evidence of the infection, or they may be developed from earlier macules. They may be small or large, acuminate or flat, disseminated or in groups. They commonly occur during the first six months after infection, but relapsing forms may develop some time later. While frequently generalized, they have sites of predilection, such as the face, neck, flexor surfaces, and ano-genital region.

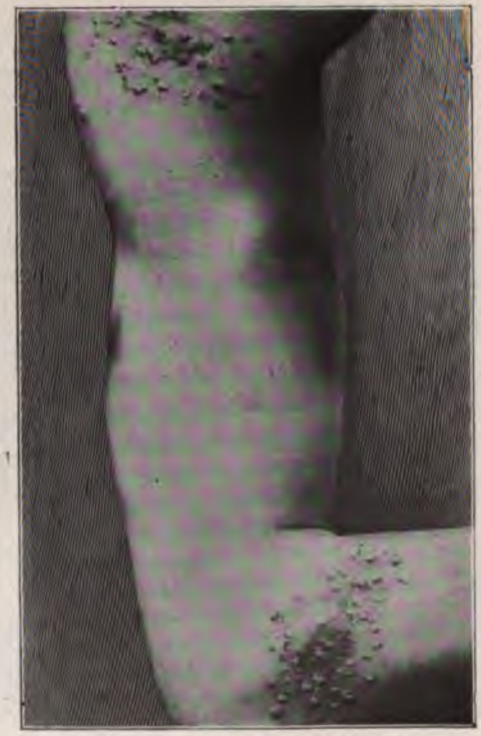
Fig. 285



Papular syphiloderm.

Small Acuminate Miliary Papular Syphiloderm (*Syphilitic lichen, Follicular syphiloderm*).—In this eruption the lesions are millet-seed- to hemp-seed-sized, circumscribed, globular, acuminate, reddish or salmon-reddish, firm elevations of the surface, or minute nodules upon the skin, generally symmetrically developed, often over the entire body, closely set and occasionally grouped in crescentic figures. When viewed with care, a minute vesicle, pustule, or scale may often be detected on the conical apex of each papule; the vesicular or pustular lesions rarely developing, however, to such an extent as to become a characteristic feature of the eruption. The color is at first, especially in blond skins, a mixture of salmon and red; later the darker and browner shades appear. When generalized, the eruption is well developed, especially over the posterior surface of the body, the occipito-cervical and scapular regions, the buttocks, and the calves of the legs, though it is often distinct about the anus and genitalia. Like several other of the syphilodermata, its earlier manifestations are more symmetrical than its later, whether these be tardy or relapsing or both. Involution occurs by resorption of the plastic exudate, minute and usually scanty, dirty-whitish scales encircling the base of each lesion. The eruption occurs during the first six months after infection, and its evolution is complete within two weeks. Where the lesions last for a considerable time, they leave pigmentation and permanent pitting. The eruption as a whole is indolent in undergoing involution, at times persisting for several weeks or months, and though it is quite amenable to vigorous treatment it responds more slowly than several other forms of cutaneous syphilodermata.

FIG. 286



Corymbose papular syphiloderm.

Diagnosis.—The eruption is to be distinguished from keratosis pilaris, the lesions of the latter being less distinctly papular, less vividly colored, and occurring more extensively on the extremities.

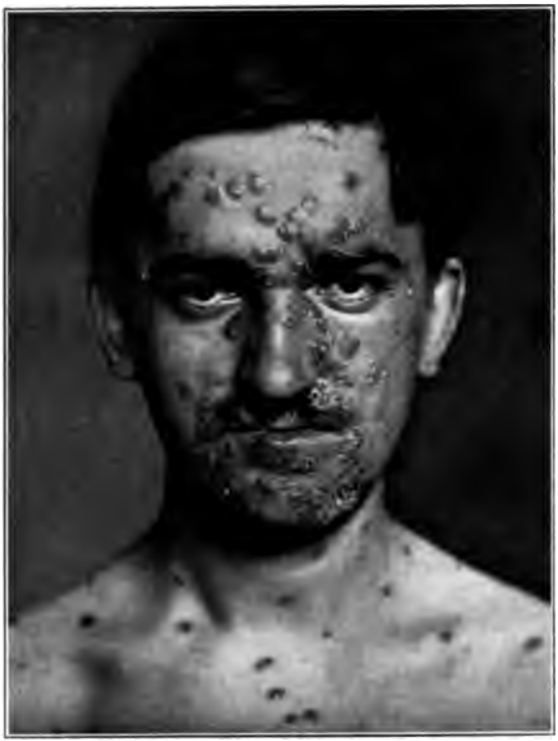
From psoriasis it is distinguished by the distribution of the lesions, which in the latter disorder occur particularly about the extensor surfaces of the joints and in the scalp, while the miliary papular syphiloderm occurs quite commonly on the face, neck, and parts of the trunk not particularly selected by psoriasis. The individual brownish, acuminate, follicular papule of the syphiloderm, often grouped, with moderate scaling, is, in most cases, distinctly different from a patch of psoriasis. Squamous eczema is distinguished by the presence of itching, more marked scaling, less infiltration, a lighter tint of redness, and poorer definition of the patch. From pityriasis rubra pilaris it is distinguished by the absence in the latter of any tendency to vesiculation or pustulation in connection with the individual papules; by more profuse scaling when patches are formed by coalescence of the lesions; and by the frequent association of a palmar hyperkeratosis, a facial seborrheic dermatitis, and a pityriasis of the scalp. Finally, the dark, hyperkeratotic, follicular papules occurring on the dorsum of the fingers are characteristic of pityriasis rubra pilaris. Lichen planus is distinguished by its flat papules, having an irregular or angular base, by their linear or stellate arrangement, by the peculiar violaceous color, and by the constant associated itching sensations. The concomitant symptoms of the disease, if present, are valuable aids in the differentiation of all the above-mentioned disorders.

The Lenticular Papular Syphiloderm. — This often follows the macular form, the lesions being flattened or hemispherical, firm, pea- to finger-nail-sized, circular or oval, and they may be found on all parts of the body. They are seen most commonly on the forehead, face, neck, trunk (chiefly over the back), the flexor surfaces of the limbs, and about the ano-genital regions. They may be smooth and glossy, or scale covered, and are often surrounded at their base by a fringe of epidermis. Their color varies from a light-red early to a yellowish- or brownish-red later. Below the knees they may be purplish. On the face they select the hair-line on the forehead. On the scalp they may form into papulo-pustules. They may be disseminated or grouped; in the latter case frequently assuming the *corymbose type.* The latter lesion is often a relapsing one, and is produced by a large papule being surrounded by a group of smaller ones. Corymbose lesions may be irregularly disseminated among papules of other types, or occur independently.

On the face, and occasionally in other situations, the lenticular papules may become of large size, when they are termed *hypertrophic.* When of small size and flattened, they have been termed *small, flat, papular syphiloderm.* These lesions develop slowly, last one or two months, then disappear, leaving temporary pigmentation or depressed spots. Pustules may develop upon them, producing a *papulo-pustular syphilide.*

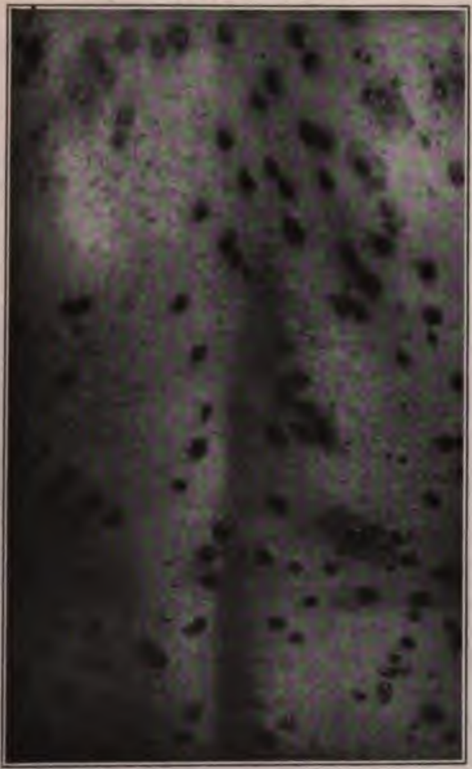
Discoid Papular Syphiloderm (*Large, flat, papular syphiloderm*).— These lesions are circular or oval in shape, and in size vary from that of a finger-nail to that of a pigeon's egg, the major portion being usually nummular. The lesions occur as well-defined, firm papules, with a raised border and a shallow depression in the center, though at times, especially in moist situations, the top of each plaque is a smooth, flat plane. They present the usual variations in color, and may scale at the edge or over the top. They seldom have a generalized distribution, but are limited to special regions. On the face they may form rings by involution of their centers. On the trunk or other parts of the cutaneous surface they may form patches by coalescence of different lesions. The patches when scale covered resemble psoriasis (*papulo-squamous syphiloderm*). They may occur along with lenticular papules or independently, usually, however, following the latter, and not infrequently as a relapsing eruption. The *annular papular syphiloderm*, which occurs most frequently in the colored race, and especially on the face, presents a well-defined, raised margin, which may be smooth or be made up of individual smaller papules, and may be formed either by the clearing center of a discoid patch or independently. In rare instances concentric rings occur, and by coalescence of several of these a gyrate configuration is produced.

FIG. 287



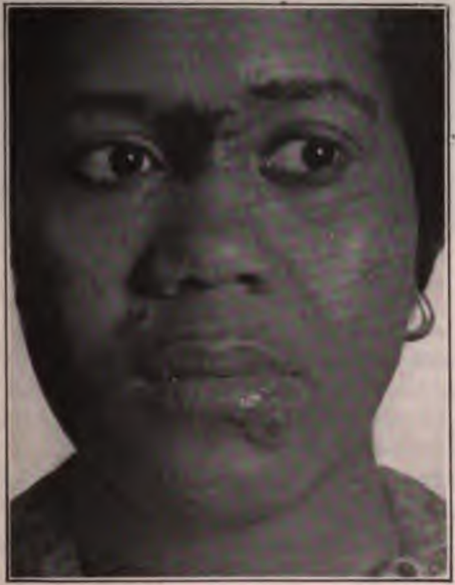
Lenticular papular syphiloderm.

FIG. 288



Lenticular papular syphiloderm.

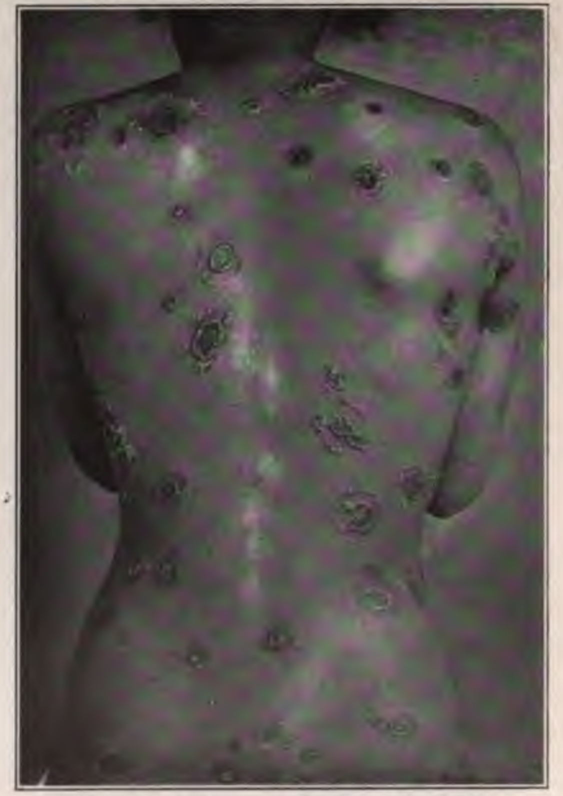
FIG. 289



Large flat papules in a negro.

In a study of annular lesions of syphilis occurring in the negro, Hazen[1] noted their development from both large and small flat papules, including the scaling (papulo-squamous) variety; from follicular papules, from papulo-pustules, and from maculo-papules; also in two cases of hereditary syphilis. Their evolution occurred both by peripheral extension, with clearing of the center, and by the coalescence of individual lesions in a ring formation.[2]

Fig. 290



Annular papular syphiloderm. (Fordyce.)

The *papulo-squamous syphiloderm* bears, in many instances, a close resemblance to psoriasis, but it may usually be readily distinguished from the latter by a consideration of the following points: The syphilide, as a rule, is not generally distributed, though such a distribution occasionally occurs, and the lesions are apt to be less symmetrically disposed than the patches of psoriasis. The individual papules present greater infiltration; are dark-red or brownish-red in color; and are covered with more or less adherent, grayish or darker colored scales. Frequently, the papule is surrounded by a collarette of scales, while the scales of psoriasis spread in an imbricated manner, covering the entire lesion and extending beyond the border. Interspersed among the scaling patches of the syphiloderm may be found individual brownish-red papules characteristic of syphilis, and possibly other lesions presenting a multiform eruption. The lesions are more apt to be found on the flexor surfaces, about the face, and in other situations not commonly selected by psoriasis. In the latter disease the color of the papules is reddish; the scales are white, shining, imbricated, abundant, loosely attached, and cover the entire lesion, as above stated. They elect the extensor surfaces and are found abundantly in the scalp, and uncommonly on the face and palms, areas frequently attacked by the syphilide. Subjective symptoms are absent in the latter disease but may be present in the former. In the presence of the concomitant symptoms of syphilis, exhibited as mucous patches and moist lesions about the ano-genital region, with some constitutional disturbance, the diagnosis is made without difficulty.

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[1] Jour. Cut. Dis., 1913, xxxi, p. 148.

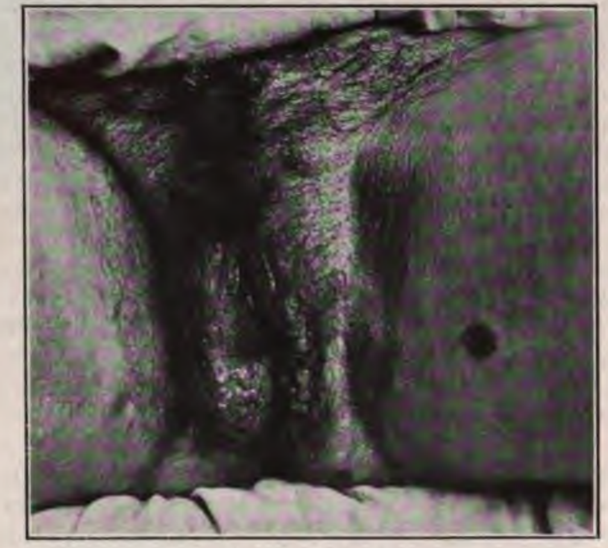
[2] *Cf.* Atkinson, Jour. Cut. Dis., 1883, i, p. 15; Fox, H., ibid., 1908, xxvi, pp. 67–109, idem, Archiv, 1912, cxiii, p. 315; Gilchrist, Maryland Med. Jour., 1900, xliii, p. 200.

Moist Papules (*Mucous patches, Condylomata.* Fr., *Plaques muqueuses;* Ger., *Schleimhautpapeln, Feigwarze*).—Moist papules occur on portions of the body where two surfaces come in apposition, and present characteristics differing from those in other situations, due to heat, moisture, and friction. Their common sites are the genital region, about the anus, vulva, scrotum, and thighs; beneath the mammary glands in women; in the axilla and naso-labial folds; at the angles of the mouth; and between the fingers and toes. They may be well defined or have an indefinite contour; may be soft, spongy, or firm; and present a grayish or brownish, secreting surface. Beginning as flat papules, they may by maceration become more flattened and indefinite in outline, being covered with a thick mucoid secretion; or, as is often the case, especially in the genital region, become hypertrophied, distinctly elevated, well outlined, and present the characteristics described as condylomatous. When the secretion is removed, the surface of the lesion is seen to be pinkish or light- or dark-red in color. About the vulva, over the perineum, and about the anus large plaques may form by coalescence of individual lesions, which may become warty, papillomatous, or vegetating. The mucoid or mucopurulent secretion, which may be abundant in these regions, is characterized by an intensely offensive odor.

The secretion from all these lesions is highly infectious, and while, by autoinoculation, lesions of a similar type are induced, in new soil they produce the usual initial lesion of syphilis and are responsible for the major portion of new infections.

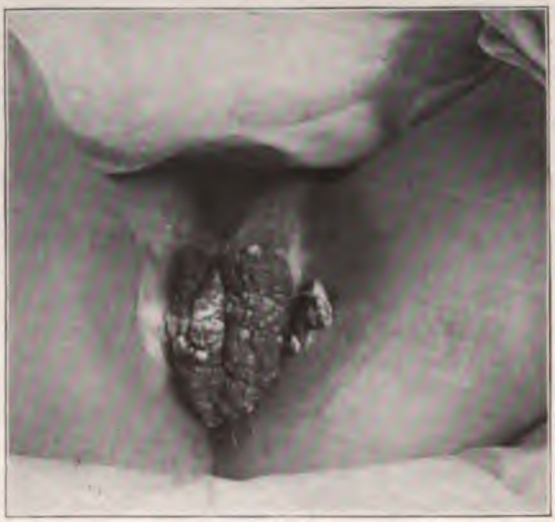
Mucous patches are merely syphilitic papules occurring in moist situations, flattened by reason of the apposition of affected surfaces and by contacts necessitated by the functions of the parts involved. They form upon all mucous surfaces, but especially in the mouth, where they are the most annoying and the most persistent symptoms of syphilis, complicating both the early and the later stages of the disease.

Fig. 291



Condyloma latum.

Fig. 292



Vegetating condylomata of the vulva and anus.

The patches are roundish or oval, tumid, flattened or very slightly depressed, pale-rosy or whitish spots, moistened with mucus, and either developing as such or resulting from hyperemic plaques, or dispersed among or upon the latter. They often resemble the patches produced on the mucous membrane by penciling the latter with a crayon of silver nitrate. When carefully inspected, many of them exhibit a loosened and partially detached film of membrane covering the tissue, beneath which a reddish, raw-looking surface appears. One or several may be present, and they occur most commonly during the active period of the disease. They are most often seen inside of the lips, near the angles of the mouth; also on the buccal mucous membrane, the fauces, the tonsils, and on the tongue. On the dorsum of the latter, reddish, smooth areas may be noted; while on its under surface lesions simulating condylomata may occur, presenting warty and vegetating growths. At the angles of the mouth some thickening, with consequent fissuring, not infrequently occurs. Erosions may be present, making the ingestion of hot liquids and acids difficult and painful. Superficial ulcers, covered with grayish-white exudation and surrounded by a red areola, occur; while deeply situated and destructive ulceration may attack the tonsils.

Opaline plaques occur as flattish, smooth, bluish-white or lead-white, firm, slightly indurated, and roundish or highly irregular areas. They are relatively painless when not the seat of ulceration. They are visible on the dorsum of the tongue, on the mucous lining of the cheeks, and at the angles of the mouth, in which latter site they are situated often in part on the mucous surface and in part on the skin of the lip. The thickened epidermis is at times covered with adherent, not readily removed scales, between which fissures form; and the patch, at first almost insensitive, becomes exceedingly tender and painful.

These patches are for the most part leukoplasic, due chiefly to irritation of the mucous surfaces by tobacco smoke, yet recurring in syphilitic subjects; and they are preceded often by typical mucous patches. They are almost exclusively seen in men.

In addition to the above described conditions, the mucous membranes are affected, both early and late in the disease, with lesions resembling those occurring in the skin, the differences between the two types being those induced by heat and moisture. Usually at the moment of the outbreak of general syphilis or soon after, a pharyngeal or pharyngo-nasal blush may be seen extending irregularly or symmetrically over the parts, accompanied often by engorgement of the tonsils. There are then associated pain and swelling, and complications may arise producing laryngeal hoarseness, cough, dyspnea, aphonia, and nasal discharges, with a moderate amount of obstruction of the nares. In the later stages gummatous infiltrations of the mucous membranes occur in both circumscribed and diffuse forms, superficial and deep. In the diffuse superficial forms, both the mucous and the subcutaneous tissues are involved in a firm thickening, best studied on the surface of the tongue, which then appears polished and smooth, at times as if covered with a thin, transparent varnish. Patients exhibiting this condition will often describe a subjective sensation of "slipperiness." These thickenings may involve the deeper structures by every gradation, producing eventually lobulated masses with intervening fissures, tender, raw, and excoriated. The surface of the tongue is then, as a rule, covered with a foul, dirty-grayish coat, and it is occasionally notched at the edge with deep ulcers. At times the tongue is mottled, with patches of redness alternating with the yellowish-white of the deposit on the surface of the membrane, and more rarely the tip is covered with florid, verrucous, filiform growths.

The deeper gummata involve the body of the tongue, and they are felt as submucous, diffuse or circumscribed, dense thickenings (usually tolerably well defined), which soften, ulcerate, and leave exposed to view extensive losses of substance. The floors of these excoriations are deep ulcers, indurated, sloughing, and with membranous shreds over the surface. The fissures of the sides of the tongue described above may here also produce deeply ulcerated notches in the substance of this organ. Deformities of this class are relieved markedly after cicatrization, even when considerable loss of tissue has resulted.[1]

Diagnosis.—Mucous lesions are to be distinguished with care from simple aphthous patches in the mouth the result of indigestion or local disturbances; also from smokers' patches (*leukoplakia buccalis, psoriasis linguæ, leukoplasie*), and from lichen planus of the mouth. In external features these patches may resemble one another, but in only one affection (syphilis) are there other signs of infectious disease. The chief points of difference are: singleness, for the most part, of aphthous sores, and often exquisite tenderness; multiplicity, as a rule, of mucous patches, and much less soreness, though when ulcerated the soreness may be a conspicuous feature. Linear streaks and bands (often quite insensitive) of leukoplasic patches are found, especially along the gums, on the lines of the inner cheek (representing contact with the approximated upper and lower teeth), and in the pocket posterior to the wisdom tooth. The flattened and often isolated patches of lichen planus of the tongue have an almost characteristic lead-white color.

Condylomata are to be differentiated from verruca acuminata. In the absence of the concomitant symptoms of syphilis, some difficulty may arise. The narrow base and pointed top of verruca acuminata, even when the lesions are abundantly developed and massed together, immediately distinguish it from the condyloma, whose base is broad and infiltrated, and surface flat, verrucous, or papillomatous.

Palmar and Plantar Syphiloderm (*Squamous syphiloderm*).—In consequence of the thickness of the epidermis of the palms and soles, the papular or papulo-squamous syphiloderm of these regions is presented under somewhat atypical forms. The eruption in these situations may occur in symmetrical distribution, early in the disease, as a part of a generalized efflorescence; or somewhat later as a relapsing manifestation, in which case the lesions are apt to be more localized, asymmetrical, and persistent; and finally, many years after infection, as a late manifestation, when it is exhibited in typical development. Here, as in the relapsing eruptions, the lesions may be asymmetrical and more or less localized, though the entire palm or sole may be involved, and even in symmetrical arrangement.

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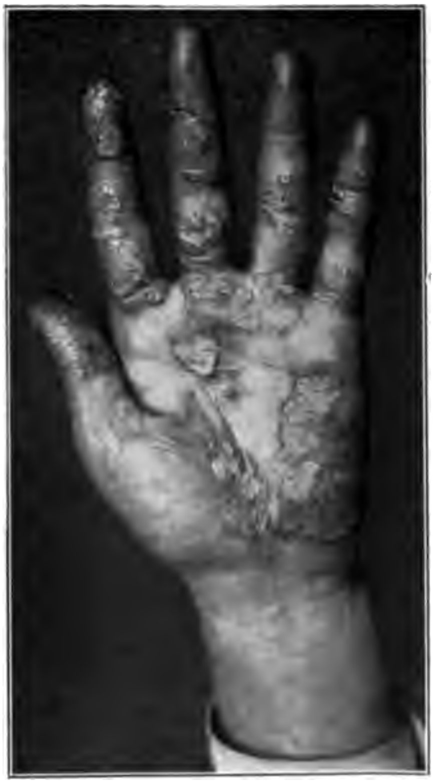
[1] Culver, Jour. Cut. Dis., 1919, xxxvii, p. 48; Syphilis of the Tongue. Report of nine cases.

Papules and nodules which occur on other portions of the skin are prevented from forming on the palms and soles owing to the peculiar anatomical structure found here. The pathological manifestations are consequently displayed rather as thickenings, separations, frayings, and stainings of the epidermis. Here, therefore, are seen dull-red macules, covered throughout or merely at the edges by scales or epidermal shreds; and minute, firm, corneous thickenings, few or many, often without color in consequence of the depth of the bloodvessels beneath the opaque, horny layer. Occasionally, with a pointed instrument, pinhead-sized and larger deposits resembling chalk may be pried from circular beds in the palms and soles where the lesions first develop (*syphilide cornée* of the French). These and similar spots frequently are covered with dirty-whitish, often tenacious, half-loosened epidermic flakes, which are characteristic. In other cases, usually in consequence of the motion of the hand or foot or of the exigencies of toil, irregular, angular losses of epidermis occur resembling the fracture of a pane of glass. The attached portions of the epidermis project at the edges only over deep fissures, broad exulcerations, or a dull-red, tender, and newly-formed epidermic layer. At times, distinctly elevated and circumscribed papules or nodules of the usual livid-red color, varying in size up to that of a coffee-bean or slightly larger, are seen, which may be aggregated in patches having a tendency to assume a circinate outline. The eruption is frequently symmetrical in the center of both the palms and soles, but is rarely found upon the dorsum of the hands and feet, and then never developed

FIG. 293

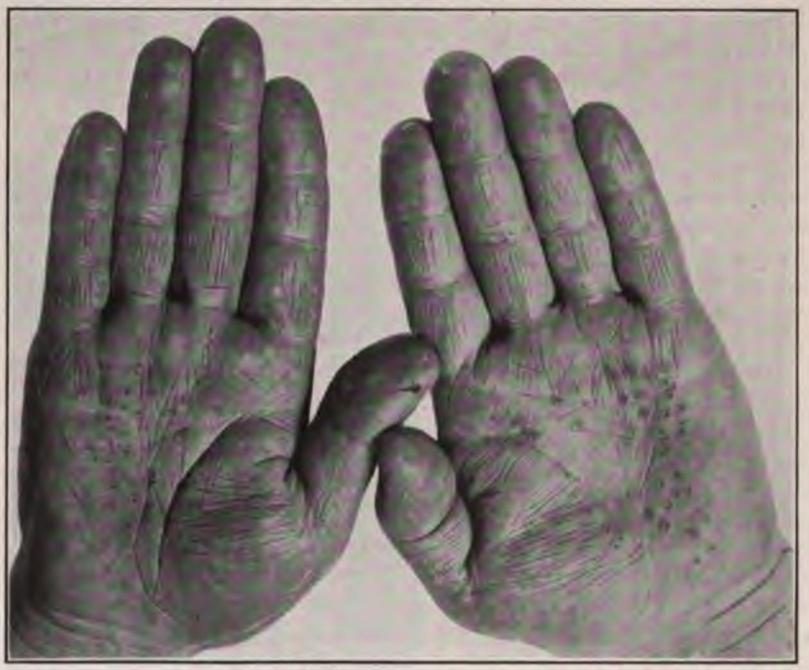


Squamous syphiloderm, late eruption.

typically, but always by extension from the palmar or plantar regions. It may extend to the lateral surfaces of the hands, feet, fingers, and toes, as well as over the wrists and ankles. Occasionally, the entire surface of one palm may be covered with papulo-squamous lesions, which by coalescence have produced diffuse involvement, being, however, well defined at the margins from the normal integument.

A well marked keratoderma[1] occasionally occurs in late syphilis, usually involving one foot. The plantar surface becomes thickened, hyperkeratotic and at times verrucous. Deep fissures may result, particularly about the heel. In some instances verrucous patches develop over the foot and ankle. Examples of involvement of both feet are recorded by Zauche and Flurin, Baker and others. Sequeira describes a pseudo-elephantiasic condition of the leg associated with keratoderma.

FIG. 294



Punctate keratoses on palms in late syphilis. There were similar lesions on the plantar surface of the feet. Lesions disappeared after one injection of arsphenamin.

Diagnosis.—The eruption in these situations is to be distinguished chiefly from psoriasis and eczema. The former disease, with rare exceptions, is not limited to this region, and only occurs here coincidently with lesions distributed in other situations. When occurring here, the scaling in psoriasis is more profuse and the scales are more readily exfoliated, leaving a reddened, tender epidermis beneath. Eczema involving the palms and soles usually invades also the dorsal surfaces of the hands and feet. On the palms a more uniform involvement is apt to occur, or the lesions will be distributed about the margins, not selecting particularly the center of the palm. Extension to the fingers, also, is more frequent, and the edematous infiltration of the tissue produces characteristics differing from the cellular induration occurring in the syphilide. In eczema the lesions are apt to be disposed in particular areas irritated by articles used in the occupation, the latter being operative to a less extent than in the case of the syphilide. In either case, associated lesions, or a history of their having been present, are important aids in diagnosis. In all cases confirmatory evidence may be obtained with the Wassermann reaction.

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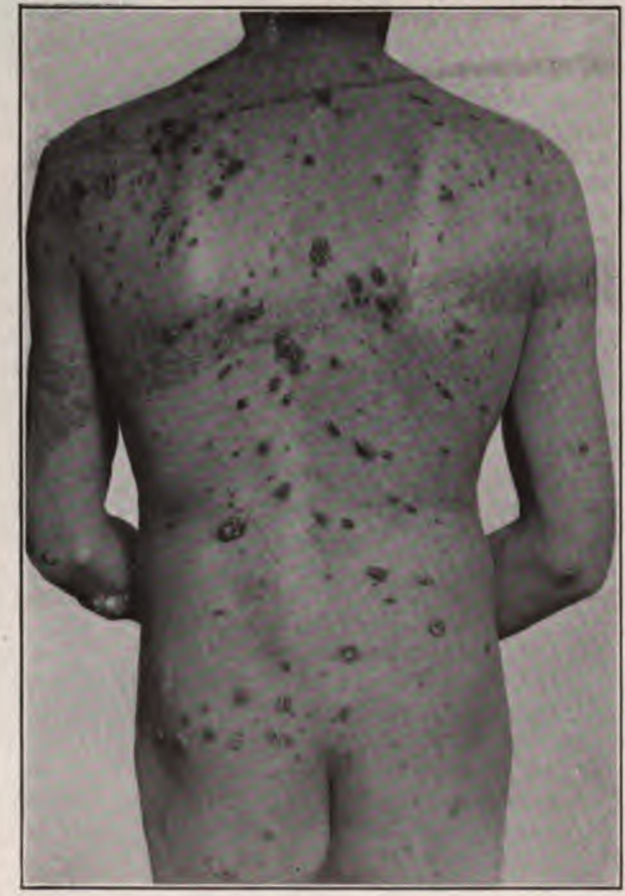
[1] Zauche and Flurin, Bull. d. la soc. franc de Derm. et de Syph., 1909, xx, p. 394; Keratose syphilitique plantaire. Morris, Diseases of the Skin, 1911, 5th Ed., p. 487. Sequeira, Ibid., p. 263. Ealer, Jour. Cut. Dis., 1915, xxxiii, p. 517: Tertiary Luetic Keratodermia. Baker, Ibid., 1918, xxxvi, p. 220: A case of Syphilitic Keratodermia.

Syphiloderma Vesiculosum (*Varicelliform syphilide*).—Vesicular syphiloderma are rare cutaneous symptoms of syphilis, if they do actually occur. Pinhead- to pea-sized, conical, globoid or umbilicated, isolated or grouped, crusting elevations of the epidermis, with lucid or cloudy contents, have been observed upon the face, the trunk, and the genitalia of syphilitic subjects. The eruption is described as an early syphiloderm, often exhibiting a halo of characteristic tint, the resulting crusts being granular and somewhat lighter in color than those commonly seen in the disease. Both small and large vesicles have thus been assigned to the disease, and these, according to their resemblance to non-specific exanthemata, have been described as varicelloid and herpetic. It is thought that the larger number of these lesions are either immature pustules, exanthematous lesions in syphilitic subjects, or pure accidents of the syphilitic process. With regard to the first theory, it may be said that the pustular syphiloderm not rarely begins as a vesicular lesion; with regard to the second that coincidence of so common a disease as syphilis with other cutaneous disorders is a matter of frequent observation; and with regard to the third, bearing in mind the large quantities of potassium iodid taken for the relief of the disease and its capability of exciting a vesicular eruption, it can reasonably be concluded that some at least of the cases of so-called vesicular syphilis have been imperfectly studied. That a true syphilitic lesion represented by vesicles does occur, there is no doubt, but it is unusually rare.

Syphiloderma Pustulosum.—These lesions occur usually in the cachectic and the ill nourished. They vary in size from that of a pin's-head to a finger-nail or larger; may be acuminate, flat, or hemispherical in shape; few or many in number; grouped or disseminated; and they may develop from papules or begin as vesico-pustules. They may be surrounded by inflammatory areolas, may spring from an unaltered integument, or be subepidermic in situation and scarcely project beyond the surface. They are usually covered with reddish-brown to greenish-black crusts, which develop in strata or laminæ by accretions from below, and even when superimposed upon a moist and secreting ulcer they are adherent at the edges. Seated upon superficial or deep, sharply defined, secretory ulcers, they may occur early or late in the disease, and at either epoch may constitute trifling or grave cutaneous lesions. They have a marked predisposition for involvement of the sebaceous and pilary follicles, and they are frequently disposed about the mucous outlets of the body.

Fig. 295



Pustular syphiloderm.

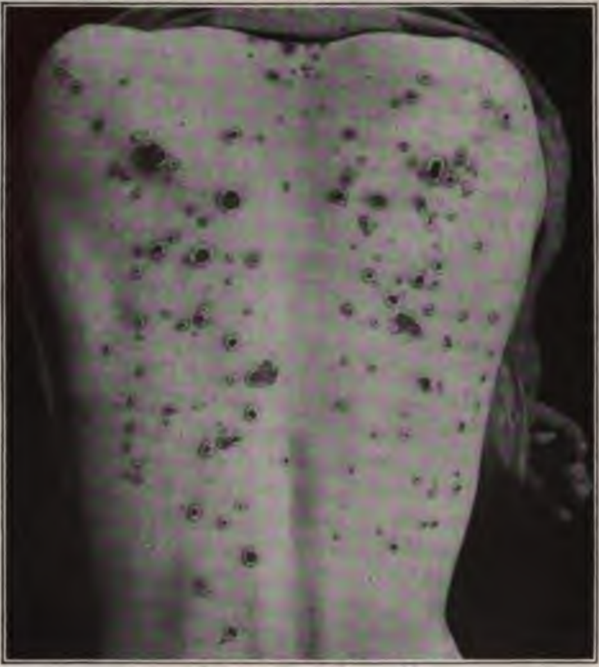
Small Acuminate Pustular Syphiloderm (*Miliary pustular syphilide*).—This exanthem usually occurs in the early months after infection, but may appear later. The eruption is usually diffuse, and the acuminate pustules are located about the hair-follicles. The condition is rare in patients of the better class, and may represent, as Jullien has suggested, a transformation from papular lesions due to pus-infections of skins that are usually unclean, irritated, or the seat of diminished vitality. The pustules are acuminate, and each contains but a droplet of cloudy serum or pus, the desiccation of which furnishes a thin yellowish, or reddish-brown crust. The fall of the latter exposes the grayish epidermal fringe of the base occasionally seen in papules of similar size. The lesions occasion little or no subjective sensation. They may be discrete, confluent, disseminated, or in groups affecting the curve of a circle. The extremities and the trunk are chiefly involved, though the disease may be well-nigh universal in distribution. Upon involution of the lesions, minute punctiform and pigmented, cicatricial, atrophic depressions occur, which later disappear. Relapses occur when treatment has been irregularly pursued. Miliary papules are often interspersed among the pustules. The eruption, aside from uncleanly conditions, is seen chiefly in the so-called "ignored" cases.

Large Acuminate Pustular Syphiloderm (*Acneform, varioliform, obtuse syphiloderm*).—This exanthem of syphilis, sometimes generalized, occurs within the first eight months after infection, and is usually the result of a cachectic condition of the patient or unhygienic care of the skin. The eruption consists of small- or large-pea-sized, grouped or disseminated, acuminate or well-rounded, fairly well distended pustules, which may be seated at the orifices of the pilo-sebaceous ducts. The lesions may begin as reddish macules, pustules, or papulopustules, and may have a tinted border of a coppery hue. They have a thin roof-wall, and while usually acuminate they may flatten, after full evolution, in consequence of partial collapse. The fact that the lesions sometimes suggest umbilication has led writers to use the term "varioliform" in their description, an unfortunate term, which tends to introduce confusion in the description of strictly syphilitic lesions. The eruption may be scanty or profuse; be rapid or slow in evolution; may develop in crops; and may be coincident with the so-called syphilitic fever. When desiccating, the pustules furnish a dirty-brownish, occasionally blackish, crust, covering ulcers of varying depths. The scars left may be persistent, but usually lose many of their distinctive features in the lapse of time.

The eruption, aside from the covered skins of the uncleanly, is seen also on the face, about the alæ of the nose, and about the mouth in subjects of the disease who are anemic, cachectic, or who have been long given to dissipation.

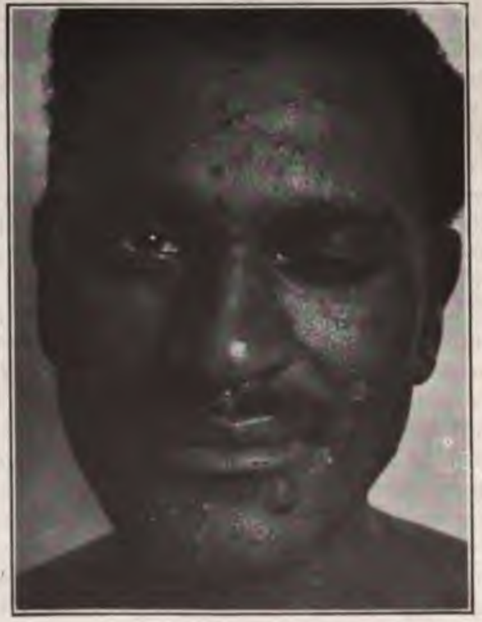
Diagnosis.—The diagnosis between this eruption and variola is established readily, in view of the rapid changes occurring in the last-named disease, the febrile phenomena, the order of appearance of the variolous exanthem, and the evidence furnished by other non-cutaneous symptoms of syphilis which are usually present. The drug exanthemata usually are characterized by more pronounced subjective sensations; the several forms of impetigo are seen very rarely elsewhere than on the face and hands; and acne, limited chiefly to the face and shoulders, never furnishes a distinct ulcer beneath its crusts, and is accompanied over the scalp and elsewhere by characteristic comedones and other stigmata of a sebaceous-gland disorder.

Fig. 296



Large, flat, pustular syphiloderm.

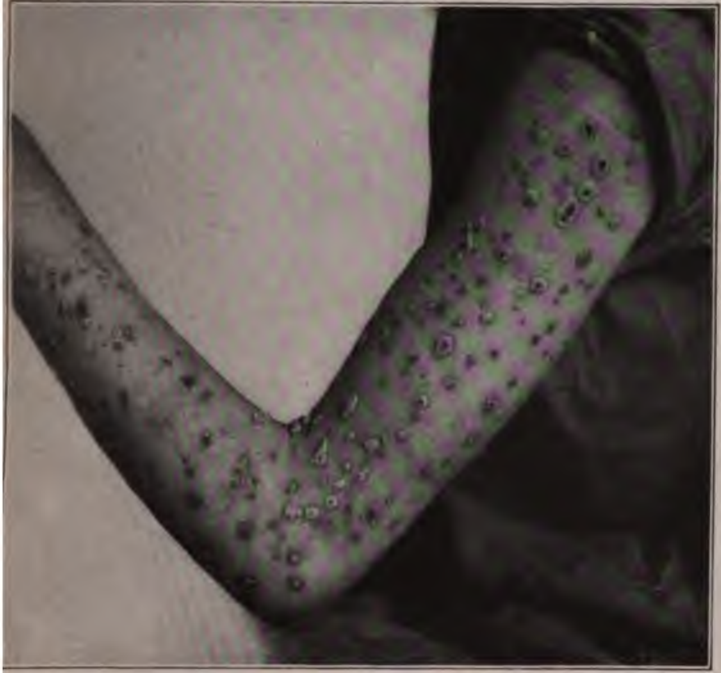
Fig. 297



Aceneform lesions and keratitis in secondary lues.

lat Pustular Syphiloderm (*Impetigoform and ecthymaform syphilo- a*).—This is a relatively frequent manifestation of syphilis oc- ing upon the face, the scalp, the trunk, the flexor surfaces of the emities, and in the genital region, usually within the first year r infection. The exanthem exhibits a decided tendency to char- ristic and circular grouping about the mucous outlets of the body. h groups are composed of small or large, flat, discrete pustules, head- to pea-sized or larger, originating as reddish macular lesions, ch tend to dry into flattish, irregular, dirty-yellowish or brownish, erent crusts. These crusts either exceed the limits of the diseased

FIG. 298



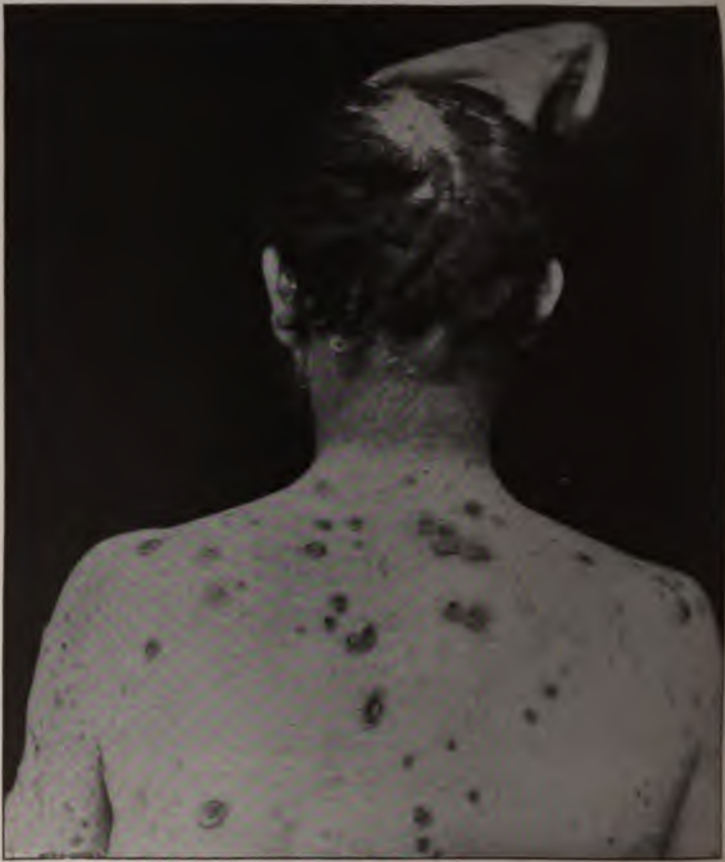
Large, flat, pustular syphiloderm.

ce beneath, or are conspicuous upon a dull brownish-red area flamed, eroded, and at times even ulcerated aspect. Often the ules are so closely set as to become confluent, in which case a e convex crust like a carapace will completely cover the involved . The eruption is of pustulo-crustaceous type, and is usually nable to judicious treatment.

ne larger lesions of this type represent simply the same features n exaggerated extent. In these there occur superficial ulcerations red with flattish crusts, which are blood-mixed, and reddish- and nish-brown in color. The superficial variety of this syphiloderm stinguished from the deep chiefly by the extent of its ulcer, the size of its superimposed crust, and the lighter, dull-red areola which encircles it.

Pustulo-ulcerative Syphiloderm.—The deep variety, like the superficial, may be limited to the scalp, face, neck, and flexor aspects of the extremities, or it may be diffused much more widely. The entire surface of the body may be covered with discrete lesions of this type in cases of unusual neglect or of profound cachexia. The eruption is usually of late occurrence, but in the so-called "galloping syphilis" of the French it may be precocious in development. The lesions are purulent and have a deep, infiltrated base, with a dark-brown halo. Incrustation follows, with the formation of a conical, roundish or oval, blackish-brown crust, beneath which lies a clean-cut ulcer, the sharp edges of which are usually exactly covered by the incrustation. The crust thickens by concretions from the ulcer beneath and spreads

Fig. 299



Large pustulo-crustaceous syphiloderm of the scalp and body.

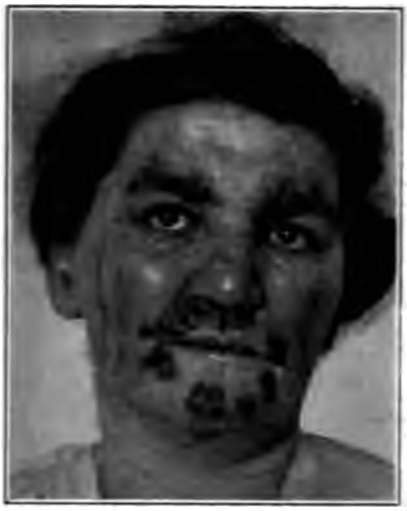
at the periphery, while it thickens in the center. In this way the appearance of the stratified crust resembles that of an oyster-shell, a condition described by some authors as rupia, a term often employed as the name of a disease. The ulcer exposed after removal of the crust is of characteristic syphilitic type in its deep, sharp, punched-out, clean-cut edges, and purulent secretion commingled with blood. At times it attains a diameter of several inches and has a circular contour. The degree of destruction it may produce is inversely proportioned to the constitutional vigor of the subject and the treatment pursued. It is usually a grave and may be a malignant exanthem, though under favorable circumstances it is amenable to judicious treatment. It may be an early, though often it is a late symptom of the disease. The pigmented scars left are characteristic and indelible.

Diagnosis.—Impetigo is readily distinguished from the pustular syphilodermata by its more superficial character, the crusts presenting a "stuck on" appearance, the frequent presence of vesicles, and the absence of other signs of syphilis. Ecthyma at times presents some difficulty. Here the lesions are found largely below the knees, are irregularly placed, and while a certain degree of cachexia is usually present, other signs of syphilis are absent. In doubtful cases a Wassermann test should be employed.

Syphiloderma Bullosum (*Pemphigus syphiliticus*).—Bullæ in acquired syphilis are also relatively rare lesions. They are rarely numerous, pea- to large-nut-sized elevations of the epidermis, filled at first with a cloudy serum which soon is transformed into pus, often commingled with blood. They have usually a characteristic halo about the periphery, are roundish or oval in contour, are usually discrete, rarely disseminated, and after development they produce characteristic crusts with underlying ulcers, identical in feature with the rupioid sequels of large syphilitic pustules. The eruption is localized by preference upon the extremities, more particularly the lower extremities, and is indolent in its course. It is always significant of the cachectic condition in the subject of the disease, and occurs often in the victims of chronic alcoholism who have been infected with syphilis. It is to be distinguished from pemphigus vulgaris by its characteristic crusts and ulcers, considered in connection with the history and associated symptoms of syphilis.

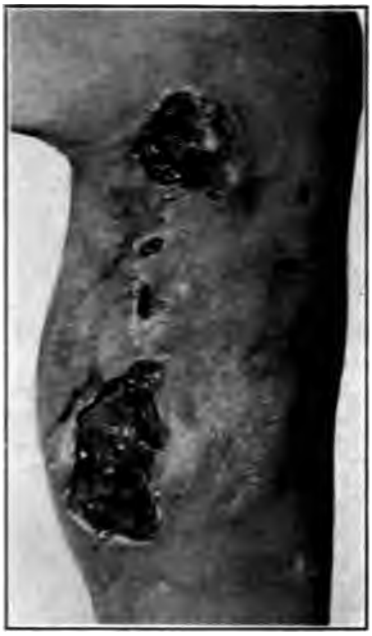
Vesicular Syphiloderm.—In this eruption [illegible] within the first year after infection [illegible] longer the lesions are multiple [illegible] aggregated in oval crescentic [illegible] of a pin's head. They vary in size from that [illegible] a small pea and involve the entire [illegible] the surrounding tissue. [illegible] quadrating [illegible] is peculiar in that they rarely [illegible] depigmentation. The eruption [illegible] of more regions of the body, as the forehead, the chin, the nucha, the buttocks, and the outer surface of the thighs. It is less often disseminated than grouped. Occasionally, there may be but a single lesion, the recognition of its character usually demanding some skill on the part of the diagnostician. When occurring in groups, the typical circinate appearance of the syphilodermata in general may be wanting, the patches having an irregular boundary; but at times the circular, reniform, or horse-shoe-shaped outline is distinct, with an enclosed area of integument unaltered or the seat of atrophic changes. Whether resolving by absorption or degenerating by ulceration, they commonly leave the typical cicatrices of syphilis.

Fig. 300



Nodular syphiloderm. (Howard Morrow.)

Fig. 301

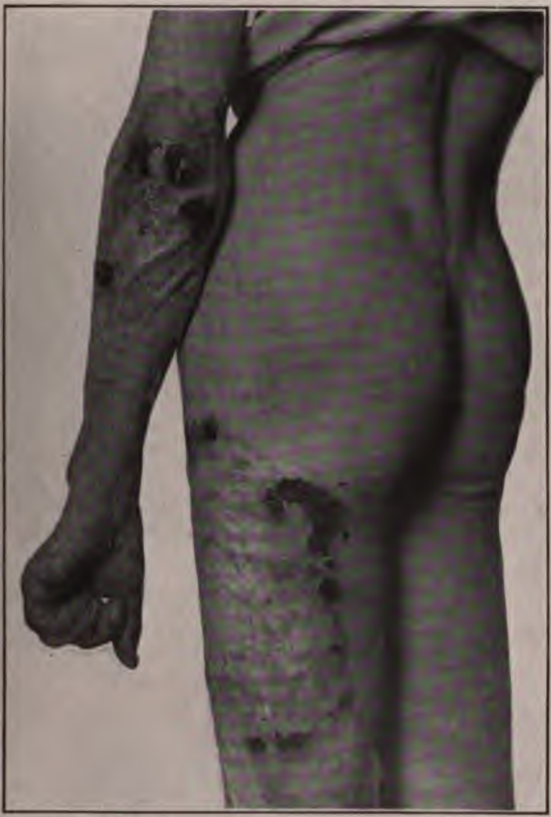


Nodular gummatous and ulcerating syphiloderm.

At times the lesions assume a ***serpiginous*** character and distribution, in which case secondary changes commonly occur. They may become covered on the surface with a thin, yellowish crust; may lose their firmness and become soft, and of a rather more lurid red in hue; from colloid or suppurative degeneration they may vegetate luxuriantly and become the seat (especially in the scalp) of warty growths, covered with a semipurulent secretion of disgusting odor (*syphilis papillomatosa, syphiloderma frambesioides*); or, finally, they may ulcerate, the superimposed crusts thickening in bulk, deepening into greenish or blackish shades, and covering typical syphilitic ulcerations, characteristic floor, edges, base, and secretion. The degeneration e latter case may be rapid and the destruction extensive.

e course of the eruption is indolent, months usually elapsing e full evolution is accomplished. In untreated cases there may be uced a generalized and symmetrical syphiloderm. It is rare, ver, even in hospital and dispensary cases, to observe such lopment. The more superficial, generalized and symmetrical the lesions, the briefer, as a rule, is the interval between such an tion and the date of infection. The later the lesions, the more hey asymmetrical, localized, and profound in their involvement e deeper tissues. This syphiloderm occurs more frequently in the and fourth year, less often in the second, and more rarely in the tenth or fifteenth year of the disease. Resolution occurs by tion, leaving in the site of the nodules, according to their age, nd contents, livid and pigmented maculations, or characteristic nted, atrophic, cicatriform areas. Scars following the ulcerative

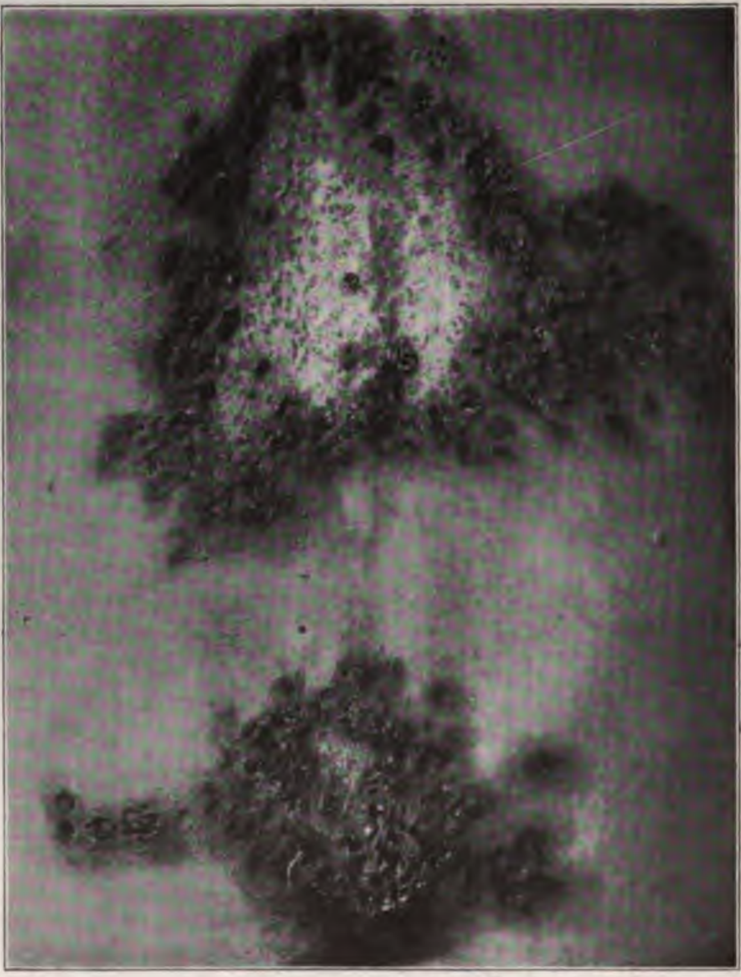
Fig. 302



rating and nodular syphiloderm. Note varicose veins in the scar on the thigh.

lesions are typical in color, shape, and career, the pigmentation of both cicatrix and areola blanching from center to periphery and leaving a delicate, dull-whitish, glazed, or slightly desquamating surface. Ancient relics of this process resemble thin, small-coin- and larger-sized, circular sheets of mica.

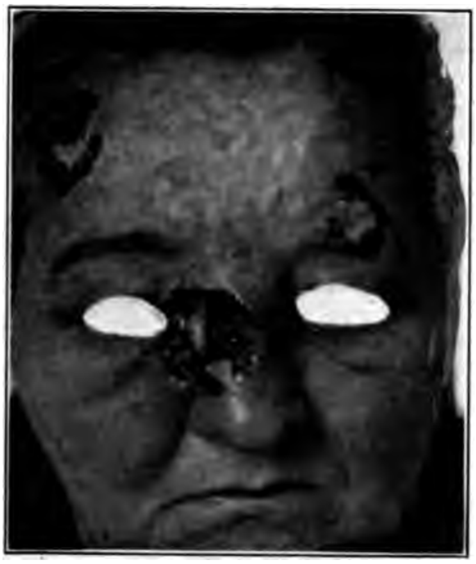
FIG. 303



Ulcerative nodular syphiloderm.

Various combinations of the nodules of syphilis with other lesions may occur, which has given rise to a number of different terms expressive of these associations. The rarer generalized forms are commonly of multiple type. The circumscribed groups, whether serpiginous or limited to a single region, frequently present variations which may cause confusion. For example, the affected patch may cover the surface of an entire limb or buttock, or merely involve the nose and upper lip; or possibly in irregular extension only the face. Whether of the resolutive or ulcerative class, the circumscribed patches commonly are sluggish in development and career, often sprinkled with superficially ulcerated points set in areas of atrophic or scarformed tissue; or, again, they may furnish a ring of ulceration, deep or superficial, about a healing center; or a group of well-defined ulcers may be beset with nodules of different types. The strictly serpiginous patch extends, whether over an ulcerated or partly healed center, by advancing a ridge of closely agglomerated or fused nodular lesions, crusted, ulcerated, or merely resolving by degeneration when treatment has been pursued. Even in exaggerated types of the serpiginous patches, the process in untreated cases is one of advance, repair, ulceration, and scarring in different grades at different points in the same area. The rupioid features seen in the larger pustular lesions are displayed here more rarely and less classically. It is of interest in the study of these cases to note that in almost every instance the disease either has been unrecognized, and therefore untreated, or is one occurring in the subjects of cachexia, debauchery, or extreme poverty.

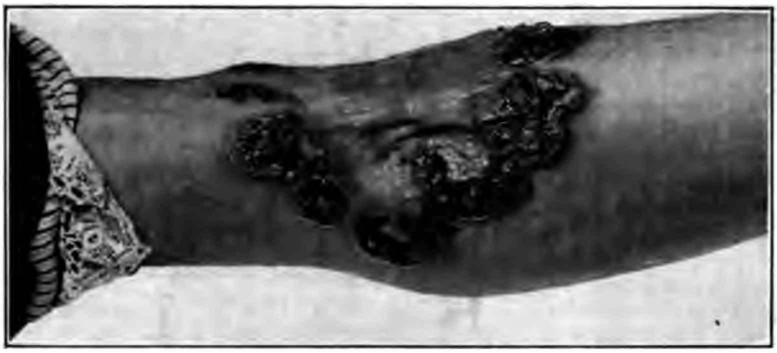
FIG. 304



Ulcerating serpiginous syphiloderm.

Diagnosis.—The diagnosis is between lupus vulgaris, lepra, epithelioma, acne rosacea, and psoriasis. In lupus, the age of the subject, the character of any scars left upon the body-surface, the chronicity of the disease, and the absence of a history of polymorphism, will point usually to the nature of the disease. A close resemblance between lupus and late syphilis about the nose is occasionally noted. The rapidity of the ulcerative process and bone-involvement likely to occur in syphilis are important points of distinction. The nodules of lepra are very much more indolent than those of syphilis, and have a characteristic oiled or varnished look, never the livid or dull-crimson color of syphilitic lesions. Set upon the forehead, the nodules of syphilis, near the line of the hairs, never give the leonine aspect of those at the lower border of the forehead and over the eyebrows of the leper. In epithelioma the age of the subject and the history of the disease are always significant. In the early stage of epithelioma characteristic "pearls" often may be recognized. While the patient suffering from epithelioma may enjoy excellent general health, the imprint of cachexia is often distinct in nodular syphilis of the skin. In the later stages of epithelioma, the ulcer with everted edges and eroded, hemorrhagic floor, "varnished" with a translucent secretion, is totally different from the "punched-out" syphilitic ulcer, with its puriform secretion and discolored crusts. The deep infiltration of even the desquamating nodular syphiloderm distinguishes it from the circular patches of psoriasis. In acne rosacea the telangiectases, characteristic redness, and frequent pustular lesions are suggestive, when considered in connection with the absence of ulceration. But in both sexes, subjects of syphilis in the middle periods of life, with nodules limited to the nose and head, often present themselves with marked rosacea from spirit-drinking, when the most careful examination is needed to detect the coincidence of the two disorders.

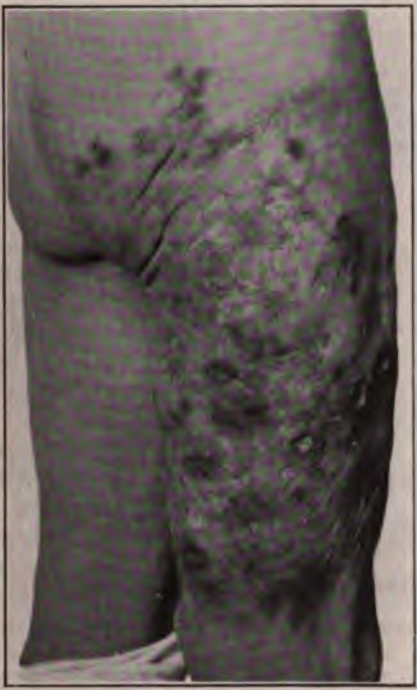
Fig. 305



Ulcerating syphiloderm somewhat resembling blastomycosis.

Gummatous Syphiloderm.—Gummatous lesions, as a rule, are late manifestations of the disease. In rare instances, in cases which might be classed as malignant or precocious, they may form a part of the early manifestations, in which case they are apt to be more generalized and symmetrically placed. Commonly they develop as one or relatively few subcutaneous, strictly circumscribed, firm, well-defined, painless, and indolent nodules or tumors, which when first observed are scarcely han a pea. They are then covered with an unaltered integu- nd are movable. Very slowly they increase in size until they he dimensions of a marble, an egg, or even of bodies of a larger ooner or later they become attached to the overlying skin, ssumes a livid reddish or purplish hue, surrounded by a hyper- eola. Finally, at one or at several points, the skin becomes and is incapable of further resistance, the gumma bursts, and k, sanious secretion, the gummy character of which has given n its name, escapes. When the inflammation has been active, retion may be wholly or partially purulent, and in this case is

FIG. 306

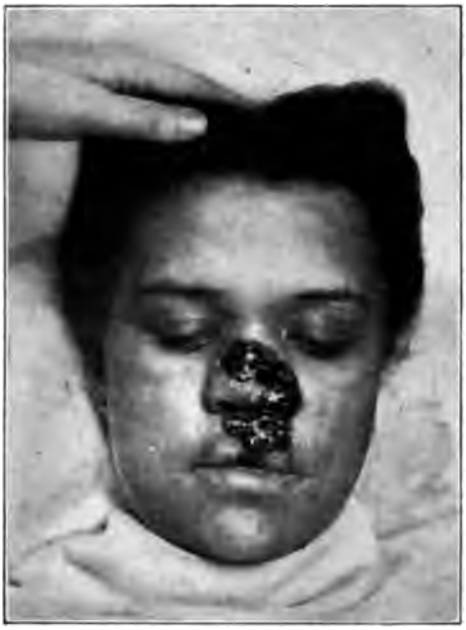


Ulcerating syphilitic gummata.

d either by the contents of the tumor or by the peripheral which participates in the process. Ulcers result, which oc- lly are fistulous in type, roundish or oval in contour, with clean- es and purulent floor, often extending to the subcutaneous tendons, aponeuroses, cartilage, or bone. Thin and yellowish r bridges of undermined skin often extend between several ions and usually melt down in the destructive process. When s progressing, which is the rule as regards the ultimate result, tion springs from the floor of the ulcer, the edges contract, e gummatous lesion eventually exhibits the appearance of a ulcer, save in the thin, purplish, pigmented appearance of the outlying integument. The scars are typical, pigmented at first, and bleaching from the center, and they may in rare instances be attached to the periosteum of the bone. Considering the depth of the process, the gumma of the skin is, as a rule, succeeded by less evidence of destruction than is threatened at the height of the process. About the neck cicatrices may be roundish in shape and slightly puckered; upon the extremities and the trunk they are usually circular or oval.

But one gumma may appear upon the person of a single individual, and when this is the case it will usually be found upon the leg. After this region, the forehead is most often attacked. Half a dozen or more may at times coexist; in rare cases hundreds form. Gummata may develop upon any part of the body, more particularly over the head (face, nose, lips), scalp, thighs, legs and arms, buttocks, and genitalia. When situated over the trunk on a nerve, they may become the seat of severe neuralgic pain. They are amenable to skilful treatment, and they may undergo resorption, leaving little trace of their former existence.

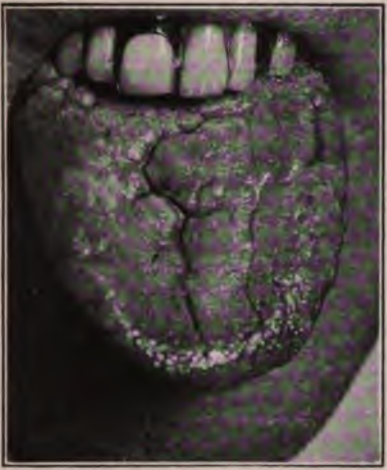
FIG. 307



Gummatous syphiloderm.

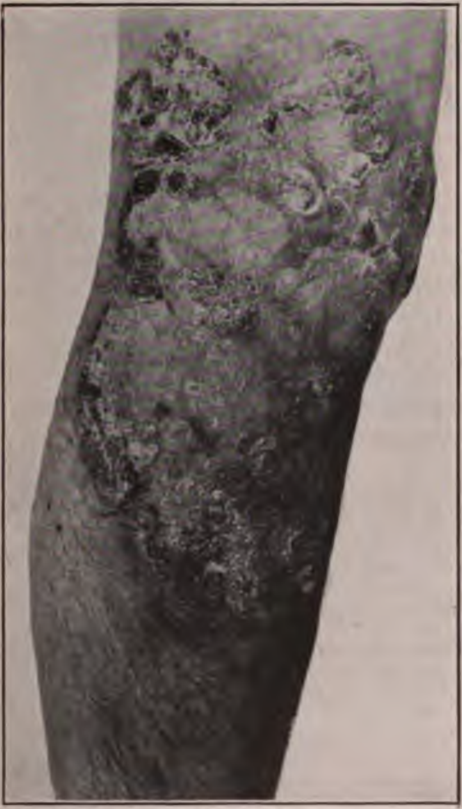
Diffuse Gummatous Infiltrations of the Skin and Hypoderm are either distinctly outlined, which is the rule, or occasionally ill defined at the border, varying in extent from a coin-sized patch to an irregularly outlined infiltration covering the integument of an entire limb. The central portion and borders of such an area may be constituted of partly fused, originally discrete lesions, as in the instance of the nodular lesions above described; or they may be made up of a thin plate of infiltration, breaking down at various points and showing actively ulcerating areas. In extreme cases loss of tissue may be great, furnishing the picture of a group of typical syphilitic ulcers having a sloughing floor and a precipitous edge. The patch or patches often form curiously outlined parts of circles in the classical figures of the letter *S*, the horse-shoe shape or the kidney, these composite groups including equal-sized circlets of infiltration, with border ulcers; or a larger central patch with gummatous infiltration is surrounded by smaller patches set in a circle about the former, as in the arrangement of pearls in a brooch. In less classical features there is presented

FIG. 308



Lingual gumma.

FIG. 309



Rupoid syphilide.

the rare picture of a swollen, engorged, almost elephantiasic organ (leg, vulva, nose), crusted, corded, ridged, knobbed, and seamed with larger or smaller ulcers or scars.

Diagnosis.—Gummata are to be distinguished from fibrous, carcinomatous, and lipomatous tumors, as also from indurated and enlarged lymphatic ganglia. As gummata of the skin occur in preponderance below the level of the knees, and are for the most part single or relatively few in such situation, by their position alone they frequently may be differentiated from each of the new-growths mentioned, no one of which occurs by preference upon the lower extremities. As they are, moreover, relatively late lesions of syphilis, a history of preëxisting symptoms of that disease usually can be obtained.

The element of time is of chief importance in the diagnosis, as the evolution of gummatous syphilis is more rapid than that of most tumor-formation affections. The characteristic "pearls" of epithelioma, and its situation chiefly on the face, will serve to suggest the diagnosis when the gummatous lesions are on the extremities. Lupus of the extremities is rare. Gummata of the face are confused most often with the two disorders last named. Invariably, in all doubtful cases in the male sex, the testicle should be examined, as frequently a tell-tale gummatous infiltration of the epididymis or testicle proper, unrecognized by the subject of the disease, clears up the doubt.

Reinfection and Superinfection in Syphilis.[1]—A second attack of syphilis initiated by a primary lesion was of rare occurrence under the older methods of diagnosis and treatment. During the past several years many such cases have been recorded by competent observers.

Superinfection has also been the subject of many reports. In this type a new local lesion develops after a fresh inoculation in an individual already the subject of syphilis. This lesion usually assumes the type that is characteristic of the stage at which the disease has arrived following the earlier infection. It is frequently a non-indurated ulcer which develops after a short period of incubation, contains but few *Spirochetæ pallida*, and is not followed by the development of other symptoms of the disease but is purely a localized manifestation.

Congenital Syphilodermata.[2]—Congenital syphilis may present active manifestations at birth or in early infancy, or at a later date, early adult years. Recent investigation appears to prove ajor portion of infections are of maternal origin. Opinion this question. The character of the disease, including the manifestations, varies, depending upon the period of gestach infection of the fetus occurred and the stage and virulence ase in the progenitors. When infection occurs early death t and the fetus be expelled in a macerated state. When later, it may be born at term or prematurely, and may or xhibit symptoms of the disease. According to Kassowitz[1] r premature delivery occurs in 40 per cent. of syphilitic s.

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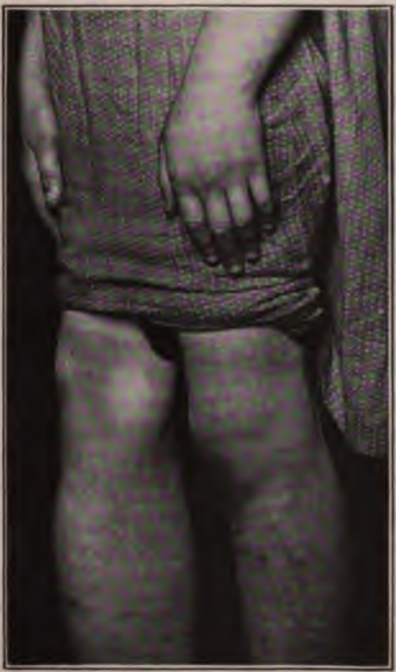
[1] *Cf.* Western, Brit. Jour. Derm., 1914, xxvi, p. 280; Hoffman, Muenchen. med. Wchnschr., 1914, lxi, p. 2063; Parounagian, New York Med. Jour., 1915, ciii, p. 153; de Laviergne, Amer. Jour. Urology and Sexology, 1916, xii, p. 18; White, Brit. Med. Jour., 1917, xi, p. 509: Reinfection in syphilis; observations in 27 cases; Klauder, Jour. Cut. Dis., 1918, xxxvi, p. 514: Superinfection in Syphilis; report of cases; Schamberg, Trans., Sec. on Derm., Amer. Med. Assoc., 1919, p. 98: A second attack of syphilis, two years after the first; Pollitzer, Jour. Amer. Med. Assoc., 1920, lxxiv, p. 775: Case report reinfection.

[2] Boardman, Jour. Cut. Dis., 1914, xxxii, p. 545: Syphilitic Heredity and Congenital Syphilis. (An excellent review of the subject of pertinent literature, references and findings.) Hyde, Med. News, December 4, 1897: What Conditions Influence the Course of Infantile Syphilis? Heimann, New York Med. Jour., September 23, 1916: Congenital Syphilis. Wile, Jour. Cut. Dis., xxxiv, p. 645: Some Observations on the Transmission of Syphilis, with Particular Reference to a Paternal Source of Infection. Post, ibid, 1916, xxxiv, p. 589: Congenital and Bone Syphilis. Fraser, Archiv. of Derm. and Syph., 1920, i, p. 491. "The Pathology of Congenital Syphilis." An excellent discussion with review of literature.

FIG. 310



t saber shin and left saber ulna in a congenitally syphilitic girl.

the early manifestations of congenital syphilis are exhibited first four weeks after birth and practically always within hs. It is rare for such symptoms to develop subsequent to

al syphilis differs from the acquired form chiefly in the a primary lesion, greater severity of symptoms, a marked ng of cutaneous lesions with little or no chronological order evelopment, and in the presence, frequently, of bullous utaneous symptoms may be present at birth, in which case the disease is usually severe and the prognosis grave; or the child may be born with a clean skin, be plump and well nourished, and the cutaneous manifestations occur at a later date. The earlier the date of appearance of lesions, the more intense, as a rule, is the disorder. Often the first symptoms exhibited are significant of visceral involvement, and are, in brief, those of marasmus. Emaciation progresses rapidly; the skin seems stretched unnaturally over the facial bones; the expression is that of physical distress; the cry becomes a fretful moan; the integument loses its normal color, and acquires instead a sallow or muddy tint. The resemblance of the facies of these emaciated infants to those of the aged is striking.

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[1] tz, cited by Osler and Churchman, Osler's Modern Medicine, iii, p. 489.

Coryza is an early symptom, due to the specific involvement of the Schneiderian membrane. The discharge from the nares (at first serous, later purulent) desiccates sufficiently to obstruct the nasal passages, or, in consequence of the tumid condition of the membrane lining the passages, it is prevented from escaping. At times, crusts accumulate externally about the nasal orifices, and they are seen to be similar to those that are prone to form also at the angles of the mouth. In this way, the characteristic "snuffles" of the syphilitic infant are induced, in consequence of which it is obliged, when nursing, to release the nipple from the mouth in order to respire, an act often accompanied by a hoarse cry, the latter being due to the tumid and actively secreting mucous membrane of the larynx. The breathing of a syphilitic infant, even when asleep, or awake and undisturbed, is often sufficient to arouse a suspicion as to the nature of the disease from which it is suffering. The mouth is frequently the seat of mucous patches and the vulva and anus often exhibit condylomata.

Bullæ in congenital syphilis may be early or late manifestations of the disease, and they may be represented by a single lesion on the palms or soles (the site of their predilection), the fingers, toes, or extremities; or they may constitute a symmetrical generalized efflorescence. They are not infrequently the first manifestation of the disorder. In severe cases, the bullæ are ill-developed, and the integument will be seen to be marked here and there by small-coin-sized and larger disks or plaques of macerated epidermis, separated from the corium by a thin film of serous, sanious, or purulent fluid, in quantity insufficient to raise the roof above the general level of the integument. When fully developed, they may be conical, rounded, flat or flaccid, and be surrounded by an infiltrated border of dark-reddish or violaceous hue. Their color varies with the color of their contents. In extreme cases they may be distended with blood only. Their subsequent career is concluded by shallow or deep ulcerations, the floor of each bulla secreting a sanious discharge. Crusts may be formed if the patient survives. A fatal termination is usually announced by flattening or collapse of the blebs. The eruption may be uniform, due to the fact that bullæ represent the site of feeblest resistance in the epidermis, and the fluid exudate mechanically separates the rete from the tissue beneath. The lesions may be limited to the palms and soles, which is the more common occurrence; or they may be generally diffused over the cutaneous surface; and, finally, they may be commingled with pustules, maculo-pustules, condylomata, and mucous patches of the anus, the mouth, and the nares.

Macules, in congenital syphilis, appear early, may be discrete, irregular as to shape and size, and of a pinkish or dark-red color. They occur first over the region of the buttocks and extend from there over the limbs, involving also the face and neck. By coalescence, a more or less diffuse, coppery-red, or violaceous, glazed or moist and secreting surface is formed, affecting an entire region, such as the genitalia, the trunk, the thighs, the neck, the palms and the soles. Desquamation frequently follows, which is in sharp contrast to the course of the macules occurring in acquired syphilis. Infiltration of the erythematous areas occurs, the skin becomes thickened, and not infrequently in the extensive patches deep excoriations and fissures occur. From the fluid exudate irregular crusts are formed, producing a general aspect somewhat similar to that seen in eczema, yet differing markedly in color from the latter disease. In rare instances, a diffuse, reddened, scaling condition develops, which resembles to a marked degree the general exfoliating dermatitis described by Ritter. In moist regions the epidermis covering the erythematous areas frequently becomes removed, leaving a raw, exuding surface.

The *papules* of congenital syphilis resemble in many respects those of the acquired form and likewise vary as to size, shape, evolution and involution. They may be acuminate, convex, flat, large or small, smooth or scale-covered; they may spring from preceding macules or develop independently; and may terminate in pustules, bullæ, or condylomata. Small papular forms may be of extensive or limited distribution, and occur chiefly on the limbs. They may be diffuse or grouped, scale-covered, or capped with a pustule. More commonly, dull-red or violaceous papules of lenticular size occur either in symmetrical or asymmetrical arrangement, and are either discrete or agglomerated in patches of infiltration. About the buttocks and other regions the papules may scale at the apex, while over the palms and soles they may constitute by fusion a desquamating, epidermal patch; or, commonly about the ano-genital region, the interdigital spaces, the axillæ, and the face, they may become moist and secrete a puriform mucus. By vegetation or by hypertrophy, the papules develop into flat or fissured condylomata, varying in size from that of a small coin to a lesions an inch or more in diameter, with corresponding variations in the degree of their elevation from the affected surface, which are covered with an offensive yellow or yellowish-white discharge. These condylomata may be few or numerous; at times a large portion of the cutaneous surface is covered with large, moist, secreting papules. Occasionally, they may become ulcerated and encrusted. Rhagades frequently occur about the angle of the mouth upon the site of an infiltrating erythematous lesion or papule, occurring as the result of movement of the affected parts. The linear scars resulting from these are characteristic of the disease. At the height of their development, opening and closing of the mouth is both difficult and painful.

Vesicles in hereditary syphilis are rare. They are described as having a conical apex and serous contents; as being closely set together about the lips; and as springing from a violaceous, infiltrated patch. Crocker (p. 883) describes vesicles of millet-seed size appearing singly or in groups, and with little or no redness at their base, which subsequently developed into pea- to hazelnut-sized bullæ. Grindon[1] described 2 instances of true vesicle formation occurring in hereditary syphilis, in both of which the vesicles were rather extensively distributed.

Pustules in congenital syphilis may be discrete or confluent, localized, or, very rarely, generalized. As above described, they not infrequently occur consecutively to papular lesions, and are an evidence of a cachetic condition. They are prone to occur in groups about the mucous outlets and in association with other lesions of the disorder. They may ulcerate and become covered with bulky, dark-colored crusts. Pustules may be seen in typical development by the side of the nail, occasionally involving the matrix, and producing in this situation considerable swelling of the digit, with an ulcerative sequel, which commonly results in distortion or in ultimate loss of nail-substance.

Small abscesses resembling furuncles may occur, which may be few or numerous, and are characterized chiefly by their indolence.

Nodules and *subcutaneous gummata* may develop in congenital syphilis. but only as late manifestations of the disease, one or more years elapsing before their appearance. Their behavior is like that observed in the acquired form of syphilis, although the destruction induced by their degeneration in very late manifestations may be of the most intractable type. The subsequent ulceration and destruction of tissue may be marked in these cases, particularly about the anus and mouth. Usually there is a history of preceding parental disease, and coincident symptoms or sequels of such disease as is manifested by altered teeth (as described by Hutchinson), an ancient keratitis, and bone deformities.

Mucous patches are constant symptoms of the disease, and they represent papules of the mucous membranes that differ from those in the skin only because they are moistened, macerated, and flattened by juxtaposition of neighboring tissues. They are surrounded usually by a lurid halo. and they may have the pearly whiteness always seen when the epidermis of mucous membrane is wholly or partially detached from the corium; or they may lose this protecting covering in shreds or patches. leaving a denuded or ulcerating surface. They may be isolated or broadly confluent, and be oval, circular, or decidedly linear in shape. the last-named appearance being characteristic of patches existing at the angles of the mouth.

Diagnosis.—The diagnosis of congenital syphilis usually presents little difficulty. A combination of lesions, such as condylomata about the ano-genital region, erythematous and papular lesions about the mouth, associated with rhagades; the characteristic rhinitis, possibly also laryngitis, with associated cachexia, leave little doubt as to the nature of the malady. Pemphigus and bullous impetigo are not limited to the palms and soles and are unaccompanied by the associated conditions above described. Eczema and intertrigo of the genital region are differentiated by the lighter color of the lesions and the absence of infiltration and constitutional impairment.

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[1] Jour. Cut. Dis., 1910, xxviii, p. 284.

Prognosis.—Congenital syphilis is a grave disorder, but many infants survive when efficiently treated. The longer the interval between birth and the development of symptoms, the more favorable becomes the result. Deformities not infrequently occur and involvement of the central nervous system is to be remembered.

Etiology.—Syphilis, in the course of which appear the syphilodermata, is produced by infection, either accidental, intentional, or *in utero*. The causative organism reaches the blood through the medium of the lymphatics, and is subsequently distributed throughout the body. The physiological secretions of the infected, uncontaminated with pathological products, are believed to be incapable of acting as virus-carriers; but, especially in the recently infected, such contamination is of frequent occurrence, and is effective in the transmission of the malady.

The methods of transmission may be immediate, as in sexual congress, in kissing,[1] in medical examinations, and in nursing at the nipple, by which act the child may infect the nurse with the secretion of the mucous patches in its mouth; or the infant may, instead, receive the disease from excoriations on the breast of the nurse. The author has seen many examples of infection on the fingers of physicians acquired in medical examinations; also several on the eyelids, and several on other parts of the face and neck, including two just within the nose. The latter appear to have been due to picking or rubbing the parts with the fingers contaminated with the infective material from syphilitic patients. The disorder may also result through the medium of utensils charged with an infectious secretion, such as drinking-cups, the needles of the tattooer wet with saliva commingled with diseased mucus, or the lancet of the vaccinator covered with infected blood. Generally it may be said that all the discharging and moist syphilodermata are sources of danger to a sound individual, both in the acquired and the inherited forms of the disease. By these and other similar methods persons of both sexes and all ages may become infected.

The lesions may be accentuated by external irritations, such as a hot bath, friction, uncleanliness, especially in infants by means of the soiled napkin; while late lesions may be determined to certain situations by injury.

Of the vast number of microörganisms alleged to be etiological in syphilis, only three need be mentioned. The first of these, the bacillus of Lustgarten,[1] discovered in 1885, is largely important on account of the great interest its discovery excited and the controversy which followed as to its rôle. The second is *Cytorrhyctes luis*, isolated by Siegel,[2] which is important only from the fact that it led to the discovery of the third and most important of these microörganisms, *Spirocheta pallida*. Siegel's cytorrhyctes was considered of sufficient importance to be investigated by the German Academy of Sciences, which appointed Schaudinn and Hoffmann to undertake its study. During this investigation Schaudinn[3] discovered a motile spirillum now universally accepted as the cause of syphilis.[4] *Spirocheta pallida* has been demonstrated in practically every lesion of syphilitic origin. All the organs of the new-born child affected with syphilis have been shown to contain the spirochetes in large numbers. They have also been isolated from tissues and organs in experimental syphilis.[5]

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[1] Schamberg, Jour. Amer. Med. Assoc., September 2, 1911, p. 783: An Epidemic of Chancres of the Lip from Kissing.

The spirochetes first penetrate into the interstices of the tissues, and after that reach the blood- and lymphvessels. In the tunica intima of the lymphatic vessels, spirochetes which have come from these sources cause changes in the connective tissue.

Spirocheta pallida[6] averages in length 6 to 15 microns, although in some instances it may measure 16 to 20 microns. Its transverse measurement varies from one so minute that it can hardly be seen up to three-fourths of a micron. Its spirals number from 6 to 14 or more; the length and depth of the spirals varying from one to one and one-half microns. It is cylindrical, thus differing from other spirochetes, which are flattened. Another peculiarity is its spiral shape, whether in motion or at rest. It is a very delicate microörganism, and is destroyed rapidly in the absence of moisture. It has been kept alive for several days,[7] but as a rule it retains its pathogenic power outside of the body for only a few hours (Metchnikoff). For immediate examination, the dark-background illumination method is preferable (Landsteiner and Mucha). This procedure can be practised in the ordinary routine of office examination as follows: Having the necessary condensor and a light of sufficient intensity, and also the proper objective and ocular for the microscope, a smear which has recently been prepared is examined, when the motile organisms are plainly visible in the field, and with some experience are readily differentiated from other spiral organisms. The smear is best made by thoroughly cleansing the initial lesion, then producing irritation with a sharp instrument, which induces some serum to exude. A droplet of normal-salt solution is applied to this, then the material is drawn into capillary pipettes, from which it is placed on the slide. If examined within a short time after removal, little difficulty is experienced in finding the organisms. The chief staining methods that are employed are the Giemsa, as modified by Schaudinn and Hoffmann; Marino's method,[1] and Levaditi's modification of Ramón y Cajal's silver stain (particularly valuable as a tissue stain). The quicker method is by the use of India ink, or by the use of the Goldhorn stain.[2] The organism has been grown in pure culture by Noguchi,[3] who isolated six strains from material obtained from orchitis in rabbits and seven strains from initial lesions, condylomata, and papules in the human subject.

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[1] Wien. Med. Jahrbuch, 1885, p. 517.

[2] Abhandlungen d. K. preuss Akad. d. Wissenschaften, 1905, Abt. iii, s. i–xv; and Münch. med. Wochenschr., 1906, i, p. 63 (quoted by Metchnikoff: "A System of Syphilis." Power and Murphy, London, 1908, i, p. 49).

[3] Arbeiten aus der k. Gesundheitsamte, 1905, xxii, p. 527.

[4] For a review of early work concerning *Spirocheta pallida*, see Lévy-Bing, Paris, Octave Doin, 1907; and Hoffmann, Verhandlung. der Deutsch. derm. Gesellschaft, 1907, p. 193 (750 articles).

[5] Centralb. f. Bacteriologie, etc., 1907, xliv, p. 223.

[6] Hartmann and Prowazek, Arbeiten aus der k. Gesundheitsamte, 1907, xxvi, p. 14.

[7] Centralb. f. Bacteriologie, 1906, xli, p. 741.

Pathology.—The histological changes in syphilis have been studied recently by Unna,[4] Fordyce,[5] Crocker,[6] and others.[7]

The histological findings vary according to the lesion examined. In all forms except the macular syphiloderm, the changes are similar, varying chiefly in the degree of involvement of the bloodvessels and the amount of cellular infiltration. The vascular changes in all forms are characteristic, being most marked in late lesions. A cellular infiltration, composed of lymphocytes, plasma-cells, and occasionally multinuclear and giant-cells, is characteristic of the disorder. In the primary lesion there are found an increase in the bloodvessels and changes in their walls; a perivascular and more profuse cellular infiltration; changes of a greater or less degree in the collagen and elastin; and secondary changes in the epidermis. According to Hoffmann,[8] the spirochetes first enter the lymph-spaces of the rete, where they are protected against phagocytosis; or find their way into the lymphvessels of the papillæ. After about three weeks a reaction occurs, due to multiplication of the organisms, indicated by an inflammation of the lining of the lymphatics and a perilymphangitis, followed by proliferation of the capillaries, cellular infiltration, and an increase in the connective tissue. According to Ehrmann, the spirochetes first penetrate into the interstices of the connective tissue, and after that find their way into the blood- and lymphvessels. In a fully developed chancre, Fordyce found acanthosis, followed by new capillary formation and perivascular cellular infiltration, with invasion of the epidermis by leukocytes, diffuse cellular infiltration in the papillary and upper reticular layers of the corium, dilatation and increase in the blood- and lymph-capillaries, and some thrombosis. In the same area thickened and occluded vessels were noted; while in the adjoining tissue endo- and peri-arteritis occurred, with a perivascular infiltration composed largely of plasma-cells. Bands of hypertrophic connective tissue surrounded some of the vessels, a part of the active infiltration.

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[1] Annales de l'Institut Pasteur, 1904, p. 761.

[2] Jour. Exper. Med., 1906, viii, p. 451.

[3] Jour. Amer. Med. Assoc., 1912, lviii, p. 1163.

[4] Histopathology.

[5] Jour. Amer. Med. Assoc., 1907, xlix, p. 462: The Vessel Changes and Other Pathological Features of Cutaneous Syphilis (with 12 microphotographs).

[6] Dis. of the Skin, 3d ed., p. 845.

[7] Dennie, Jour. Cut. Dis., 1915, xxxiii, p. 509; The Histopathology of Some of the Secondary Skin Eruptions of Syphilis. (A study of the situation of Spirocheta pallida in the lesions). Fordyce, Ibid., 1915, xxxiii, p. 802. The Pathology of Syphilis. (A valuable presentation of the subject as a whole).

[8] Die Etiologie der Syphilis. Monograph, 1906.

In the macular lesion the bloodvessels are dilated, and there is found a perivascular cellular infiltration, consisting of small round cells. The vessel walls exhibit proliferative changes, due to the inflammatory reaction present. The normal wavy line between the corium and epidermis may be obliterated by the pressure from the products of the inflammatory reaction in the corium, or be normally present. In this lesion no changes occur in the appendages other than a moderate cellular infiltration.

In the large papule the chief changes are found in the corium, in which situation a marked cellular infiltration, composed of lymphocytes and plasma-cells, chiefly occurs, with vascular dilatation and thickening of the vessels by proliferation of the cells of their endothelial and fibrous coats. Plasma-cells multiply by mitosis, and by proliferation of connective-tissue nuclei spindle- and spider-cells are formed. According to MacLeod, the plasma-cells frequently occur in rows, due to infiltration between the collagenous bundles. In the epidermis, acanthosis, edema, and parakeratosis are found, with infiltration of polynuclear leukocytes.

In the small miliary papule Fordyce described an infiltrate surrounding the hair follicle, composed of a reticular structure holding new-formed bloodvessels and round, plasma-, and connective-tissue cells in its meshes. The vessels in the center were obliterated, while those at the periphery were dilated, thrombosis occurring in the superficial ones. At a distance the bloodvessel walls were thickened and surrounded by a mantle of round and plasma-cells. Fatty degeneration was noted in the coil-glands, which were surrounded by the cellular infiltrate above described.

In the nodular syphilide the vascular changes and cellular infiltration resemble to a marked degree those found in the papules above described. The bloodvessels are increased in number and show evidence of endarteritis. In the cellular infiltration numbers of polynuclear and giant-cells may be noted in addition to the plasma-cells, lymphocytes, and connective-tissue cells. In most cases a certain number of mast-cells and leukocytes also occur. According to Fordyce, the bloodvessels are increased in number, are the seat of an obliterating endarteritis, and are exhibited as solid cords of irregularly grouped cells, with pale-staining nuclei, accompanied by giant-cells with both peripheral and central nuclei. At the edge of the infiltration the bloodvessels show hyperplasia of all the coats and their lumen is narrowed. Thrombosis occurs in the lymph spaces, and fatty degeneration and regressive changes are noted in the connective tissue. The evolution of giant-cells in the condition is believed by a number of observers to be from plasma-cells. According to Fordyce, they are produced in most instances by thrombosis of the vessels, with proliferation of the endothelium, and he explains the extension of the serpiginous forms by a progressive thrombosis of the vessels at the periphery of the lesion.

According to Neumann, gummata are recrudescences about the vessels, produced by a latent virus aroused to activity. Neisser[1] explains their development by the lighting into activity of spirochetes that have remained dormant. The minute structure of the gumma shows a dense infiltration of plasma-cells, lymphocytes, and fibroblasts, areas of caseation, and marked vascular involvement. The vessel-walls are thickened, their caliber reduced, and many are obliterated. By degeneration of the cellular deposits, a gummatous mass is produced, which breaks through the epidermis, producing the typical ulcer; or by their resorption an atrophic area replaces the normal structures. The overlying epidermis is thinned before rupture, and subsequently is destroyed by ulceration.

Diagnosis.—The differentiation of the various cutaneous manifestations of syphilis from disorders which they resemble has been referred to under their individual descriptions. While in most cases the recognition of these lesions is a comparatively simple matter, in certain cases, owing to a close resemblance to other disorders, difficulty arises. It is therefore necessary in all cases to investigate in the fullest manner the history of the disease; of all prior skin lesions; of a possible primary sclerosis; of adenopathy; of all disorders affecting other organs of the body; and in women miscarriages and abortions. Often a single extra-cutaneous symptom is a valuable aid in establishing the diagnosis of syphilis. Not infrequently, the true nature of a peculiar eruption is decided by finding a characteristic patch on the tongue or the tonsil. It is therefore essential to examine the mucous membranes, the bones, and the viscera in doubtful cases, as they rarely fail to give evidence of systemic invasion when the disease is exhibited for any length of time.

Other cutaneous disorders occur during the course of syphilis, which are to be recognized and properly interpreted. The medicamentous eruptions, particularly those induced by the ingestion of potassium iodid, occur commonly and are at times confusing.

The diagnosis of syphilis has been largely perfected in all its stages by laboratory methods, consisting, early, in the microscopic demonstration of *Spirocheta pallida*, later in the Wassermann reaction, and still later in the luetin test.

Microscopic examination of the primary lesion during the first few days after its appearance reveals the presence of *Spirocheta pallida*, and thus renders an early positive diagnosis possible; whereas, in the past, much time was lost in waiting for the development of other symptoms. After two or three weeks, or in some cases a little longer period, the Wassermann reaction becomes positive, and this test then remains of the greatest value during the entire active stages of the disease, and of marked value, also, in the later stages. In the later and so-called latent periods of the disease, the luetin test becomes more useful.

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[1] Die experimentalle Syphilisforschung nach ihrem gegenwärtigen Stande. Monograph, 1906.

The Wassermann reaction, while a most valuable addition, should be viewed as confirmatory of other findings, rather than as an absolute proof in itself.[1] Fildes[2] found in a review of the literature that the Wassermann reaction was reported as positive in 65 per cent. in primary, 90 per cent. in secondary, 83 per cent. in tertiary, and 44 per cent. in latent syphilis; and quotes Boas' figures as 73 per cent. in primary, 97 per cent. in secondary, 95 per cent. in tertiary, and 37 per cent. in latent syphilis. Certain other disorders, such as nodular leprosy, frambesia, sleeping sickness, and scarlet fever, not infrequently yield a positive Wassermann reaction. It is therefore evident, in view of the fact that it may occasionally be negative in positive cases and *vice versa*, that it must be considered only as a valuable aid in diagnosis.[3] By many this reaction is looked upon as a symptom, which may be in abeyance from treatment or otherwise for varying periods of time, and may then reappear. Again, there are certain cases, particularly in congenital syphilis, where the reaction apparently remains permanently positive.

From the above, it becomes evident that, in the absence of a few disorders which rarely come into consideration, repeated negative Wassermann reactions over a considerable period of time (at least two years) usually, but not always, mean eradication of the disorder. A single test in doubtful cases should not be regarded as conclusive in either direction, and here repeated tests must be employed. A point of importance that every practitioner of medicine should recognize relative to these tests is the fact that a positive reaction does not necessarily mean that the cutaneous lesions under consideration are due to syphilis. A scar-leaving acne and other chronic dermatoses not infrequently are present in patients exhibiting no cutaneous evidences of syphilis. With the present refinement of technique, the element of error is reduced to a minimum and the employment of the Wassermann test is essential.

The luetin test[4] is chiefly of value in the late and latent stages of syphilis when the Wassermann reaction may be negative. Its value

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[1] Ravaut, Annales, May, 1914, v, p. 285. [2] Brit. Jour. Derm., 1911, xxiii, p. 13.

[3] See Fordyce, New York Med. Jour., September 26, 1914; Craig, Amer. Jour. Med. Sc., January, 1915. Summary of present status of the test. Discussion based on 18,000 personally performed tests. For discussion of contradictory reports see Wolbarst, Interstate Med. Jour., February, 1915; Uhle and Mackenney, Jour. Amer. Med. Assoc., September 14, 1915; Shropshire, South. Med. Jour., March, 1916; and Litterer and Watterson, Ibid., April, 1918; also see Solomon, Jour. Amer. Med. Assoc., 1920, lxxiv, p. 788. Agreement in Results of the Wassermann Reaction: a Study of Tests Performed by Two Laboratories in 3000 Successive Hospital Admissions. For references regarding technic and standardization of the test see section devoted to Wassermann technic under general diagnosis.

[4] *Cf.* section on General Diagnosis for technique and description of the reaction.

through the various Boards of Health, printed slips explaining all these matters are furnished the patient at his first visit. Before this was done it was the rule among men who saw many patients to furnish them with a printed copy of a similar nature. As a rule, assurance may be given that the course of the disorder is often benign, that the disease is productive of little discomfort and in most cases is curable, but that it requires strict adherence on the part of the patient to the rules laid down for its management. The patient should be instructed concerning the value of a nutritious diet, the importance of obeying the laws of hygiene and in doing everything possible to promote his physical resistance. In most cases hot Russian or Turkish baths should be interdicted, inasmuch as they tend to promote hyperemia and favor the occurrence of eruptions. Cool or tepid baths are to be employed sufficiently often to insure cleanliness and may be used in any way that is convenient. The teeth, mouth and gums require constant care. As a prophylactic measure the use of a tooth brush several times daily is a matter of importance. In case any disease of the gums is present it should be treated. Equal parts of tincture of cinchona and tincture of myrrh rubbed over the gums twice daily is of value when tenderness or other local changes in the gums occur. In case the teeth require attention from any cause this should be given by a competent dentist.

Tobacco in every form is injurious and its use should be interdicted. Malt liquors, wines and spirits should rarely be allowed, and if so only under the direction of the physician. They may be of some service in the cases of depleted subjects, and need not be prohibited entirely in those long accustomed to their use. The improper use of stimulants is in the highest degree detrimental. As all of the remedial agents mentioned above are used to some extent, their individual description will follow, although some of them are employed to a very limited extent at this time. The difficulties attending the administration of arsphenamin are of such a character that its universal employment cannot be expected, and it is for this reason that some of the older methods are still used.

MERCURY.—*Ingestion.*—The internal administration of mercury has been employed to a greater extent than any other method, and has been described by eminent authorities over the entire world. It is being used less and less, however, as other methods have become better understood: While certain recent observers believe that very few patients thus treated entirely recovered from the disorder, yet the records of cases of men with large experience show comparatively satisfactory results. Should this method be selected, the treatment, as with other methods, should be instituted as soon as the diagnosis is made. When employed generally it was found that it could be tolerated by practically 98 per cent. of patients. In France and America the protiodid has been most extensively prescribed. It is usually administered in pill or tablet form in doses of $\frac{1}{6}$ (0.01), $\frac{1}{5}$ (0.013), $\frac{1}{4}$ (0.016), or $\frac{1}{3}$ (0.022) of a grain three times daily. The dose may be increased gradually, according to the necessities of the case, from $\frac{1}{2}$ (0.033) to 3 (0.2) and even 4 (0.266) grains in twenty-four hours. Beginning with a minimum dose, this remedy is to be steadily administered and the quantity used daily increased until the degree of tolerance of the patient has been ascertained. Should the stools become frequent, pain be excited or slight effect be produced upon the mouth, as indicated by a metallic taste, moderate increase in the quantity of saliva, or any noticeable degree of tenderness of the gums, the dosage is to be gradually diminished until these symptoms disappear. Often the withdrawal of $\frac{1}{2}$ (0.033) or $\frac{1}{5}$ (0.013) of a grain daily will be sufficient to enable the patient to tolerate the quantity thus diminished. The medication is to be continued until the symptoms of the disease have disappeared, and subsequent to that at intervals for an indefinite period.

In the "tonic treatment of syphilis," the dosage is increased only on each third or fourth day until irritative effects are produced when, after an interval of two days, the quantity taken at the time of the production of such effects is reduced one-half. This reduced quantity is termed the "tonic dose" and is thereafter continued throughout the treatment.[1] With relapses or recurrences the dose is again to be increased until these are overcome.

Other preparations that may be used in the early stages are the mild chlorid, the tannate, blue mass, the bichlorid, the bicyanid, and hydrargyrum cum creta. The latter preparation was in the past a favored one with some English authorities. Morris[2] preferred the blue pill given in dosage of 1 to 3 grains three times daily. The bichlorid and biniodid are, as a rule, reserved for the later stages, when they are given in connection with potassium iodid, as they are soluble salts. In certain cases the mercuric iodid may be substituted for the mercurous iodid, beginning with a minimum dose of $\frac{1}{64}$ (0.001) and increasing this gradually to $\frac{1}{40}$ (0.0016) or, rarely, to $\frac{1}{20}$ (0.0033), either in pill or in solution. The average dose of $\frac{1}{40}$ (0.0016) of a grain in pill form administered three times daily, soon after eating, is tolerated by the majority of patients without deleterious effects.

Calomel may be administered in 1- or 2-grain (0.066–0.133) doses three times daily in combination with an opiate to prevent its action on the bowels, or in $\frac{1}{10}$-grain (0.0066) dosage every hour during the day for two, three or four days. Gray powder in 1-grain (0.066) doses, or the tannate in $\frac{1}{2}$ to 1 grain (0.0033–0.0066) three times daily may be employed. The bichlorid in doses of $\frac{1}{20}$ to $\frac{1}{12}$ (0.0033–0.005) of a grain is administered in pill form or in solution and has been extensively employed. It is perhaps better reserved for later periods, when it may be given in combination, as above stated.

A rotation of the various preparations above mentioned has often been found of value, rather than the continuous employment of a single one. The use of iron in connection with the internal administration of mercury has been the rule. The massa ferri carbonatis in

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[1] Amer. Jour. Med. Sc., xcvii, p. 17; Philadelphia Med. Times, 1882, xii, p. 337.

[2] Diseases of the Skin, 5th Ed., 1911, p. 504.

5-grain (0.33) dosage after meals, or the citrate of iron with quinin administered before meals, or other ferruginous preparations are commonly employed. It has been customary to administer mercury energetically during the first year, intermittently during the second year, with a few courses during each of the third and fourth years.

Inunction.[1]—Mercury may be introduced into the system satisfactorily by the method of inunction, and has been thus used for several centuries. When so employed it is absorbed readily and its therapeutic value is great. Inunctions should be employed in every case which admits of them, since the gastro-intestinal tract is left undisturbed and, furthermore, when properly used are much more efficient. Mercurial ointment combined with lanolin is valuable for this purpose. Cleanly but less efficient substitutes are provided in the oleate of mercury in a strength of 10, 15 or 20 per cent. Schamberg, Kolmer, Raiziss and Gavron[2] found by experimental work that when inunctions were used the major portion of mercury was absorbed through the skin, and in some instances a small amount through inhalation. They further found that calomel was readily absorbed and efficient. They recommend the following formula for calomel inunction: lanolin, grains 15 (1.), adipis benzoinati, grains 30 (2.), hydrargyri chloridi mitis, grains 45 (3.), this amount to be employed at each treatment. Cole[3] found calomel inunctions inferior to ordinary blue ointment from a clinical standpoint. The latter preparation is the one most largely employed. From ½ to 1 dram (2.–4.) may be rubbed into the skin at night before retiring and the area cleansed in the morning by washing. Thorough cleansing of the area with soap and water followed by sponging with an antiseptic solution before the application of the ointment, lessens the tendency to the development of a dermatitis. Continuous applications to a single portion of the skin are likely to induce such an inflammation, and it is, therefore, wise to select on successive evenings a fresh portion of the integument for the local application, preferably that where the epidermis is relatively thin, as, for example, the flexor aspects of the joints, the inner faces of the thighs, the sides of the chest, and the loins; but surfaces where there is a growth of hair should be avoided. The ointment in some cases may be well rubbed into the soles of the feet, which have previously been soaked in warm water, after which the stockings may be drawn over the feet for the night. When applied by an attendant, the back is the region usually selected. Welander[4] advises spreading the ointment on cotton, wool or linen, and wearing it over a limb or over the surface of the chest. Should local irritative effects be produced, these subside readily, as a rule, after suspending the treatment and using a warm alkaline solution, followed by a bland dusting-powder. In the case of patients with unusually sensitive skins, the ointment may be diluted with lanolin, lard, or olive oil; and to make the inunction less disagreeable it may be scented with lavender, rosemary or bergamot. Inunctions may be repeated daily for thirty days or longer, after which a complete rest may be had for several weeks, when their employment for another similar period may be begun. In certain cases, after one period, they are applied at weekly intervals for some time, then a more energetic period is employed. At any time that a relapse or recurrence happens, they are used more energetically. As the administration of mercury by inunction is, when well done, most efficient, it should be used in all practicable cases where this drug has been selected. Its quantity is to be carefully adjusted to fit the exigencies of the case, and it may be practised nightly, weekly, or less frequently so long as there is a possibility of relapse. It has recently been suggested by Schamberg that sufficient mercury is absorbed when inunctions are given three times weekly, and that by this method a greater amount of mercury can be given with safety than by other means.

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[1] Wile and Elliott, Jour. Cut. Dis., xxxv, p. 594; "Further studies on the mode of absorption of mercury in the inunction treatment of syphilis." Reference in this to a previous article on the same subject.

[2] Trans. Sec. on Derm., Amer. Med. Assoc., 1917, p. 208; "Experimental studies of the mode of absorption of mercury when applied by inunction."

[3] Ibid., 1919, p. 72; "A clinical study on the use of calomel inunctions."

[4] Archiv, 1897, xl, p. 257.

Fumigation.—One of the most effective methods of administering mercury is by fumigation in a vapor bath. It never was used extensively, but there were some who employed this means only of administering mercury. It had its chief value in the cases that were more or less generalized, and others which were uninfluenced by other methods of mercurial treatment. It also was employed when patients could only remain under the direction of the physician for a short time, for the reason that a large amount of treatment could be administered in a short period in this way. From ½ to 1 dram (2.–4.) of calomel, metallic mercury, the bisulphid, the black oxid, or hydrargyrum cum creta may be employed for each bath. It is common to order 1 scruple to 1 dram (0.33–4.) each of calomel and cinnabar. The patient is stripped of his clothing and seated in a chair, the patient and chair being completely enveloped in blankets, which are closely fastened at the neck of the bather. Beneath the chair is an alcohol lamp, surmounted by a metallic vessel containing water in ebullition, the hot vapor of which in a few moments induces copious perspiration. When this result is obtained the lamp is brought beneath a metal plate containing the substance to be volatilized. The patient remains exposed to the vapor about ten minutes after this process of sublimation is finished, and retires at once to bed without cleansing the skin, the fumigation preferably being conducted before retiring at night. In the morning a bath may be taken for the purpose of cleanliness. A Schering or other fumigating lamp may be used for the generation of vapor.

Subcutaneous and Intramuscular Injection.— This method, which was first advocated by Scarenzio[1] in 1864, and popularized by Lewin,[2] has been extended largely since its acceptance as a scientific procedure. It is efficient and speedy and is now the method of choice of an increasing number of authorities. In common with some of the other methods employed, injection provides for the exclusion of the medicament from the gastro-intestinal tract, and accomplishes the desired result with a minimum and exactly mensurable dosage. The objection to its systematic employment outside of hospitals is the need of a physician to administer the dose. The injection of mercury into the deep muscular tissue (the gluteus in its thickest part, with the muscle wholly relaxed; the trapezius above the upper scapular angle, with equal lack of tension), as well as when practised more strictly hypodermatically, requires all antiseptic precautions both as to the point where the needle is inserted and as to the instrument itself. These injections occasionally have proved fatal (calomel, gray oil); serious disturbance has followed in several instances from visceral troubles; and the attacks of syncope which result in certain cases from these injections have presented alarming and even dangerous features.

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[1] Trans. Internat. Derm. Cong., London, p. 376.

[2] Die Behandlung der Syphilis mit subcutaner Sublimatinjektion. Berlin, 1869.

The technic of mercurial injections is of importance. A syringe of a capacity of not less than 2 c.c., constructed wholly of glass and armed with iridoplatinum needles, is used. The operation is conducted with strict aseptic precautions. The patient, when the intramuscular injection is being given, preferably lies prone, though other postures are advised. The needle is plunged vertically into the deep part selected —post-trochanteric fossa (Smirnoff); intersection of a horizontal line two finger-breadths above the great trochanter and of a vertical line separating the inner third of the buttock from the outer two-thirds (Galliot); or the middle of a line connecting the anterior-superior spinous process and the base of the coccyx.

Among the soluble salts of mercury employed for injection, all of which are best administered intramuscularly. the most popular and efficient are the bichlorid, the biniodid, and the benzoate. Corrosive sublimate may be administered in dosage of $\frac{1}{12}$ (0.005), $\frac{1}{8}$ (0.008), $\frac{1}{4}$ (0.016) grain or more daily, or preferably every second day, in periods of about twenty injections each. Many formulæ have been suggested, for example the following of Staub, the result of the experimental work of Hepp:

℞—Hydrarg. chlorid. corros.,	gr. xviij;	1	20	
Ammon. chlorid.,	gr. xviij;	1	20	
Sod. chlorid.,	ʒj;	4		
Aq. dest.,	f℥iv;	120		M.

Dissolve, filter, and add the white of one egg in distilled water sufficient to make ℥iv (120.). 15 minims of the solution contain about $\frac{1}{12}$ of a grain (0.005) of the sublimate.

Lewin's formula is as follows:

℞—Hydrarg. chlorid.,	gr. xxiv;	1·5
Sodii corros. chlorid.,	gr. xvj;	1
Aq. dest.,	℥iiijss;	100

Fifteen minims (1.) or more may be employed.

A simple and convenient method of employing the bichlorid of mercury is to make a 1 per cent. solution of the salt in normal salt solution. Fifteen minims of this are used in the first, 20 in the second and subsequently 25 (or more) at each injection, the last dosage representing ¼ of a grain of the salt.

Other soluble salts employed are the succinamide, cyanid, oxycyanid, cacodylate, iodo-cacodylate, peptonate, and lactate.

Gottheil[1] recommends the following formulæ:

℞—Hydrarg. iodidi rubri,	gr. j;	0 06
Sodii iodidi,	gr. j;	0 06
Aq. dest.,	ʒiv;	15

℞—Hydrarg. benzoatis,	gr. j;	0 06
Sodii chloridi,	gr. j;	0 06
Aq. dest.,	ʒiv;	15

Of the first of the above, 10 minims may be administered at each injection, and of the latter 10 or more may also be employed.

Fournier[2] says of the biniodid that it is an active and well-tolerated salt, but is inferior in its therapeutic results to calomel or gray oil.

Of the insoluble salts of mercury, the preparations used most extensively are calomel, gray oil, and salicylate.

Metallic mercury was introduced as an intramuscular injection by Lang, of Vienna, and consisted of mercury in a finely subdivided form suspended in liquid fat (oleum cinereum), which contained 40 per cent. of the metal. Lang's modified formula[3] consists of metallic mercury, 2 parts; sterilized anhydrous lanolin, 1 part; and sterilized liquid paraffin, 1 part. Of this preparation each dose should consist of ⅔ of a grain of mercury. A formula indorsed by Lambkin, which is claimed to be painless, consists of:

℞—Hydrarg. pur.,	ʒijss;	10
Creosote absolut.,	ʒijss;	10
Acid. camphoris,	ʒijss;	10
Palmitin, q. s. ad.	℥iijss;	100

Ten minims represent 1 grain of metallic mercury.

All metallic mercurial preparations are warmed before using.

Calomel is administered in suspension in vaseline oil, olive oil, mucilage and water, or glycerin and water, and is given in 1-grain dosage. It may be used with creosote and camphor, as above suggested for metallic mercury, and is stated to be less painful.

The insoluble salicylate of mercury, while, according to many authorities, less efficient than metallic mercury or calomel, is extensively employed and may be prepared as follows:

℞—Hydrarg. salicylat.,	gr. xv–xxiv;	1 50
Mucil. acac.,	gr. viij;	533
Aq. dest.,	f℥vss;	165 M.

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[1] Hare, Modern Treatment, 1911, ii, p. 115.

[2] Cited by Lambkin, A System of Syphilis. Power and Murphy, 1908, ii, p. 236.

[3] Cited by Lambkin, loc. cit.

A Pravaz syringeful of the above is the usual dose. A popular and simple method is to have it prepared as a suspension in liquid albolene.[1] A pure preparation of this drug is thoroughly mixed in a mortar with liquid albolene, using all necessary precautions as to cleanliness, and is placed in one-ounce, wide-mouth bottles, the whole then being sterilized, after which it may be kept for some time and used as necessary. Either a 10 or 20 per cent. suspension is prepared, of which a quantity representing ½ to 2 grains is administered. Ten minims of a 10 per cent. suspension represents 1 grain of the salt.

The technic for its administration may be as follows: A sterile glass syringe of the capacity of 2 c.c. (or 30 minims), with an irido-platinum needle of good caliber and at least 1¼ in. length, is necessary. The injection may be given with the patient standing or in the recumbent position. The area on the buttock above outlined is thoroughly cleansed, and the needle after sterilization is filled with sterile liquid albolene and plunged into the muscular tissue. The syringe is then detached, leaving the needle *in situ* for sixty or more seconds. At the end of this period, if no bleeding occurs through the needle, the syringe, containing the proper amount of the suspension, is reattached and the contents injected. As injection of the material into a vein might be followed by unpleasant consequences, the above technic is necessary. If bleeding occurs through the needle, it indicates the presence of the latter in a vein, in which case it must be withdrawn and a new site selected. The emulsion is to be thoroughly shaken during the waiting period immediately preceding its injection, to insure a perfect suspension. The injection is practically painless at the time, but is followed for a day or two by a feeling suggestive of a bruise. Slight induration may follow if some of the injected material escapes into the subcutaneous tissue. As a rule, this can be avoided. The injections are employed usually once weekly and on alternate sides of the body.

Intravenous injections of mercury have been employed, but not at all generally. The salts thus used are the bichlorid, the biniodid, the cyanid, and the benzoate. The method was introduced in 1893 by Bacelli. Pernet[2] states that the method is especially effective in syphilitic diseases of the eye. Lydston[3] believes that in forty-eight hours a patient may be brought completely under the influence of mercury by intravenous injections (15 to 25 minims of a 2 per cent. solution of the bichlorid of mercury). Ten cases are reported improved. The median basilic or median cephalic vein in the forearm was selected, care being taken to place the needle fully within the lumen of the vein and to remove the tourniquet before the discharge of the mercurial solution from the syringe. By this method a rapid effect is produced, but its disadvantages outweigh the advantages, according to most authorities. Heidingsfeld[4] recommends the intravenous injection of oxycyanid of mercury in arsphenamin relapses, in dilution of 1 : 1000, and in doses of 0.02 grain.

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[1] Gottheil, Jour. Amer. Med. Assoc., 1907, xlix, p. 365: Ten Years' Experience in the Treatment of Syphilis by Intramuscular Injections of Insoluble Mercurials.

[2] Brit. Med. Jour., November 30, 1897.

[3] Jour. Amer. Med. Assoc., 1907, xlix, p. 1662.

[4] Amer. Jour. Urology, 1912, viii, No. 8.

Ptyalism, stomatitis, fetor of the breath, or a fungous condition of the gums, with inappetence and other characteristic symptoms of the ill-effects of mercury, including all grades of gastro-intestinal disturbance, are seen rarely in modern practice, and they should never occur in a properly regulated mercurial course. When they are produced, the tongue is usually tumid, and exhibits at its lateral borders the imprints of the inner faces of the molar teeth. Its surface is also covered in various degrees with a thin, dirty-grayish coat; and the odor of the breath is peculiarly offensive, being often noticeable at a distance of several feet from the patient. In such cases the food should be liquid and nutritious, both hot and cold drinks should be scrupulously avoided, and the mouth should frequently be cleansed with washes containing dilute liquor sodii chlorinata, potassium chlorate, or a weak solution of phenol. In particularly severe cases, potassium chlorate may be employed to the extent of 1 dram (4.) daily. The compressed tablets of this salt, each containing 5 grains (0.33), may be slowly dissolved in the mouth. The mercurial is to be suspended in all such cases, and iced water is to be interdicted, as gangrene has followed its use in a few instances. In milder forms tincture of myrrh and cinchona, diluted with sweetened water, or honey and water, will be sufficient for local medication of the mouth. Iodoform in pill form, each pill containing 1 grain, may be employed three times daily, allowing the pill to remain and slowly dissolve in the mouth before swallowing; or iodized phenol, well diluted (15 drops in a wineglassful of water), may be used as a mouth wash.

THE IODIDS.—Potassium and sodium iodid are the preparations commonly employed. Iodid of ammonium, rubidium and strontium are less effective. Iodipin (Merck) and lipiodol (Lafay) are iodinized oils and are employed both by mouth and by injection. The iodids possess some value, without question, in every stage of syphilis. Their value lies chiefly in the late manifestations of the disease, and their use should be largely restricted to this period. While they apparently have no specific effect on the organism producing the disease, their value in removing indurations and nodules and in the healing of gummatous and other ulcers is incontestable. The value of the drug is enhanced by the administration at the same time of a mercurial, such combination being known as the "mixed treatment." Potassium iodid may be given in doses of from 5 grains (0.33) to 1-2 drams (4. 8.), well diluted with water (a gobletful, preferably), three or four times daily, one hour after eating. The larger dose should, as a rule, be reached gradually. That small dosage will frequently accomplish the result was early brought out by Hutchinson, and later emphasized by Hartzell.[1] A common method of administering the drug is to begin with 5 grains and increase 1 grain at each dose until the desired end is attained.

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[1] Therapeutic Gazette, May 16, 1898.

At times large dosage may be necessary to accomplish the result in obstinate cases. Not infrequently its administration is better tolerated if it be given in a glassful of milk. When the desired result has been accomplished, viz., the removal of nodular deposits or healing of ulcers, the dosage should be largely reduced, and at a subsequent period the drug stopped altogether and the treatment carried on with mercury.

Symptoms of iodism other than the production of cutaneous lesions, such as coryza, edema of the eyelids, abdominal tension and tenderness, and faucial irritation, are often the result of the first few doses of iodin ingested, and these symptoms may bear no relation to the size of the dose. In certain cases 1 or 2 grains (0.066–0.133) will be sufficient to produce the most disagreeable effects, which, if they occur before the remedy be suspended, may not return with even the largest doses. In a few cases potassium iodid produces violent toxic effects in any dose, owing to exceptional idiosyncrasy.

Sodium iodid may be used in the same manner as potassium iodid, and appears to have less irritating effects on the stomach. Ammonium and lithium iodid possess also, without question, some influence over the disease, but they are in most cases less efficacious than the potassium and sodium salts. When given in combination with the biniodid of mercury, the syrup of licorice may be used as a part of the vehicle, which makes a more pleasant preparation to take. For example, the following formula may be employed:

℞—Hydrarg. iodid. rubri.,		gr. ss–ij;	0.033–0.133
Potassii iodid.,		ʒij–℥j;	8–30
Syrup glycyrrhiz.,		℥ij;	60
Aq. dest.,	q. s. ad.	℥iv;	120

The common method of administering potassium iodid and the bichlorid of mercury is in combination with the syrup of sarsaparilla, one formula being as follows:

℞—Hydrarg. chlorid. corros.,		gr. iij;	20
Potassium iodid.,		ʒv;	20
Aqua,		ʒv;	20
Syrup of sarsp.,	q. s. ad.	℥viij;	240

Potassium iodid alone or in combination with mercury by the so-called "mixed method" is of value in the treatment of nodular, gummatous or ulcerative lesions and also in the nervous and visceral complications. It may be used early in the cases that assume the so-called "galloping" type and in cases in which the late lesions develop unusually early. It also is of value in relieving headache and other nervous symptoms that sometimes occur in the early stages.

Local Treatment.[1]—The local treatment of the initial sclerosis of syphilis by complete excision, lauded by Auspitz, has been practised (since the date of his paper in 1879) by Kölliker, Zeissl, Leloir, Chadznski, Mauriac, and others. The result has proved that, as a rule, such operative interference furnishes no bar to constitutional infection. Simultaneous extirpation of all lymphatic glands in the vicinity of an initial sclerosis, with ablation of the latter and a mass of tissue about it, has repeatedly proved unavailing to prevent the occurrence of systemic infection. More recently, excision has again been advocated by Hoffmann, Neisser, Hallopeau, and others. It is suggested by Fordyce[1] that when situated on the foreskin the chancre may be advantageously removed; but the major portion of observers believe that no special benefit is derived from its excision, as the infection very soon passes beyond the limits of the local lesion. Chancres should not be destroyed by caustic agents of any character, as they are likely to induce either irritative or inflammatory effects which may be followed by more intense induration. Ointments, as a rule, are objectionable except where crusted lesions are present. In these a calomel ointment is serviceable. Cleanliness with soap and water is of chief importance. Thorough cleansing with a solution of boric acid or a 1 : 2000 solution of mercuric chlorid, followed by dusting the parts with a powder, such as calomel and europhen, iodol, or boric acid, is advised, or the lesions may be dressed with a piece of soft lint saturated with lotio nigra, or an alcoholic lotion containing tannin and phenol, or boric acid.

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[1] *Cf.* Section devoted to Arsphenamin.

The phagedenic or gangrenous lesions that formerly occurred are rarely seen since the introduction of arsphenamin. When these do occur they are best treated by a continuous hot-water bath. Secreting condylomata and vegetations require bathing with soap and water, and when the odor of the secretion is offensive formalin, boric acid, phenol, or thymol may be employed as a lotion. After cleansing or disinfecting the parts with a lotion, it is sometimes necessary to use a solution of silver nitrate, 10 to 20 grains (0.66–1.33) to the ounce (30.), after which they are dusted with a powder such as above mentioned. Crusted and ulcerated areas are to be cleansed with an antiseptic solution and either dusted afterward with the powders or covered with an ointment containing ammoniated mercury, or the yellow oxid of mercury, or a compound iodin ointment. Bandaging the lower extremities, when ulcers occur in this situation, facilitates healing.

The removal of syphilodermata that occur on the face is hastened by the local application of an ammoniated mercurial ointment in a strength of 3 to 8 per cent. Mercurial ointment, 1 to 2 drams (4.–8.) to the ounce (30.) of cold cream salve or vaseline, red oxid, from 2 to 4 grains (0.133–0.266), to the ounce (30.) may be similarly used.

The local treatment of syphilitic lesions of the mucous surfaces is both hygienic and medicinal. Catarrhal conditions of adjacent mucous surfaces (oral and nasal cavities, vagina) require attention. The parts should be kept free from all irritation (tobacco in all forms, iced and hot articles of food and drink, condiments, acetous and alcoholic fluids in the mouth; coitus and irritating injections of the vagina;

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[1] Trans. Sec. on Derm., Amer. Med. Assoc., 1912, p. 114.

napkins that have been soiled over the ano-genital regions of infants). Locally, the silver nitrate crayon is effective in the management of moist patches, applied once daily or every second or third day. Occasionally, stronger caustics are required, such as mercuric nitrate or nitric acid. Mouth washes containing potassium chlorate, myrrh, and honey; 15 to 20 drops in water of Bellamy's iodized phenol; milk of magnesia; very dilute lotions of tincture of ferric chlorid; or dilute hydrochloric acid, a teaspoonful to a pint of sweetened water; and phenol washes, are required in different cases. In very great soreness and tenderness of the mouth, only the blandest applications are tolerated, such as thin flaxseed tea, oatmeal gruel as a wash, and gum-acacia water. A few formulæ are appended:

℞—Potass. chlorat.,		ʒj;		4	
Mel. depurat.,					
Tinct. myrrh.,	āā	℥ss;		15	
Aq. dest.,	ad.	℥vj;	ad.	180	M.

Sig.—A teaspoonful in water as a wash for the mouth and throat.

℞—Tinct. iodin.,		ʒss;		2	
Phenolis,		ʒj;		4	
Glycerin.,				6	
Alcoholis,		ʒij;		8	
Aq. dest.,	ad.	f℥j;	ad.	30	M.

Sig.—Fifteen to 20 drops in water as a lotion for the mouth.

℞—Potass. chlorat.,		ʒj;		4	
Aq. menth. piperit.,	āā	℥vj;	āā	180	M.

Sig.—Gargle and wash for the mouth; to be used slightly diluted.

ARSPHENAMIN.—(Salvarsan, "606," *Dioxydiamido-arseno-benzene-dihydro-chlorid.* $C_{12}H_{12}N_2O_2As_2HCl + H_2O$).[1]—Arsphenamin is a yellow powder, soluble in water, and has a strongly acid reaction. It is furnished in ampules, containing quantities ranging from 0.1 gram to 3.0 grams. Exposure to air causes deterioration and the formation of toxic substances, so that each ampule should be tested before use to determine possible defects. Arsphenamin is very extensively used in the treatment of syphilis today and largely recognized as a potent agent in combating the disease. Uniformity of opinion as to the method of its employment does not exist. For the relief of the clinical manifestations of the disease it has shown unusual capabilities, but the dangers attending its use must be borne in mind, though these have been largely eliminated through improvement in technic and a careful study of the causes of these untoward effects.

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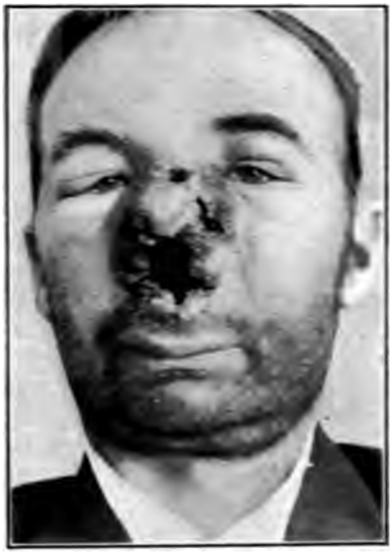
[1] For reports expressing views on the use, dangers, and efficiency, of salvarsan, see Ehrlich, Hata, Chemotherapy (translated by Newbold, revised by Felkin); MacKee Med. Jour., New York, October 21, 1911: A Comparison of the Results Obtained by the Intravenous Administration of Acid and Alkaline Solutions of Salvarsan. Brocq, Annales, December, 1912, iii, p. 669: Present Use of Salvarsan. Swift, Trans. Amer. Med. Assoc., Sec. on Derm., 1912, p. 101: Anaphylaxis to Salvarsan. Fordyce, ibid., p. 114: The Administration of Salvarsan in Syphilis. Post, ibid., p. 141: The Proper Places of Mercury and Salvarsan in the Treatment of Syphilis. Howard Fox, ibid., p. 154: The Relative Value of Mercury and Salvarsan from a Serologic Point of View; Nichols (Jour. Amer. Med. Assoc., 1912, lviii, p. 603) The Present Status of Salvarsan Therapy in Syphilis. Keyes, Jour. Cut. Dis., 1912, xxx, p. 420: Notes on Salvarsan. Finger, Wein. klin. Wochenschr., 1913, No. 15: Mercury and Salvarsan. Pusey, Amer. Jour. Med. Sc., 1913, cxlvi, p. 497: The Present Situation in Syphilis. Leredde and Jamin, Bull. Soc. Franc. Derm., 1914, p. 91; ibid., 1914, p. 267. Dangers of salvarsan disappear after first injections. Fordyce, Jour. Cut. Dis., 1914, xxxii, p. 1. Ormsby and Mitchell, Jour. Amer. Med. Assoc., 1916, lxvi, p. 867. Ormsby, Ibid., 1917, lxviii, p. 949. Stokes, Archiv. of Derm. and Syph., 1920, ii, p. 303.

The drug has arsenic as its active base, and was elaborated through scientific research by Ehrlich and his associates in their endeavor to obtain a remedial agent fulfilling the ideal "*therapia sterilisans magna.*" Although the ideal was not achieved with respect to syphilis, nevertheless the drug assumed a position of importance in the treatment of the disease.

Methods of Administration.—The method of subcutaneous administration of arsphenamin which was largely employed early has been entirely abandoned, owing to its accompanying disadvantages. Among these was pain of more or less serious character at the time of its administration, and it not infrequently was followed by the formation of long-continued indurations and abscesses. Intramuscular injections with the drug in solution, or as an emulsion in an oil, were more successful, were attended with fewer unpleasant after-effects, and were used largely for some time. Some authorities still use this form of injection in certain instances, and believe the method to be safer than the intravenous method, which is now being employed almost exclusively.

FIG. 311



Malignant lues ending fatally, not controlled by arsphenamin. (Morrow.)

Intramuscular Method.—Arsphenamin may be given in solution intramuscularly but is preferably incorporated in an oil, several of which have been recommended. A simple and convenient method is to place the required dosage of arsphenamin in a small, sterile glass mortar and add to this 2 c.c. of iodopin or other oil. This is thoroughly ground with a pestle until a perfectly smooth emulsion is made. The emulsion is then drawn up into a glass syringe, and one-half of the contents injected into each side of the gluteal region. Before the injection, the skin should be cleansed and the area painted with tincture of iodin. An irido-platinum needle, No. 19, one and one-half inches in length, should be employed. The technic outlined for giving insoluble salts of mercury should be followed here, in order to avoid getting arsphenamin into a vein. No immediate reaction occurs from this method, but both local and systemic symptoms of varying grades are sometimes noted from the third to the sixth day or from the eighth to eleventh day—local swelling and induration, with pain, tenderness, and elevation of the temperature, and at times a chill, nausea and vomiting. All of these subside soon without sequels except local induration, which also disappears in time by absorption.

Intravenous Administration.—Freshly distilled water is essential for the solution of the drug, as water that has been kept any length of time becomes contaminated, and even though sterilized at the time of use the products of dead microörganisms produce unpleasant symptoms in the presence of arsphenamin. Freshly prepared sodium hydroxid solution is also necessary, as well as sterile 0.5 per cent. saline solution, prepared with chemically pure sodium chlorid and freshly distilled water. A narrow-necked, graduated, glass-stoppered, sterile cylinder measure of 300 c.c. capacity is used, into which 30 to 40 c.c. of sterile distilled water are placed, after which 0.5 arsphenamin is added and vigorously shaken until it goes into solution. To this solution approximately 19 drops of 15 per cent. sodium hydroxid solution is added, drop by drop. A precipitate forms, which is gradually redissolved. A slight excess of sodium hydroxid solution is not detrimental but, in fact, is of service, as has recently been demonstrated by Schamberg.

This produces a clear yellow solution to which sufficient sterile 0.85 per cent. saline solution, or sterile water, is added to make a total of 125 c.c. Each 25 c.c. of this solution contains 0.1 gram of arsphenamin. The disodium salt produced by the addition of ⅓ more sodium hydroxid than is required for neutralization is less likely to be precipitated in the blood-stream, but is more hemolytic and is rather more destructive to the vessels. Kolmer and Yagle[1] find that the excess of sodium hydroxid should not exceed ⅕ the amount required for neutralization in order to lessen hemolysis.

Many varieties of apparatus for injecting arsphenamin intravenously have been devised. A simple method is to use a Whitall-Tatum Company percolator, small size, Oldberg type, capacity 250 c.c., to which 3½ feet of rubber tubing is attached. The latter should be heavy and with a small lumen. A small window is placed 2 inches from one end by inserting a glass tube, and to the other end is attached a rubber stopper to fit the percolator. A needle of the size recommended for intramuscular injections may be employed, but this should not have a long bevelled point. At the time of injection the patient should be in a reclining position. After the skin of the arm at the elbow has been thoroughly cleansed a tourniquet is applied to the middle of the arm, thus distending the veins. A small amount of saline solution or distilled water is placed in the percolator and allowed to fill the tube and needle. The latter is then inserted into the lumen of the vein and as soon as blood is seen in the window the tourniquet is removed and the solution allowed to flow in. When certain that the sterile water is flowing properly into the vein, the arsphenamin is placed in the percolator and allowed to pass through, following which another small amount of sterile water is used. It is entirely unnecessary to make incisions over the vein, as the needle can practically always be inserted in the manner above described. The gravity infusion method is strongly urged, rather than injection by means of a syringe, and the solution should be allowed to flow slowly into the vein. Arsphenamin should never be administered in concentrated nor acid solution.[1]

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[1] Jour. Amer. Med. Assoc., 1920, lxxiv, p. 643; Hemolytic Activity of Solutions of Arsphenamin and Neoarsphenamin.

Neoarsphenamin is a yellowish powder, readily soluble in water, the solution having a neutral reaction. It contains as the active constituent dioxydiamindo-arseno-benzene-mono-methane-sulphate of sodium ($C_{12}H_{11}O_2\ As_2N_2CH_2O\ SO\ Na$) with organic salts. It is furnished in glass ampules, each containing a single dose, ranging from 0.15 gram to 4.5 grams. Neoarsphenamin contains 21 per cent. of arsenic, while arsphenamin contains 31 per cent. In comparison, therefore, 0.9 gram of the former is equivalent to 0.6 of the latter.

According to Schamberg, twice as much neoarsphenamin is necessary to obtain the same clinical effects. Originally it was recommended to dissolve each 0.15 gram of the preparation in 25 c.c. of freshly distilled water for intravenous injection. Ravaut[2] reduced the amount of water to 10 c.c. for 0.9 gram, and has been followed by most other observers in the use of concentrated solutions. At the present time 0.9 gram is dissolved in from 5 to 20 c.c. of water.

Extended observation appears to prove arsphenamin to be more efficient than neoarsphenamin, but attended with slightly more dangerous possibilities. Whitehouse and Clark,[3] Nelson and Harris,[4] Capelli,[5] Schamberg[6] and many others, have demonstrated the superiority of arsphenamin over that of neoarsphenamin. On the other hand, several observers have found the latter preparation more efficient. The advantages of neoarsphenamin are its ease of preparation for immediate use, requiring merely freshly distilled water (cool) for making the solution. The small quantity of solution in which the drug may be introduced was of particular value in the recent war in the treatment of soldiers near the front. The injection of neoarsphenamin is followed less frequently by reactions. Experimentally Schamberg, Kolmer, Raiziss and Weiss demonstrated that it does not hemolyze the red cells *in vitro*, and they conclude that it produces less disturbance in the blood and tissues than arsphenamin. Furthermore, clinically it appears as efficient, as a rule. In a recent communication Schamberg,[1] after extended observation of the use of neoarsphenamin, states that it cannot be predicted which compound of the two will ultimately be accorded preference.

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[1] MacKee, Jour. Cut. Dis., 1912, xxx, p. 199.; Amer. Jour. Exp. Med., 1911, xiv, p. 497.

[2] Annales, April, 1913, p. 206

[3] Jour. Cut. Dis., 1913, xxxi, p. 633.

[4] Jour. Amer. Med. Assoc., December 26, 1914, p. 2277.

[5] Derm. Wchnschr., 1915, lxi, p. 907.

[6] Loc. cit.

Precautions.—A careful chemical and microscopic examination of the urine should precede each injection. Arsphenamin should be administered in small dosage in all cases exhibiting disease of the liver, kidney, heart, lungs, circulatory or nervous systems when not of syphilitic origin and avoided if these disorders are grave. Idiosyncrasy is a factor.

ARSPHENAMIN AND MERCURY.—The major portion of practitioners use a combination of arsphenamin and mercury. These agents are commonly employed in courses with intervals of rest. The interval between injections of arsphenamin has varied from one to fourteen days, the number of injections in each series from three to eight, and the number of series in a year usually three, but possibly more. A uniform method does not exist. A reference to the appended bibliography will illustrate the gradual evolution of this treatment. From the published results of various members of the Medical Corps, U. S. Army, the method described below was apparently efficient and is a fair example of that now employed.[2]

Three courses are given during the first year of early syphilis. Each course consists of six injections of arsphenamin at five- to seven-day intervals. The dosage averages one decigram to each thirty pounds of body weight, and the first injection consists of one-half the normal dose. In addition, twenty-four to thirty injections of one of the soluble salts of mercury, or nine or ten injections of one of the insoluble salts of mercury are given. This requires ten weeks. Then a rest of one month is allowed; if the complement-fixation test is positive at the end of this time the course is repeated; if negative, only the mercurial course is repeated. At the end of the second course a two months' rest is given, followed by a complement-fixation test and a third course, as above indicated. During the second year the Wassermann-negative cases are given two courses of mercury, with two rest periods each of four months. The Wassermann-positive cases are treated as directed in the first year, except that the length of time of the rest period is doubled. Guy[3] recorded good results in the treatment of four hundred patients with this method. In 1916 Pollitzer[1] described an efficient method of treatment founded on the theory of fractional sterilization as employed in the laboratory. This requires the administration of arsphenamin in as large dosage as is compatible with safety and repeated before the microörganisms not destroyed in the original treatment have had time to reproduce to any marked extent. He recommended in the chancre stage three full doses of arsphenamin given on three successive days, followed by twelve injections, each consisting of 2½ grains of the salicylate of mercury at weekly intervals. When this was employed before the appearance of a positive Wassermann it was sufficient to eradicate the disease. Later cases received three injections as above outlined, followed by eight injections of mercury, after which a rest of two months was taken and then the treatment repeated. After a second rest of two months a third period was given. This requires approximately one year. In cases of long standing before treatment is instituted, it has to be continued until a negative Wassermann is obtained, after which two courses are given, even though the reaction continues negative. After a year or more of constantly negative tests he advises a provocative treatment, followed by a spinal fluid examination.

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[1] Jour. Amer. Med. Assoc., 1919, lxxiii, p. 1883.

[2] *Cf.* Manual of Military Urology published by the American Red Cross for the American Expeditionary Force, 1918; France, Masson and Cie., Ed.; and Manual of the Venereal Diseases, 3d Ed., revised for civilian physicians. Printed by the American Medical Association for the U. S. Public Health Service.

[3] Jour. Cut. Dis., 1918, xxxvi, p. 441; "The Treatment of Syphilis at Camp Travis." Also see Idem. Archiv. of Derm. and Syph., 1920, ii, p. 53. Observations on the Treatment of Syphilis.

The method employed by the author[2] for a period of three years has been that outlined above, with the exception that the three injections are given on alternate instead of successive days. In a large number of cases so treated the result has been satisfactory and the untoward effects have been no more numerous than when the injections were given at five-, seven-, or ten-day intervals.

Irrespective of the method employed, treatment should be given at intervals for at least a year after the disease is apparently eradicated, as shown by absence of clinical signs and negative laboratory findings. At the termination of all treatment a spinal puncture should be performed and a provocative treatment of arsphenamin given, followed by the complement-fixation test. If everything proves negative a prolonged rest is permissible.

Reactions and Untoward Effects.[3]—In the early days of the use of arsphenamin these conditions arose much more frequently than later, when the technic of administration improved, and the conditions of the patient which were contributing factors became better understood.

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[1] Jour. Cut. Dis., 1916, xxxiv, p. 633: "The Principles of the Treatment of Syphilis."

[2] Jour. Amer. Med. Assoc., 1920, lxxv, p. 1. A Valuable Method of Employing Arsphenamin in Syphilis.

[3] Schamberg, Kolmer and Raiziss, Jour. Cut. Dis., 1917, xxxv, p. 286, Experimental and Clinical Studies of the Toxicity of Dioxydiamino-arsenobenzol Dichlorhydrate. Stokes, Jour. Amer. Med. Assoc., 1919, lxxii, p. 241; Atropin and Induced Antianaphylaxis. Schamberg, Kolmer, Raiziss and Weiss, Archiv of Derm. and Syph., 1920, n. s. i., p. 235; Laboratory and Chemical Studies Bearing on the Causes of the Reactions following Intravenous Injections of Arsphenamin and Neoarsphenamin (second communication). Raiziss and Proskouriakoff, Ibid., 1920, ii, p. 280; The Chemistry of Arsphenamin and its Relation to Toxicity. Roth, Ibid., p. 292; Some Salient Facts Regarding the Toxicity of Arsphenamin and Neoarsphenamin. Strickler, Munson, Sidlick and Strauss, Ibid., p. 692; A Clinical Study of the Reactions following the Intravenous Administration of Arsphenamin in Non syphilitic Persons.

The improvement accomplished in the manufacture of the drugs has also lessened these to a notable degree. The early reactions were largely due to impurities which have been reduced of late, thereby lessening the occurrence of these effects. It may be said that at present while these various conditions described below do occur, the proportion of patients so affected is small compared with the great numbers treated.

The reactions are classed as "early" and "delayed." The former occur during the first few hours after the injection and not infrequently begin during the administration of the drug, while the latter, or delayed reactions, occur after intervals varying from twenty-four hours to several days. The symptoms of the immediate reactions are flushing and edema of the face, suffusion and swelling of the eyes, dyspnea, and signs of fullness in the head and constriction at the throat, precordial distress, and at times cyanosis. The pulse, which is at first full and bounding, becomes rapid and weak and nausea and vomiting are not infrequent. A peculiar taste or odor is complained of and likened to ether or chloroform. These symptoms usually pass off within a few minutes and, as a rule, are not dangerous. An injection of 10 minims of a 1 : 1000 solution of adrenalin, which may be repeated if necessary, is of value in overcoming this reaction. According to Schamberg, these early vasomotor symptoms are due to traces of impurity in the drug, which induce paresis of the bloodvessels characterized by dilatation and not infrequently by leakage of serum into the tissue. They may be regarded as vasoparetic in character.

At times, in from one to four hours after the injection, a chill may occur followed by a rise in temperature of three to five degrees, accompanied by nausea, vomiting, diarrhea, headache and pains in the back and legs, which also pass off after some hours. During the first twenty-four hours various cutaneous lesions occur, such as urticaria, herpes, erythema—both simple and multiform in type, all of which are commonly mild in character and soon disappear.

Herxheimer Reaction.—This consists of activation of the syphilitic lesions present, and is only of importance when lesions exist in the nervous structures or bloodvessels, where their renewed activity might produce harmful results. The cutaneous lesions become more marked temporarily, after which they subside. The reaction occurs within the first twenty-four hours and is thought to be due to the action of the drug on the parasite, being somewhat similar to that seen in lesions of tuberculosis following tuberculin injections.

Delayed Reactions.—These occur in from twenty-four hours to several days or longer, and are largely gastro-intestinal in type. They may occur alone or be accompanied by a cutaneous exanthem, such as a scarlatiniform or morbilliform erythema, or icterus. When the latter occurs the liver has been involved by the direct action of the arsenic. In rare instances a general exfoliative dermatitis occurs, which may last for several weeks or several months and occasionally is grave. In a recent case of this type seen by the author there was a marked reduction in the number of leukocytes and an increase in the eosinophiles. This blood picture was noted by Moore and Foley[1] in a fatal case accompanied by cutaneous manifestations not of this type, but in which marked changes were found in the bone-marrow postmortem. Renal disturbances occur and are exhibited by albuminuria, which is usually not serious, and occasionally by suppression of the urine. In rare instances hemorrhagic encephalitis occurs, the symptoms of which begin two and three days after the injection. In these there is severe headache, and mental disturbance followed by coma and death, as a rule.

Neurorecurrences indicate involvement of the brain, cord or spinal nerves with syphilis due to insufficient treatment. They commonly involve the optic or auditory nerve, inducing derangements of vision or hearing. These symptoms are not due to arsphenamin, although they were emphasized in the literature after the employment of this drug. Neurorecurrences have been largely eliminated since more intensive treatment has been employed.

SULFARSENOL has recently been employed successfully in the treatment of syphilis. Lévy-Bing and Gerbay[2] recorded their experience in two hundred cases. The drug is dissolved in sterilized distilled water and given as an intramuscular injection. No reactions have followed its employment and the therapeutic results are said to be excellent.

The Treatment of Congenital Syphilis is mainly that of the acquired form, with such modifications as are required by the tender age of the subject of the disease and by the special character of the eruptive and other symptoms in the infant or child. The mother, who is demonstrably the subject of the disease, requires antisyphilitic treatment during any pregnancy where there is possibility of taint of the product of conception. This is especially important in pregnancies succeeding those terminating either in abortion or in the birth of a syphilitic child. The syphilitic child, when the disease is inherited, should be kept at the breast of the mother and not suckled by any other woman. All syphilitic infants require special provision for their nutrition; cod-liver oil is generally indicated. Inunction, using 10 to 30 grains of mercurial ointment, is to be practised by anointing the swathing-band with a strong or modified mercurial salve, the motions of the child being in general sufficient to insure a proper medication by introduction of the medicament. As the skin of the abdominal surface in these young patients is generally sensitive, care should be taken to suspend the application of the unguent and to apply a dusting-powder until any resulting dermatitis is relieved.

Internally, calomel or the gray powder, $\frac{1}{10}$ of a grain to 1 grain (0.006–0.06), may be applied to the tongue after trituration with the sugar of milk or given in milk. Potassium or sodium iodid is indicated in the cases presenting nodular, gummatous or ulcerative lesions. The potassium salt may be given in doses of from $\frac{1}{10}$ to 2 or 4 grains (0.006 to 0.133–0.266), administered in solution three times daily, or oftener when required. Iron is indicated generally, and in particular the iodid of iron, which may be given in the form of syrup, 2 to 5 drops in solution. The dosage is to be varied with the age and vigor of the child. Lesions of the mucous surfaces (mouth, anus, nares) require special hygienic care, and the use of lotions of boric acid, formalin, chlorinated soda, and, especially soap and water, is desirable. These should be followed often, particularly about the ano-genital region, with the application of dusting-powders. The eruptive symptoms in inherited syphilis are to be treated like those in the acquired disease, due care being taken to protect the tender skin from irritation. The tars and stronger mercurial salves should not be employed over the skins of very young infants.

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[1] Archives of Derm. and Syph., 1920, n. s. i, p. 25; "Serious Reactions from the Salvarsan and Diarsenol Brands of Arsphenamin."

[2] Annals Mal. Vén., January, 1920, p. 15; Intramuscular Injections of Sulfarsenol.

Arsphenamin and neoarsphenamin[1] have been largely used in congenital syphilis with good results. As in the adult form, an absolute technic has not been developed. The combination of a mercurial has been proved to be most efficacious. The technic also is similar to that employed in the adult, and usually comprises several series of arsphenamin injections, given at four- to seven-day intervals and six to ten injections at each period, the dosage being about 0.01 gram of arsphenamin per kilo of body-weight. Many of these cases require prolonged treatment. Inunctions with mercury are commonly used with arsphenamin. Fordyce and Rosen[2] have had excellent results by using neoarsphenamin and mercury by intramuscular injection. These drugs are given in courses of six to eight injections at weekly intervals. Mercuric chlorid is employed in palmatin and administered in doses of $\frac{1}{10}$ to $\frac{1}{8}$ grain. Neoarsphenamin is dissolved in freshly distilled water and administered in the following doses: 0.075 gram for infants three to eight weeks; 0.1 gram from two to six months; 0.15 gram from six months to one year, and 0.15 gram to 0.2 gram from one to two years. A special needle has been devised for these injections—no untoward results have followed.

Prophylaxis.—By a series of carefully controlled experiments, Metchnikoff and Roux demonstrated the fact that syphilis may be aborted if a strong calomel ointment (calomel 20, lanolin 40 parts) be thoroughly applied to the site of inoculation within a period of six hours. The value of this measure in the prevention of syphilis has been abundantly demonstrated in the medical service of the armies of Great Britain, the United States, and other countries.

Prognosis.[3]—The duration and probable outcome of the various syphilodermata have been suggested in connection with the description of the lesions. As a rule, but little difficulty is experienced in clearing up the clinical manifestations. The former exceptions to this rule, which included particularly mucous-membrane lesions, those involving the palms and soles, and rarely other lesions, are now largely overcome by the use of arsphenamin. It may be said, therefore, that with judicious treatment on the part of the physician and with complete coöperation on the part of the patient, the disorder, so far as cutaneous symptoms are concerned, may be with rare exceptions, entirely overcome.

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[1] *Cf.* Boardman, Jour. Cut. Dis., August, 1914, xxxii, p. 545; Syphilitic Heredity and Congenital Syphilis, for discussion of arsphenamin treatment and references.

[2] Jour. Amer. Med. Assoc., 1920, lxxv, p. 1385; A Method of Treating Congenital Syphilis—*Cf.* this article for technic and details.

[3] *Cf.* Keyes, Jour. Cut. Dis., 1910, xxviii, p. 449: Some Elements in the Prognosis of Acquired Syphilis. Pollitzer, Jour. Amer. Med. Assoc., 1920, lxxiv, p. 775; General Prognosis of Syphilis in the Light of Recent Progress.

The prognosis relative to ultimate cure of the general disease in acquired syphilis may also be said to be good. The earlier treatment is instituted the better the prognosis. In cases where efficient treatment is given before generalization occurs and before the Wassermann test has become positive the disease is usually entirely eradicated. The proportion of cases in which a Wassermann reaction remains positive is not known, nor is the significance of such a condition entirely understood. In the larger proportion of cases the patient may be placed in a condition in which he is not liable to relapses, and is unable to communicate the disease to others or transmit it to his offspring.

Congenital syphilis, on the other hand, is more grave, and, as stated in the description of symptoms, the earlier the manifestations appear after birth, the more grave the prognosis. Many children, however, with proper treatment and hygienic care, make complete recoveries, with no recurrences.

LEPRA.[1]

Synonyms.—Leprosy, Satyriasis, Elephantiasis Græcorum, Leontiasis, Lepra Arabum. Fr., *Lèpre, Ladrerie;* Ger., *Aussatz;* Norweg., *Spedalskhed.*

[1] Danielssen and Boeck, Traitè de la Spedalskhed, etc. (with atlas), Paris, 1848. A. Hansen, Archiv, 1871; Cong. méd. de Soc. méd. de Copenhagen, 1884. Van Dyke Carter, Leprosy, etc., 1874. Jones, New Orleans Med. and Surg. Jour., March, 1878. Solomon, Trans. Louisiana State Med. Assoc., 1879. Bemiss, New Orleans Med. and Surg. Jour., 1880, n. s., vii, p. 923. Barnes, Arch. of Med., December, 1881, vi, p. 201. Leloir, Traitè de Lèpre (planches), Paris, 1886. Morrow, Jour. Cut. Dis., 1889, vii, p. 147, and Twentieth Century Practice, xv, p. 403. D. W. Montgomery, Jour. Amer. Med. Assoc., 1894, xxiii, p. 136, and Med. Record, 1902 (spontaneous cure in a leper family). Unna, Internat. Cong. of Derm., London, 1896: Zambaco, Leprosy, and Syphilis; Lepra-Conferenz, Berlin, October, 1897 (three volumes, full bibliography). Dyer, Phila. Med. Jour., 1898, ii, p. 567. Bracken, Minnesota State Board of Health, 1901, and Phila. Med. Jour., 1898, ii, p. 1309. Santon, "La Leprose," Paris, 1901 (plates). Victor Babes, "Die Lepra," Wien, 1901 (68 illustrations, 8 colored plates, and bibliography to date). McDonald, T. Jonathan, Jour. Amer. Med. Assoc., 1903, xl, p. 1567 (examination of 150 cases in Hawaii). Campana, Lepra, 3d ed., 1907. Wheery, Jour. Infec. Dis., 1908, v, p. 507; Jour. Amer. Med. Assoc., 1908, l, p. 1903. MacLeod, Brit. Jour. Derm., 1909, xxi, p. 309. Dyer and Hopkins, Jour. Amer. Med. Assoc., 1910, lv, p. 909 (Diagnosis of Leprosy). Unna, Trans. II Internat. Sci. Cong. on Leprosy, Bergen, August 16–19, 1909; Annales, October, 1910, p. 481 (abstr. Brit. Jour. Derm., 1911, xxiii, p. 164). Gurd and Duval, Jour. Cut. Dis., 1911, xxix, p. 274 (Experimental Leprosy and its Bearing on Serum Therapy). Currie, Clegg and Hollman, Public Health Bull., No. 47, U. S. Marine Hosp. Service, September, 1911, p. 3; abstr. Jour. Cut. Dis., 1912, xxx, p. 752 (Studies upon Leprosy; Cultivation of the Bacillus of Leprosy). Abstr. ibid., p. 23 (Attempt at Specific Therapy in Leprosy). Zinsser and Carey, Jour. Amer. Med. Assoc., 1912, lviii, p. 692 (A Contribution to the Study of Rat Leprosy). Smith and Rivas, New Orleans Med. and Surg. Jour., October, 1912, lxv, p. 265 (Cultural and Biological Characteristics of the Lepra Bacillus). Dyer, Jour. Amer. Med. Assoc., 1913, lxi, p. 950 (The Dermatologic Aspects of Leprosy). Montgomery, Trans. Sect. Derm., Amer. Med. Assoc., 1915, p. 84. Illustrations of the History of Leprosy. Morrow and Lee, ibid., p. 96; Symptoms and Diagnosis of Leprosy, with a report of cases.

Definition.—Leprosy is a chronic, contagious, and infectious disease, produced by *Bacillus lepræ* and characterized by cutaneous and constitutional symptoms, producing various deformities and mutilations of the human economy, and usually ending fatally.[1]

Leprosy is believed to have originated in the Orient and to be as old as the records of history. Together with a group of dermatoses, probably of a different nature, it was recorded in the Hebrew Scriptures.[2] Once prominent in the list of scourges of the Old World, its prevalence today is restricted. From the second to the seventh century it was abundant in Europe. Galen, in the second century, writes of it in France, Germany, and Spain. In the eleventh and twelfth centuries it spread all over Europe. In the fourteenth century it began to decline, and by the end of the seventeenth century it was to be found in only a few isolated localities. Early in the sixteenth century it had nearly disappeared from Italy, and somewhat later from France. At this time Denmark was free. It remained in Scotland for some time after leaving England, the last case being reported in the Shetland Islands in the latter part of the eighteenth century. During the last century it made its appearance and spread in new localities. About the middle of the century it was introduced into the Hawaiian Islands, where a serious epidemic prevailed. Morrow affirms that here, as in other newly-infected districts, the disease was at first quickly fatal and was usually of the nodular type, but of late years it has been less serious, and the milder or maculo-anesthetic type is the more prevalent.

At present leprosy is common in Iceland; throughout the whole of Asia, including the islands of the Indian Ocean; in South America, especially in Columbia, Venezuela, the Guianas, and Paraguay;[3] in Mexico, Central America, the West Indian Islands, particularly Cuba, and in the islands of the Pacific Ocean. It is found also throughout Africa, being more abundant throughout North and South Africa than in other portions of that continent. In Australia the disease is found chiefly in Queensland, New South Wales, and Victoria (Castellani). It is also found in Spain, Portugal, Italy, Turkey, Greece, the islands of Crete and Cyprus, the Balkans, Norway, Sweden, Russia; and to a variable degree in France, Germany, Denmark, Belgium, Holland, and Austria-Hungary. It also occurs in the province of New Brunswick, in Canada. In England and Scotland, where it once was prevalent, it is now among the rarest of diseases. The disease is represented also, in what is reported as a diminishing frequency, in the dependencies of the United States—the Hawaiian Islands, Porto Rico, and the Philippine Islands.

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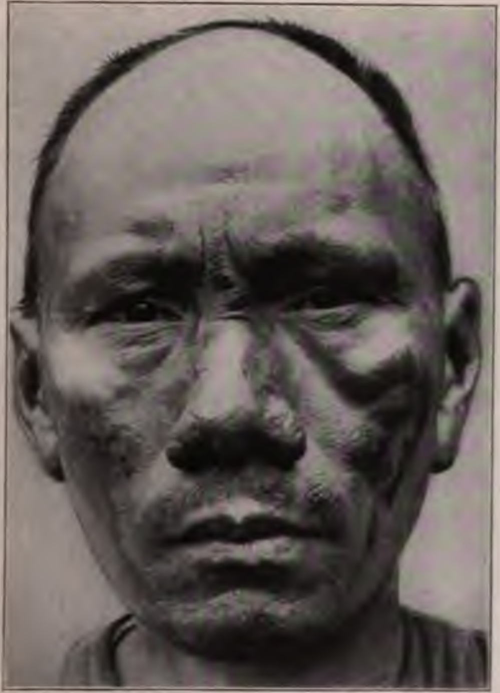
[1] Ormsby, Reference Handbook of the Medical Sciences, p. 484.

[2] McEwen, the Biblical World, No. 3, September, 1911: The Leprosy of the Bible in its Medical Aspects; ibid., No. 5, 1911: The Leprosy of the Bible in its Religious Aspects.

[3] Lindsay (Brit. Med. Jour., September 21, 1912, p. 682) states that in Paraguay the disease is rapidly spreading at the present time,

the United States[1] the disease first appeared in Louisiana, in esota[2] and other Northwestern States (where it was imported the Scandinavian Peninsula), and also to a lesser degree in h Carolina. At present it is chiefly found in South Carolina, ida, Louisiana, Texas, California, Minnesota, Wisconsin, and . Isolated cases occur in all the larger cities, including New York, on, Philadelphia, Chicago, and San Francisco. It is variously ated that the number of lepers in this country is between 250 500. In 1902, a report made by a commission of medical officers e United States Marine Hospital Service to the United States stated that there were 278 cases. In view of the difficulty ining reliable statistics, it is thought by most observers that present time the number is much greater than the above report indicate.

FIG. 312



Lepra. (Howard Morrow.)

ptoms.—The disorder has been described as occurring in several Danielssen and Boeck described these as the nodular and the

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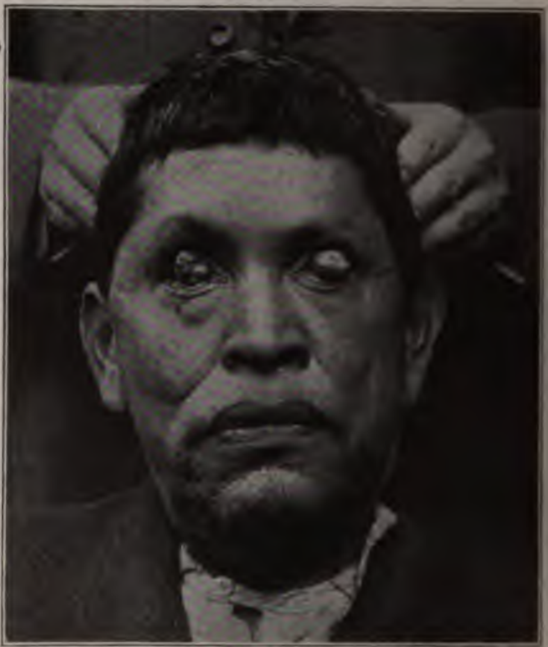
zer, Jour. Cut. Dis., 1911, xxix, p. 261: Historical Sketch of Leprosy in the tates (a good description of the history of the disease in the United States, the geographical distribution, and the probable number of cases).

kmann (Jour. Amer. Med. Assoc., lxi, 1913, p. 946) states that the old imported leprosy are gradually dying, and at the present time there remain but few cases.

anesthetic. The terms *Lepra tuberosa* and *Lepra maculo-anesthe* as adopted by Hansen and Looft,[1] seem more acceptable, as they mo nearly express the condition. While as a rule each affection runs i special course and is marked by symptoms so entirely different fro those belonging to the other as to make it appear a distinct disease, y some cases exhibit symptoms which are common to both forms, an in these the relationship is evident. They have a common etiologi factor, *Bacillus lepræ*, which, however, is in a different anatomi location and varies as to numbers in the two forms.

As in the case of other contagious and infectious diseases, th clinical history may be divided into stages. The period of incubatio

FIG. 313



Leproma of ocular globe. Epithelial horn pointing upward from eye. (Howard Mo

has been studied by competent observers and cannot be said to h a definite length. It is estimated to extend from a period of a weeks to many years. Before the eruptive and characteristic st develops, various prodromal symptoms such as may precede infectious disease occur. Among these may be mentioned fe chilliness, malaise, headache, gastro-intestinal disturbance, epista mental depression, drowsiness, insomnia, pains in the limbs, various anomalies of the motor and sensory apparatus, especially the limbs. As above stated, these symptoms may occur for but few weeks before the eruptive manifestations, or they may be present for years.

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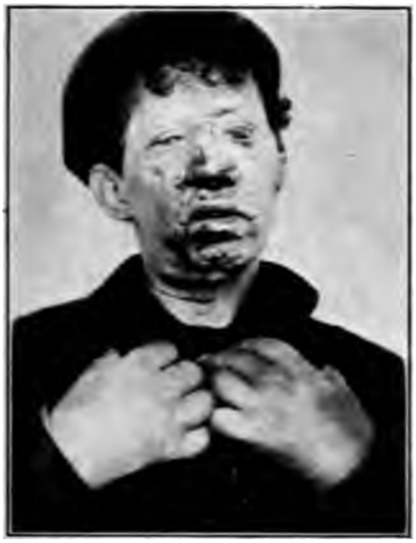
[1] Leprosy in its Clinical and Pathological Aspects. English translation by Wa London, 1895.

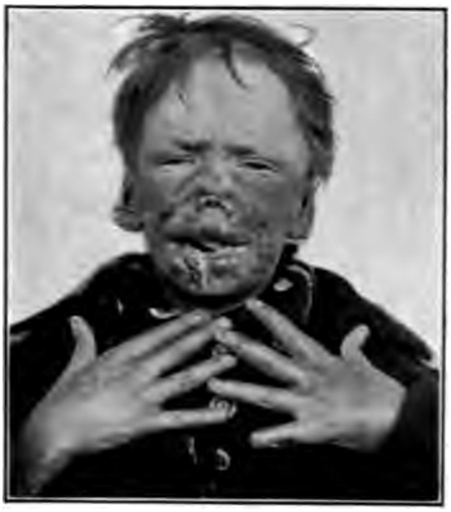
Lepra Tuberosa (*Tubercular, nodular, or tegumentary leprosy*).—From 10 to 50 per cent. of all cases of leprosy are of the nodular type, the larger proportion apparently holding good for colder climates. This type appears first as a macular eruption, of varying persistence, which may come and go, but in which finally the skin becomes infiltrated with characteristic nodules. The macules are well defined, round, oval, or irregular in shape, and vary in diameter from 1 to 10 cm. or more. Dependent upon the race of the patient, the color varies from a light-red to a purplish or bronze shade. The lesions may appear slightly elevated or infiltrated, or quite smooth and shiny, and somewhat hyperesthetic. Sooner or later, these patches become permanent and infiltrated with nodules, which are pea-sized, yellowish- or reddish-brown in hue, and enlarge more or less rapidly, some of them becoming as large as a walnut or larger. Their surface presents a shining appearance, as if varnished or oiled, and the skin may be soft and natural, or present slight desquamation. They may be grouped, forming areas of roundish or irregular contour, or may be isolated. By fusion broad infiltrations are formed, from the surface of which new nodules spring. These may be either cutaneous or subcutaneous in situation, and are softish or firm to the touch. The development of these lesions is not limited to the site of the previous macules, for some appear on apparently normal skin, and their efflorescence is preceded by febrile

FIG. 314



Leprosy in a Greek, aged thirty years. Tumefaction of the skin of the hands and nodules in the cornea. (Morrow.)

FIG. 315

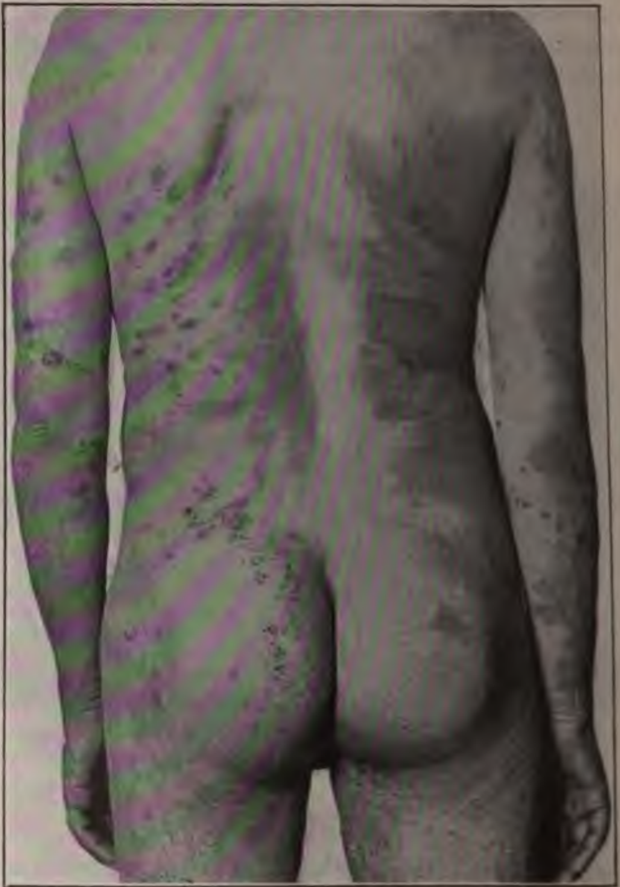


Nodular leprosy of the face and nerve lesions of the hands in a German boy aged fifteen years. (Morrow.)

symptoms more or less severe. Their development is not one of gradual progress, but rather of successive crops, each new efflorescence being preceded by febrile symptoms and the appearance of epistaxis. The lesions are often in various grades anesthetic.

The site of predilection of leprous nodules is the face, and thei massing in great numbers upon this region produces the characteristi

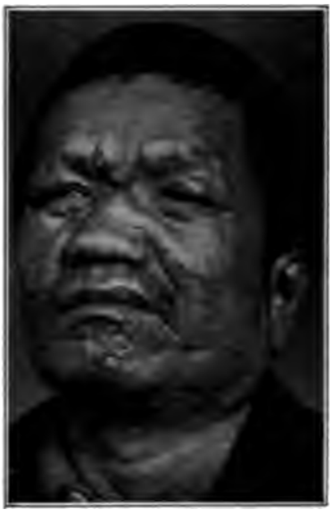
Fig. 316



Macular leprosy.

deformity of countenance which has given the disease one of its r *Leontiasis* ("face of a lion"). In such faces the nodules are ar in parallel series above the brows, down the nose, over the c the lips, and the chin. In consequence of the infiltration and de ment of the lesions, the brows deeply overhang the globes of th the eyelids become affected with partial ptosis, the lips pout, a ears are so studded with nodular masses as to project from th of the head. Unna[1] says that the lobe of the ear is a favorite and early site. The lesions are also frequently found on the forearms, thighs, and other portions of the extremities. To a lesser degree they are found on the trunk, but only on rare occasions do they occur on the palms. Involvement of the palm has been noted by Montgomery,[2] Stelwagon,[3] and the author.[4] Occasionally, with extensive development of nodules upon the face and ears, there may not be more than from five to fifty upon the rest of the body, and these may be either widely dispersed and isolated, or agglomerated in a single hard, flat, elevated plaque of infiltration upon the elbow or thigh. When confluence of nodules occurs, large plaques of infiltration may form (*lépromes en nappe*, Leloir), which are elevated and brownish or blackish in shade (*morphea nigra*). These patches are inert, lasting sometimes for years unchanged. They may be the seat of itching, or the normal sensations may become less acute. The hair falls in the involved regions, which may finally ulcerate. According to Danielssen and Boeck, these large plaques generally occur on the limbs and undergo an unusually chronic course. The nodules may degenerate into irregularly outlined, sharply cut, glazed ulcers, with a hemorrhagic or sloughing floor, some of which, especially under appropriate treatment, heal soon; while others extend deeply, often become gangrenous, and destroy much tissue. Other nodules undergo resorption and disappear, leaving pigmented, atrophic depressions; or they may lose their shape in consequence of partial resorption. A large plaque not infrequently is absorbed centrally, leaving an annular disk.

FIG. 317



Lepra. (Howard Morrow.)

The appendages of the skin show some involvement. Alopecia, particularly of the brows and eyelashes, usually occurs, but the scalp is rarely attacked. Occasionally, the nutrition of the nails is destroyed, evidenced by thinning, thickening, or other deformity. The secretions of the sebaceous and coil-glands are increased early in the disease, but later become diminished or entirely lost in the affected area. Comparatively early in the disease, small, flattish nodules form on the conjunctiva and cornea, extending to and involving the iris, and gradually filling the anterior chamber.

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[1] Histopathology, p. 604.

[2] Jour. Cut. Dis., 1899, xvii, p. 445.

[3] Diseases of the Skin, 7th ed., 1914, p. 922.

[4] Case demonstration before the Thirty-eighth Annual Meeting of the Amer. Derm. Assoc., 1914.

According to Hillis,[1] patches having raised, concentric edges, situated on the back of the pharynx and on the roof of the mouth, the back of the throat and the uvula, which are uniformly red and congested, are pathognomonic of leprosy. Later, the epiglottis, vocal cords, and other structures in the larynx become studded with nodules, as does also the nasal septum; and when ulceration occurs the cartilage and bony framework of the nose are destroyed, producing a characteristic deformity. Morrow believes that the earliest manifestations of leprosy in most cases are located in the mucous membrane of the pharynx and upper air-passages, as shown by alterations in the voice, rhinitis, increased nasal and salivary secretions, and sometimes epistaxis.

Heiser[2] noted a nasal ulcer situated on the septum at the junction of the cartilaginous and bony portion in 75 per cent. of 1200 cases. He regards this as the earliest and most constant sign.

Danielssen and Boeck have described an acute form of leprosy, manifested by continuous fever of about twelve days' duration, when, with a sudden efflorescence, raised, shiny, bluish spots appear over large surfaces of the body. These rapidly increase in volume and hardness, become confluent, and progress as far in a few weeks as does the ordinary form in years. With the appearance of the eruption the constitutional symptoms abate and the affection becomes chronic.

As a rule, the course of the disease is exceedingly slow, and years may elapse before the several changes above described are accomplished. The malady often appears to be quiescent for months at a time, after which, with the occurrence of fever, acute or subacute manifestations appear, which may be accompanied by relatively rapid ulcerative processes, followed by gangrene, in which case the disease may progress rapidly toward a fatal conclusion.

Lepra Maculo-anesthetica.—This form of the disease is more common in tropical than in cold countries. In this variety the bacilli are located chiefly in the neuroglia of the peripheral nerves, and consequently the symptoms exhibited in the part supplied by the affected nerves are those which would naturally follow their irritation, compression, or degeneration. Chief among these are the development of spots or macules, bullæ, muscular atrophy, anesthesia, motor paralysis, and finally mutilation by loss of parts. There is no regular sequence observed in the evolution of these symptoms. Usually, however, the maculæ and bullæ are among the earliest manifestations, but their appearance may be delayed for years.

The course of this form of the disease is exceedingly chronic, its average duration being estimated at about eighteen years. The appearance of the macules is usually preceded by anomalies of sensation, such as formication, a sensation of burning or stinging, or itching. The size of the lesion varies from that of a fifty-cent piece to that of the palm or larger. By peripheral extension and coalescence, large, irregular areas, having a curved contour, may be produced. At first they are reddish in color, changing with age to yellowish or brown, or even darker shades, when they tend to become slightly elevated and to desquamate. Their centers become depigmented and anesthetic, while the border may be hyperpigmented and hyperesthetic. Their commonest seats are usually considered to be the back, shoulders, face, arms (especially about the elbows), the nates, and around the knees. When the spots are fully developed, they may cover large areas of the cutaneous surface. In the anesthetic portion, and at times extending beyond it, the production of sweat is entirely suppressed and the hairs become white. The formation of bullæ is characteristic, and they may be the initial symptom, in which case they usually occur on the extremities, may be numerous, and are frequently hyperesthetic. The older are often single, large, and may be anesthetic. They appear suddenly, and if short-lived, which is the rule, have serous contents, the latter becoming purulent when the lesions persist. They heal after rupture, leaving a pale, anesthetic scar, or, by infection, develop into deep-seated ulcers.

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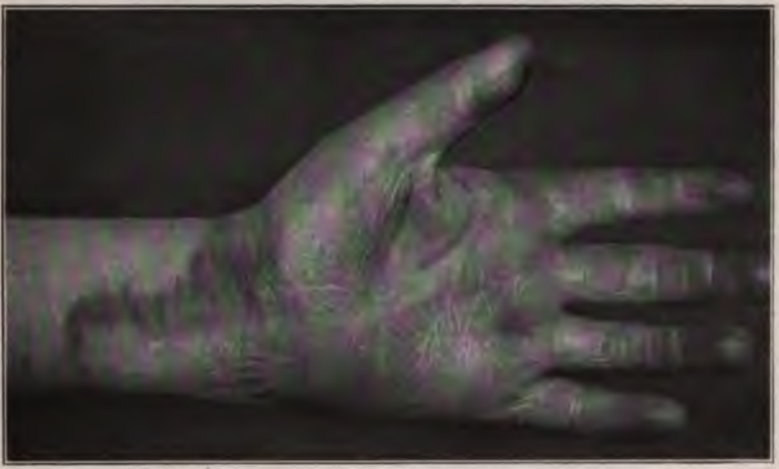
[1] Leprosy in British Guiana, 1881.

[2] New York Med. Jour., February 12, 1916, p. 289.

FIG. 318



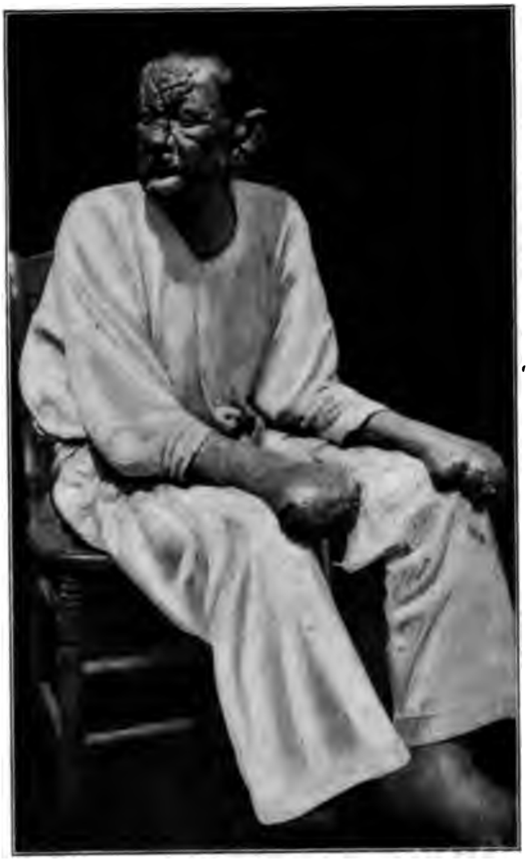
Lepra maculo-anesthetica.

When the nerve-trunks are the seat of severe neuritis, they become thickened and can be readily felt by the finger of the examiner. The fusiform enlargement of the ulnar nerve behind the olecranon process at the elbow is characteristic. Scheube calls particular attention to the involvement of the great auricular nerve, which, according to Bälz (quoted by Scheube), occurs in 90 per cent. of all cases. Other nerves especially involved may be the tibial, perineal, and, less often, the radial, median, brachial, and cervical.

Atrophic changes are noticed first in the interosseous muscles of the hands, the thenar and hypothenar being the next involved, the atrophy extending thence up to and involving the muscles of the forearms. In the hand this produces the "lepra claw," in which the proximal phalanges are extended, while the middle and distal are more or less flexed. Many other muscles are similarly involved.

Trophic disturbances through nerve-involvement are illustrated in the perforating ulcer of the foot and in the deep and destructive processes occurring in the fingers and toes. Hansen and Looft regard these changes as being largely due to external injury to a part of low vitality.

FIG. 319



Anesthetic leprosy, with mutilating results. (From a photograph of a leper in the Sandwich Islands.)

The ulcers are irregular, oval, roundish, or linear in outline; covered with thin, blackish, flattened, tenacious crusts; and anesthetic. The phalanges of the feet and hands may be attacked with necrosis, when complete exfoliation of the bone occurs. The various bone changes as revealed with x-rays are described by Houey.[1] The fingers and toes may thus disappear, leaving only soft processes, upon which the relic of a nail remains. Owing to the lack of unguent from its glands, the skin becomes dry and fissured and the seat of ulceration. To this stage of the disease the term *Lepra mutilans* was formerly applied.

From the foregoing description of the clinical course of the disease, with its subjective symptoms and objective lesions, it would seem that typical cases in the two forms are very different. The difference, however, is due to the selection of the skin in the one case for the growth of the organism, and of the nerves in the other. The so-called nodular variety frequently terminates in the maculo-anesthetic form; occasionally, the reverse is true; and in practically all cases of the nodular variety some anesthesia is present. The variety described under the heading of mixed forms simply indicates the development of nodules in a case formerly presenting symptoms of the maculo-anesthetic type; or anesthesia and other trophic disturbances following in the course of a nodular case. It is best, therefore, to look upon the expressions of the disease as exhibited in regard to the complexus of symptoms presented in syphilis. In each case there is a single morbid principle. There are in both no true varieties, and the external symptoms depend entirely upon the localization of the causative factors of the disease.

Etiology.[2]—The chief factors concerned in the etiology of leprosy, in view of the fact that the disorder is known to be produced by the bacillus discovered by Hansen, are the mode of invasion of the parasite, and the degree of contagiousness of the disease. In ancient and medieval times leprosy was considered very contagious. In 1869, however, the Royal College of Physicians declared against this theory; but Hansen and Looft quote statistics and give undeniable proof of the contagiousness of the disease in Norway. G. Thin[3] quotes a large number of cases in which leprosy was undoubtedly contracted by contagion. Among other examples, Morrow[4] quotes the case of Father Damien, a priest whose personal and family history bore no taint of previous disease, but who, after close contact with lepers in the Hawaiian Islands in a leper settlement, contracted the disease. Its development in new countries, or in new localities in the same country, can usually be proven to have been produced by contagion. The contagiousness is not marked, and it requires the closest relationship between the leprous and the healthy before the disease can be contracted. The Lepra Commission in India, in 1893, reported that leprosy in India cannot be considered an hereditary disease, and stated that the evidence which exists is hardly sufficient to establish, to an appreciable degree, an hereditary predisposition to the disease in the offspring of leprous patients. Besnier says that if any hereditary predisposition exists, it is less than in tuberculosis.

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[1] Am. Jour. Roentgenology, 1917, iv, p. 494; Bone Changes in Leprosy.

[2] Chipman, Trans. Sect. Derm. Amer. Med. Assoc., 1915, p. 108; The Etiology and Treatment of Leprosy.

[3] Leprosy, London, 1891.

[4] A System of Genito-Urinary Disease, Syphilology and Dermatology, iii, p. 562.

As to its mode of transmission from one person to another, different opinions prevail. Thin[1] believes that close personal contact is usually necessary; but that the possibility of infection being carried by an intermediate host, such as the mosquito or the acarus, is not unreasonable. Currie[2] concluded, after a series of experiments, that mosquitoes do not transmit the disease, but that it may be transmitted by flies. The rôle played by the bedbug in the transmission of the disease is undetermined. Goodhue[3] and Long[4] have found lepra bacilli in bedbugs which had recently fed on leprous subjects. Innes[5] believes leprosy is not transmitted by the bedbug. Hansen and Looft quote cases in which the affection was carried by way of the clothing. Morrow believes that in the majority of instances the contagion gains entrance to the body through the mucous membranes of the respiratory and gastro-intestinal tracts. That it spreads from one person to another seems proved by the fact of its disappearance in a community when isolation is practised. Neisser says that the number of lepers in any community bears an inverse ratio to the care taken to insure the isolation of infected persons.

Leprosy may occur at any age, seldom, however, before the fifth year, and most often between the thirtieth and fiftieth years. Damp, cold climates, as well as moist, hot ones, are most favorable for its development. As predisposing factors, lack of hygiene, dirt, filth, and poverty undoubtedly play an important rôle.

Pathology.—Histologically, leprosy belongs to the group of infectious granulomata. It is a neoplasm, made up largely of partially reverted connective-tissue cells. In this new growth a specific bacillus is present in extraordinarily large numbers. This organism bears a striking resemblance to the bacillus of tuberculosis, both morphologically and as regards its staining reactions. It was discovered by Hansen, in 1874, and his observations were independently confirmed by Neisser, in 1879. It is somewhat thinner and slightly longer than the tubercle bacillus, though variations occur. Its average length is from 5 to 6 microns. It frequently presents a beaded appearance, which has led some observers to believe that it undergoes spore-formation. Often in tissue sections the whole area is seen to be crowded with the bacilli. The organisms appear in groups, which are usually situated in large, ovoid cells, which constitute the lepra-cell. Johnston,[6] after the examination of the bacillus in a large number of cases of leprosy, described four forms in which it occurs: (1) The ordinary form commonly described; (2) fragmented type (*a*) coarse granules, (*b*) fine granules. (3) Solid type, rare, exhibited as (*a*) long form, (*b*) short form, and (4) nocardial type or streptothrichal type. This is non-acid-fast and is the parent of *Bacillus lepræ*.

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[1] Loc. cit.

[2] Public Health Bull., Washington, 1910; full abst. Jour. Tropical Med. and Hygiene, May 1, 1911, p. 138; abstr. Brit. Jour. Derm., 1911, xxiii, p. 334.

[3] Boston Med. and Surg. Jour., 1906, cliv, p. 537.

[4] Brit. Med. Jour., 1911, ii, p. 470; abstr. Jour. Amer. Med. Assoc., 1911, lvii, p. 1164.

[5] Brit. Jour. Derm., 1918, xxx, p. 91; A Leper Colony on the Borders of Abyssinia.

[6] Philippine Jour. of Sc., 1917, xii, p. 115; "The Varying Morphology of Bacillus Lepra."

In the discharges from ulcers and diseased tissues, large numbers of bacilli are present, and the same tendency to grouping in ovoid masses is often noticeable. The bacilli are found in great numbers in the nodules of the tuberous variety, whereas in the macules of the maculo-anesthetic variety they appear in smaller numbers, but are numerous in the connective-tissue sheaths of the peripheral nerves. Honeij[1] demonstrated the bacillus in the blood in 9 out of 28 cases examined. Successful inoculations of the lower animals and cultivation of the organism on artificial media outside of the human body have been made only comparatively recently. Campana claimed to have grown the organism in subcultures on glucose agar, but his observations were not confirmed. Opinion is divided as to whether the microörganism of leprosy has been successfully cultivated. The most important positive findings in this field were made by Clegg and Duval. The former, in 1909, was successful in growing an acid-fast bacillus from leprous tissue on agar in symbiosis with amebæ. This organism has chromogenic properties.[2] Duval and Wellman[3] describe efficient methods for the cultivation of the lepra bacillus from tissues. They further state that different organisms seem to have been cultivated from these tissues: (1) a non-acid-fast diphtheroid (Kedrowski); (2) an acid-fast chromogenic bacillus (Clegg); and, (3) a permanently acid-fast bacillus (Duval), which *in vitro* maintains the morphology and tinctorial properties of the Hansen bacillus of the tissues, but grows only in the presence of certain nutrients. From a bacteriological study of 29 cases, they report the cultivation of an acid-fast bacillus from 22. In 14 of these they found a chromogenic organism somewhat similar to that described by Clegg, which under certain conditions grows (1) as a non-acid-fast streptothrix; (2) as a non-acid-fast diphtheroid; and (3) as an acid-fast bacillus. These characteristics, they believe, explain the interpretations of European writers in describing *Bacillus lepræ* as a pleomorphic organism, which may be recognized as a diphtheroid, a streptothrix, or an acid-fast bacillus. In addition to the chromogenic organism, they isolated from 8 cases an organism distinctly different in its biologic character, growing only on specific media and not producing pigment. Serological tests suggest that the bacillus of Clegg is not related to the non-chromogenic, slow-growing organism of Duval. Fraser and Fletcher[4] excised material from 52 non-ulcerating nodular lesions and employed the methods of every claimant to the growth of the microörganism, but substantiated none of the findings. They concluded that the bacillus of leprosy has not been cultivated, and that the diphtheroids and other organisms are merely contaminations. They further say that their experiments led them to conclude that there is no evidence that the acid-fast bacillus of Kedrowsky is the leprosy bacillus.

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[1] Jour. Infec. Dis., 1915, xviii, p. 376; "The Leprosy Bacillus in the Circulating Blood and Excretions."

[2] Philippine Jour. of Sci., 1909, iv, p. 403; Duval, Jour. Exper. Med., 1911, xiii, p. 365.

[3] Jour. Amer. Med Assoc., 1912, lviii, p. 1427, and Jour. Cut. Dis., 1912, xxx, p. 397.

[4] Annals of Tropical Med., 1915, ix, p. 381; "The Cultivation of the Leprosy Bacillus."

Successful animal experiments also have recently been abundantly reported. Duval[1] injected 1 c.c. of a heavy homogeneous suspension of lepra bacilli into the subcutaneous tissues of the inguinal region of a full-grown *Rhesus* monkey, and after forty-six days the animal showed typical signs of disseminated infection and presented the clinical picture of human leprosy of the nodular type. Stanziale[2] reported the successful inoculation of the disease into rabbits and various laboratory animals for two or three generations.

Diagnosis.—Apart from the history, present and previous places of residence of the subject of the disease, and the clinical symptoms exhibited, the diagnosis of lepra is to be established by the presence of lepra bacilli. These organisms may be recognized in the tissues, in serum obtained from involved regions, in blood made to exude from lepromatous nodules, in nasal discharges, and in the secretions of ulcers. In tissues the antiformin method may be employed for the discovery of the bacillus.

In the prodromal stages no suspicion of the existence of the disease would be awakened in countries where it is not endemic. In the eruptive and later stages of the disorder, and particularly in well-marked cases, the recognition of the disease is simple.

From syphilis, which is also a disorder the lesions of which are polymorphic in character, lepra can be distinguished by its much greater chronicity; its larger and brownish-yellow, glazed nodules; its frequent paresthetic and anesthetic symptoms; its bullous lesions, rare in acquired syphilis; the far more extended areas of its erythematous macules; its blackish crusts, lacking the rupioid aspect of those in syphilis; its leathery, mica-tinted cicatrices; and the characteristic leonine facies of its nodular forms. As the Wassermann reaction is frequently positive in nodular leprosy, this method of diagnosis loses its value in the differentiation from syphilis.[3]

Morphea and vitiligo are unattended by constitutional changes, and more particularly exhibit no hyperesthetic or anesthetic symptoms in the affected patches.

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[1] University of Pennsylvania Med. Bull., p. 665; abstr., Brit. Jour. Derm., 1911, xxiii, p. 300.

[2] Annals Tropical Med., 1916, x, p. 165; "Experimental Researches on the Bacteriology of Leprosy."

[3] Bruck and Gessner, Berlin klin. Wochenschr., 1909, xlvi, p. 589; abstr. Jour. Cut. Dis., 1909, xxvii, p. 430 (Wassermann in Leprosy; 10 cases, 7 tubercular, 3 maculo-anesthetic; 5 positive and 5 negative, the positive all tubercular). Sequeira, Brit. Jour. Derm., 1911, xxiii, p. 190 (Wassermann Reaction in Leprosy; 2 cases, positive in one, negative in the other). Bloombergh, Philippine Jour. of Sci., October, 1911, vi, p. 335; abstr. Brit. Jour. Derm., 1912, xxiv, p. 160 (Wassermann Reaction in Syphilis, Leprosy, and Yaws. Reports that of 21 cases of leprosy 18 gave a positive reaction). Gilmour, Jour. Cut. Dis., 1913, xxxi, p. 426 (Case presentation; positive Wassermann). Nakajo and Asakura (Jour. Infec. Dis., 1915, xvii, pp. 388-400; Serologic Diagnosis of Leprosy), obtained a positive reaction in 78.79 per cent. of 33 cases examined. Sutherland and Mitra (Indian) Jour. Med. Research, April, 1915; abstr., Brit. Jour. Derm., 1915, xxvii, p. 385; obtained a positive reaction in 30 per cent. of 34 cases examined.

Multiple sarcomata, especially upon the face, are followed by much more rapid degeneration and fatal result.

All lesions of erythema multiforme can readily be distinguished from those of lepra by the absence of hyperesthesia or of anesthetic symptoms.

Syringomyelia is differentiated by its display of lesions only in regions where there is also muscular atrophy; by the much greater extent and lack of definition of areas of perturbed sensation; by diminution of the tendon reflexes, which may be exaggerated in lepra; by a marked predominance of symptoms in the upper as distinguished from the lower extremities; and by the frequent presence of scoliosis.

The nodules of lupus are not symmetrical, are far softer, and are much more often grouped than those of lepra. Further, they never have the size of the larger leprous nodules, and never have the peculiar pigmented, brownish, and oiled or varnished aspect of leprous nodules.

Prophylaxis.—As leprosy is unquestionably contagious, the question of the protection of the unaffected is important. Particular attention has been directed in America to this subject by J. C. White,[1] Hyde,[2] Morrow,[3] Dyer,[4] and others. Recently the United States Public Health Service has directed much attention to the question of the spread of the disease and its prevention. According to Blue,[5] there now exists a law preventing the immigration of patients suffering with the disease. He further states that provision for taking care of such patients has been made in Louisiana, Mississippi, and California. In Minnesota isolation of the patient at home is required. A few other States have some mention of the disease in their statutes; in a number of others nothing whatever has been done in regard to it. Blue suggests a national leper home, to be established under the Public Health Service, for the care and treatment of these unfortunates, as a humane solution of the problem of their care, and the limitation of the disease by the segregation of its victims.[6]

Treatment.—Chaulmoogra oil has the highest reputation in the treatment of leprosy. This drug is obtained from the seeds of *Gynocardia odorata*, from which it should be obtained by cold expression. It is employed by internal administration, intramuscular and intravenous injection, and by local inunction. When employed internally it is given in milk, in emulsion or in capsules, in doses varying from 15 to 200 or more minims daily. It should be given over a long period of time in order to get the best results. Gynocardiac acid, its active

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[1] Trans. Internat. Lepra Convention, 1897, i.

[2] Trans. Cong. Amer. Phys. and Surg., 1894, iii, p. 103.

[3] Loc. cit.

[4] Jour. Cut. Dis., 1911, xxix, p. 268.

[5] Jour. Amer. Med. Assoc., 1913, lxi, p. 943.

[6] *Cf.* Rucker, Trans. Amer. Med. Assoc., Sec. on Derm., 1914, p. 34: The necessity for the establishment of a National Leprosarium; and Dyer, ibid., p. 39: The Duty of the Government in Leprosy Care and Control.

·inciple, is administered as a salt in combination with either sodium · magnesium in doses of ½ to 3 grains (0.033–2). Heiser[1] obtained)od results using the oil with camphor and resorcin in the following)mbination suggested by Mercado: Chaulmoogra oil, 60 grams; ımphorated oil, 60 grams; and resorcin 4; dissolve on a water-bath ıd filter. Weekly injections of 1 c.c. are suggested, increasing the)se to the patient's tolerance. This preparation is easily absorbed ıd produces but little local discomfort. Bercovitz[2] and Coghill[3] ınd this combination and method efficient. Rogers[4] found intra-·nous injections of gynocardate of sodium superior to the use of the l intramuscularly. Good results are similarly recorded from the use sodium gynocardate and sodium gynocardate "A" by Cathew[5] and [uir.[6] Hollman and Dean[7] found the Mercado combination of value ıd also one advised by Hollman and Currie constituted as follows:

℞—Iodin,	grs. xx;	1	33
Camphor,	ʒss;	2	
Oil of eucalyptus,	ʒij;	8	
Olive oil,	℥iv–ʒiv;	147	
Chaulmoogra oil,	℥iv–ʒv;	150	

M. in order given with heat.
Sig.—Maximum dose 10 c.c. intramuscularly once weekly.

In addition, they found fatty acids of chaulmoogra oil as prepared ' Dean, administered subcutaneously, superior to chaulmoogra oil ixtures.[8] Rogers[9] suggests sodium morrhuate as an efficient prepara-)n used as a subcutaneous injection. Potassium cuprocyanide has so been of value, as demonstrated by Takano.[10] Crocker recom-ended injections of bichlorid of mercury ¼ grain (0.016) in 20 minims water intramuscularly twice weekly. This treatment was found of ırticular value in the early stages. Radiotherapy has relieved the ıtaneous symptoms and, according to several writers[11] has apparently ıtirely cleared up the disorder. Phototherapy has occasionally been used with the same results.[1] Unna recorded the successful management of the disease by the combined use of ichthyol internally in increasing doses, and the local application of 10 per cent. pyrogallic acid ointment on the arms and legs twice daily. In addition, a 10 per cent. chrysophanic acid ointment was rubbed into the cheeks and trunk together with a plaster consisting of chrysophanic acid, salicylic acid and creosote, which was applied daily to the forehead and chin. After one month's treatment of this sort a series of warm baths were employed and then the treatment was resumed.

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[1] New York Med. Jour., February 12, 1916, p. 289.

[2] Jour. Amer. Med. Assoc., 1917, lxviii, p. 1960; "The Hypodermic Use of Chaulmoogra l in Leprosy."

[3] Annals Tropical Med., 1917, ii, p. 205; "Heiser's Treatment of Leprosy."

[4] Brit. Jour. Derm., 1916, ii, p. 550; "Intravenous Injection of Sodium Gynocardate."

[5] Indian Med. Gazette, 1918, liii, p. 407; "Leprosy Treated with Sodium Gynocardate d Sodium Gynocardate 'A'".

[6] Ibid., p. 209 and ibid., 1919, liv, p. 130; "Treatment of Lepers with Gynocardate of da."

[7] Jour. Cut. Dis., 1919, xxxvii, p. 368; "Chaulmoogra Oil in the Treatment of Leprosy."

[8] *Cf.* Recent important contribution on experimental research in Chalmoogra oil rivatives by Walker and Sweeney, Jour. Infect. Dis., 1920, xxvi, p. 238; The Chemo-erapeutics of the chalmoogric Acid series and other Fatty Acids in Leprosy and ıberculosis.

[9] Indian Med. Gazette, 1919, liv, p. 218; "Subcutaneous and Intravenous Injections Sodium Morrhuate in Leprosy."

[0] Jour. Exper. Med., 1916, xxiv, p. 207; "The Treatment of Leprosy with Cyano-prol," and Sugai, Amer. Jour. Med. Sc., 1918, clvi, p. 30; "Chemotherapy of Leprosy d Tuberculosis."

[1] Malcolm Morris, Brit. Jour. Derm., 1908, xx, p. 416; Heiser, Med. Record, October , 1908; abstr., Jour. Cut. Dis., 1909, xxvii, p. 275; Wilkinson, the author and others.

Serum and vaccine therapy have not been successful. The Carrasquilla serum, which is prepared by the injection of the blood of lepers into horses, and other sera have not proved to be of much value. A substance like tuberculin prepared by Rost[2] and given by injection weekly was reported to be of some value. Arsphenamin was given a rather thorough trial and both favorable and negative results were recorded. Isaac, Senator and Benda[3] obtained negative results after two injections.

Nastin is a preparation elaborated by Deycke. It is a neutral fat obtained by making an ethereal extract of a streptothrix, which Deycke cultivated from cases of leprosy in Constantinople. Nastin is combined with benzol chlorid and made into ampules with sterilized olive oil and given as a hypodermic injection. It was claimed by the originator that it causes bacteriolysis and in suitable cases should be of value. As with other preparations, improvement and negative results have been obtained from its use. Deycke,[4] Rudolph,[5] Gottheil[6] and others report improvement. MacLeod[7] used nastin without results and then substituted leprolin and obtained decided improvement. Teague[8] after using nastin in several cases without benefit, concludes that it is purely an empirical drug treatment and not a vaccine. Wise[9] used benzol chlorid in mineral oil with better results than were obtained with nastin. Rogers[10] gave nastin a thorough trial in the Gobra Leper Asylum, Calcutta, and concluded it was useless.

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[1] Pasini, Giorn Ital., 1907; abstr., Brit. Jour. Derm., 1908, xx, p. 276; Morris, loc. cit., and others.

[2] Brit. Jour. Derm., 1908, xx, p. 90. In a discussion of cases presented by Crocker and Pernet, Rost stated that he had begun treating cases four years previously with this material and had obtained good results.

[3] Berl. klin. Wchnschr., xlviii, No. 11, p. 470; abstr., Jour. Cut. Dis., 1911, xxix, p. 365. Montesanto, München. med. Wchnschr., lviii, No. 10, p. 511; abstr., Jour. Cut. Dis., 1911, xxix, p. 366. Wellman, New York Med. Jour., November 16, 1912, xcvi, p. 996; abstr., Jour. Cut. Dis., 1913, xxxi, p. 364.

[4] Brit. Jour. Derm., 1910, xxii, p. 26; a report on the Second Internat. Sci. Cong. on Leprosy held at Bergen, August 16–19, 1909; in a discussion of the treatment of leprosy by nastin injections, several stated that they considered the employment of nastin of doubtful value.

[5] Archivos Brasileiros de Medicins, June, 1912, No. 3; abstr., Brit. Jour. Derm., 1912, xxiv, p. 608.

[6] Jour. Cut. Dis., 1911, xxix, p. 239.

[7] Brit. Jour. Derm., 1912, xxiv, p. 229.

[8] Philippine Jour. Aci., iv, No. 5, p. 329; abstr., Brit. Jour. Derm., 1912, xxiv, p. 371.

[9] Jour. of London School of Tropical Med., 1911, p. 63; abstr., Brit. Jour. Derm., 1912, xxiv, p. 82.

[10] Lancet, February 5, 1916, p. 228.

ulcerations. These so-called stages may not occur in regular order as in rare instances the tumors may precede or be the only manifestation of the disease. A striking feature is the transient nature of some of the lesions, particularly in the early stages. They will appear, remain for a varying length of time and then disappear, to reappear in the same or new locations. Even in the tumor stage lesions undergo spontaneous involution.

Prefungoid or Premycotic Stage.—The earliest symptoms of the disorder may be pruritus without visible lesions on the skin. More often an eruption resembling eczema, psoriasis, dermatitis exfoliativa or parapsoriasis occurs. The most common eruption consists of red, scaling patches of irregular outline and poor definition, which are more or less generally disseminated, or these patches may be round or oval and well-defined with slightly elevated margins. At times the lesions clear in the center and form rings. These lesions range from small coin to palm size or larger. Sometimes they are moist and closely resemble eczema or, when scaling is abundant, psoriasis is imitated. Less frequently vesicles or bullæ are present. Exceptionally the early eruption is general and has the appearance of dermatitis exfoliativa. The close resemblance of the early eruption to parapsoriasis was emphasized in the case of a patient studied by the author. In this instance the disease was considered parapsoriasis for several years by well qualified men. Later infiltrated plaques and tumors developed and the patient succumbed. Similar cases are recorded by Jamieson,[1] Hudelo and Gaston,[2] Sherwell, Unna, and Wise and Rosen.[3] While the psoriasiform plaques above described are usually the early expressions of the disease, true psoriasis may precede the disorder. Such a case was reported by Howard Fox.[4]

In a case described by Hazen and Strobel[5] the early lesions were follicular papules which, by coalescence, produced patches.

These early lesions may persist for some time or disappear spontaneously, leaving pigmentation. Itching is usually a prominent symptom, though it may be mild or occasionally absent. After a variable length of time extending over a period of a few months to several years the infiltrated lesions characteristic of the disorder appear.

Stage of Infiltration.—The lesions of this stage are flat, well defined, slightly elevated plaques of different sizes and configuration. Circinate or gyrate figures are produced by peripheral enlargement and central involution of the plaques. These lesions are usually bluish-red, but yellow and brown may be admixed. They are more persistent than the

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[1] Jour. Cut. Dis., 1901, xix, p. 440.

[2] Annales, 1904, s., iv, v, p. 1090.

[3] Jour. Cut. Dis., 1916, xxxiv, p. 95; A case of early mycosis fungoides clinically indistinguishable from parapsoriasis of Brocq.

[4] Jour. Amer. Med. Assoc., August 13, 1913, lxi, p. 330; Report of a case of granuloma fungoides, preceded for many years by true psoriasis; with review of the literature of cases presenting psoriasiform lesions.

[5] Jour. Cut. Dis., 1911, xxix, p. 147; Report of a case in a negro, with thorough discussion of the relation of the disease to the sarcomata, lymphomata, and granulomata. Extensive bibliography.

The local treatment of leprosy is largely surgical and is that outlined for the treatment of ulcers and the removal of deformed parts. One or more hot baths daily are of distinct value in most cases. A review of the above outline of treatment demonstrates the fact that no remedies are known to have a directly curable effect in leprosy. In consequence, the treatment of the disease is that suggested by indications, in which case a change of residence, if the patient resides in a district where the disease prevails, the adoption of a highly nutritious diet, the administration of tonic remedies, including quinin, iron, cod-liver oil and strychnia, and the rigid enforcement of appropriate hygiene, in connection with the administration of chaulmoogra oil or its derivatives, offer the patient the best chance of keeping the disease under control.

Prognosis.—The future of the patient affected with leprosy is in general grave. The disorder is often malignant in character, and, however protracted, a fatal result usually occurs. With a change of climate and improved hygienic conditions much has been accomplished. The Scandinavian lepers who have emigrated to the United States have been benefited greatly by the change. This was the opinion of the late Professor Boeck, who studied the condition of leprous patients who had come to this country from Norway. Cases of both maculo-anesthetic and tubercular leprosy concluding with complete recovery are now sufficiently numerous to suggest that with the advantage of all the facilities now at command for its management the future of these patients may be much more promising.

GRANULOMA FUNGOIDES.[1]

Definition.—Granuloma fungoides is a grave disease which in its early stages is characterized by superficial inflammatory lesions, and later by malignant neoplasms.

This disease was first described in 1814 by Alibert as "*Pian fongoïde*" on account of its resemblance to yaws, but at a later date, owing to the presence of mushroom-like tumors, the name was changed to mycosis fungoides.

Symptoms.—It is usual to describe the disease in three stages: The first includes lesions of superficial type with little or no infiltration of the skin. The second is characterized by lesions showing more or less infiltration, and the third begins with the appearance of tumors and ulcerations. These so-called stages may not occur in regular order as in rare instances the tumors may precede or be the only manifestation of the disease. A striking feature is the transient nature of some of the lesions, particularly in the early stages. They will appear, remain for a varying length of time and then disappear, to reappear in the same or new locations. Even in the tumor stage lesions undergo spontaneous involution.

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[1] Bibliography: For further discussion of the subject, see in addition to the authors quoted in the text: Walters, Bibliothica Medica, Abt. D. ii, H. 8, Stuttgart, 1899 (sixteen illustrations and complete bibliography); Leredde, La Pratique Dermatologique, t. iii, p. 527 (bibliography); Stowers, Brit. Jour. Derm., 1903, xv, p. 47 (bibliography); Paltauf, Mraček, Handbuch, Wien, 1909, pp. 663–791 (critical review, with complete bibliography). Discussion on Mycosis Fungoides, opened by Sequeira, Royal Soc. of Med., London, May 21 and June 4, 1914 (Brit. Jour. Derm., 1914, xxvi, pp. 213 and 249; extensive bibliography); discussion by Pringle, Sir Malcolm Morris, McDonagh, Heath, Lancashire, Bolam, Nixon, Wills, Ward, Galloway, Stowers, Abraham, Whitfield, Pernet, Graham Little, Weber, MacLeod, Dore, and Travers Smith.

Prefungoid or Premycotic Stage.—The earliest symptoms of the disorder may be pruritus without visible lesions on the skin. More often an eruption resembling eczema, psoriasis, dermatitis exfoliativa or parapsoriasis occurs. The most common eruption consists of red, scaling patches of irregular outline and poor definition, which are more or less generally disseminated, or these patches may be round or oval and well-defined with slightly elevated margins. At times the lesions clear in the center and form rings. These lesions range from small coin to palm size or larger. Sometimes they are moist and closely resemble eczema or, when scaling is abundant, psoriasis is imitated. Less frequently vesicles or bullæ are present. Exceptionally the early eruption is general and has the appearance of dermatitis exfoliativa. The close resemblance of the early eruption to parapsoriasis was emphasized in the case of a patient studied by the author. In this instance the disease was considered parapsoriasis for several years by well qualified men. Later infiltrated plaques and tumors developed and the patient succumbed. Similar cases are recorded by Jamieson,[1] Hudelo and Gaston,[2] Sherwell, Unna, and Wise and Rosen.[3] While the psoriasiform plaques above described are usually the early expressions of the disease, true psoriasis may precede the disorder. Such a case was reported by Howard Fox.[4]

In a case described by Hazen and Strobel[5] the early lesions were follicular papules which, by coalescence, produced patches.

These early lesions may persist for some time or disappear spontaneously, leaving pigmentation. Itching is usually a prominent symptom, though it may be mild or occasionally absent. After a variable length of time extending over a period of a few months to several years the infiltrated lesions characteristic of the disorder appear.

Stage of Infiltration.—The lesions of this stage are flat, well defined, slightly elevated plaques of different sizes and configuration. Circinate or gyrate figures are produced by peripheral enlargement and central involution of the plaques. These lesions are usually bluish-red, but yellow and brown may be admixed. They are more persistent than the ones, but may undergo spontaneous involution, leaving pig- l areas. Pea-sized or larger nodules are occasionally present. ce may be the site of plaques and nodules, giving it the clinical ance seen in lepra. Partial or complete alopecia is not infre- in the scalp and the hair is also lost in other regions. At times ted plaques occur early with the more superficial lesions. This s of shorter duration and is followed after several months by the ge, which is initiated by the development of tumors.

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[1] Jour. Cut. Dis., 1901, xix, p. 440.

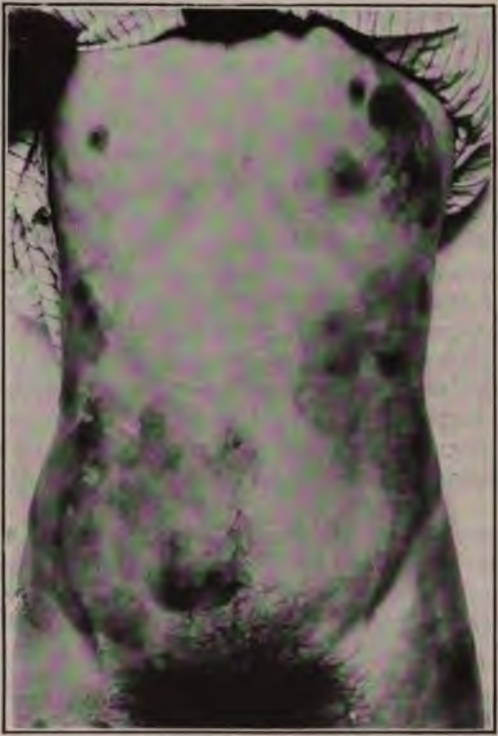
[2] Annales, 1904, s., iv, v, p. 1090.

[3] Jour. Cut. Dis., 1916, xxxiv, p. 95; A case of early mycosis fungoides clinically indistinguishable from parapsoriasis of Brocq.

[4] Jour. Amer. Med. Assoc., August 13, 1913, lxi, p. 330; Report of a case of granuloma fungoides, preceded for many years by true psoriasis; with review of the liter cases presenting psoriasiform lesions.

[5] Jour. Cut. Dis., 1911, xxix, p. 147; Report of a case in a negro, with cussion of the relation of the disease to the sarcomata, lymphomata, a Extensive bibliography.

FIG. 320



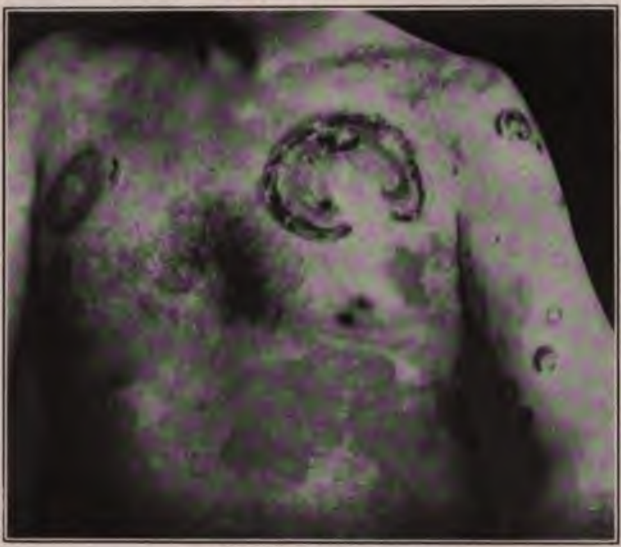
Mycosis fungoides, tumor stage just beginning. (Morrow.)

oid, or Tumor Stage.—Tumors develop usually in areas previously d, but occasionally upon an apparently normal surface. They e limited in distribution or widely disseminated, and may be on any portion of the cutaneous surface. They may be bean- ge-sized or larger. They may be sessile or pedunculated, oval ded, lobulated, and are well circumscribed. They may be bright k bluish- or brownish-red. Occasionally by coalescence large are formed inducing marked disfigurement. They may dis- like the early lesions but new ones continue to develop. Early urface is smooth or scale-covered, but later they ulcerate. In the process of ulceration characteristic mushroom-like or fungating masses are produced. At this time (the cachectic stage of Stelwagon) the general health becomes greatly altered. Symptoms of sepsis arise, exhaustion occurs and the disorder soon terminates fatally.

Sometimes tumors develop independent of the earlier stages (*tumeurs d'emblée* of the French). Occasionally lesions of the earlier stages follow. In one of Crocker's cases tumors of the face preceded the ordinary infiltrations on the skin. Little[1] reported a similar case. Senear[2] recorded a case with lesions limited to one foot with no other symptoms.

The course of the disease is progressive toward a fatal termination, though the earlier stages may extend over a period of years. The terminal stage usually ends in a period of months, and in rare instances the whole course is short. In a case recorded by Parkes Weber[3] the disease proved fatal in five months.

FIG. 321



Granuloma fungoides.

Etiology.—The disease is more frequent in men than in women. It usually occurs between the thirtieth and fiftieth years, more often after the fortieth, although the earliest symptoms have been noted in an individual fifteen years of age. Though the cause of the disease is not known, there can be little question as to its infectious character. Various organisms, chiefly of pyogenic type, have been discovered, but they must be considered secondary invaders. Inoculation experiments have been performed by many investigators, including Stelwagon and Hatch[4] with negative results. The disease is probably oduced by a specific microörganism, but the direct proof of contagion the demonstration, experimentally or otherwise, of such an organism s not been made.

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[1] Brit. Jour. Derm., 1919, xxxi, p. 46.

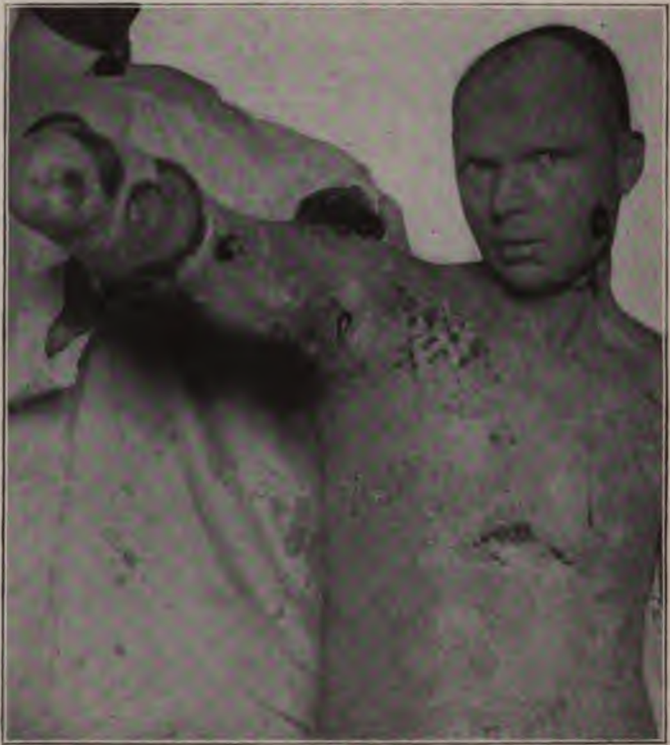
[2] Jour. Cut. Dis., 1915, xxxiii, p. 351.

[3] Brit. Jour. Derm., 1918, xxx, p. 7.

[4] Jour. Cut. Dis., 1892, x, p. 1.

Symmers[1] states "There are apparently excellent reasons for the ief that mycosis fungoides is primarily a disease of the hemopoietic stem more particularly the lymph nodes, in the course of which ersified skin changes arise, partly as a phenomena secondary to the charge of lymphotoxins or myelotoxins, or both, and partly as a ult of infiltration of the skin by the same variety of cells as charac- ize the structure of the diseased lymph nodes themselves."

FIG. 322



Granuloma fungoides.

Pathology.[2]—The disease has been studied by an unusually large mber. While the reports of different investigators apparently vary lely, a close study of the recorded observations indicates that in the

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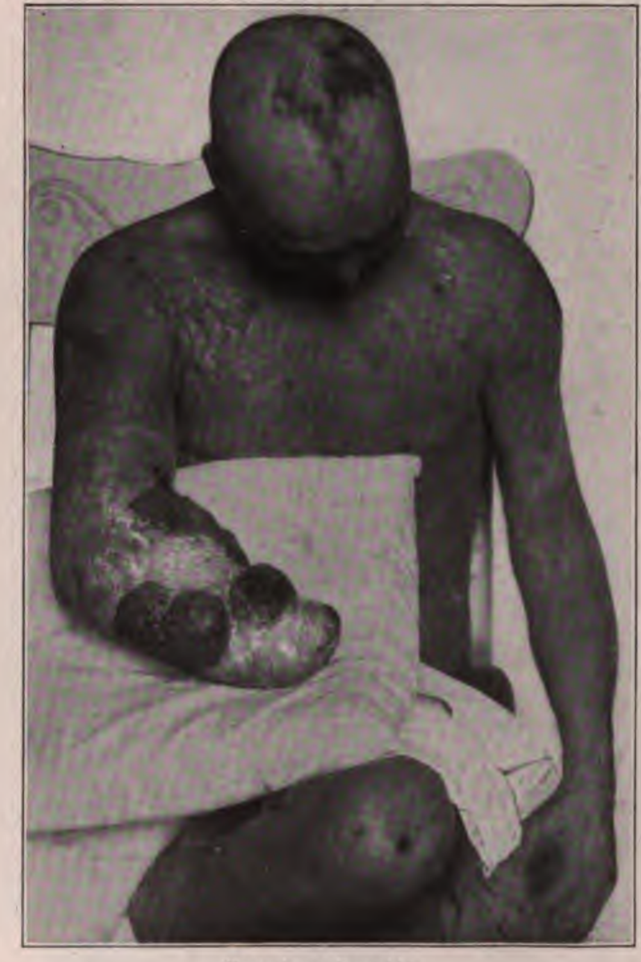
Jour. Cut. Dis., 1919, xxxvii, p. 1. The Association of Certain Cutaneous Lesions h Diseases of the Hemopoietic System.

For recent work in histopathology and review of the literature, see Knowles, Jour. . Dis., 1915, xxxiii, p. 563; "Histopathology of Mycosis Fungoides." Fraser, Ibid., 7, xxxv, p. 793. Mackay and Boyd, Ibid., 1918, xxxvi, p. 520; "A Contribution he Pathology of Mycosis Fungoides."

main there is agreement. Galloway and MacLeod[1] described in the early stages a connective-tissue cell infiltration, not only about the vessels but also about the hair follicles, the sebaceous glands, the muscles of the hair pouch, the ducts of the coil glands, and occasionally in the lymphatic spaces between the connective-tissue fibers. They concluded that the infiltration may originate in the cells of any of these structures. The infiltration in some instances is diffuse but sharply separated from the deeper portions of the corium by a horizontal line and from the rete above by a narrow layer of connective tissue. Where the cells are most compact they are supported by a very deli-

Fig. 323



Granuloma fungoides.

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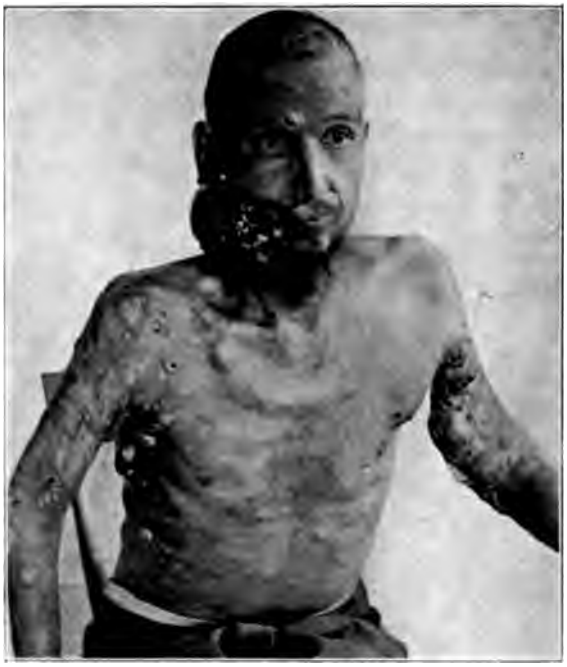
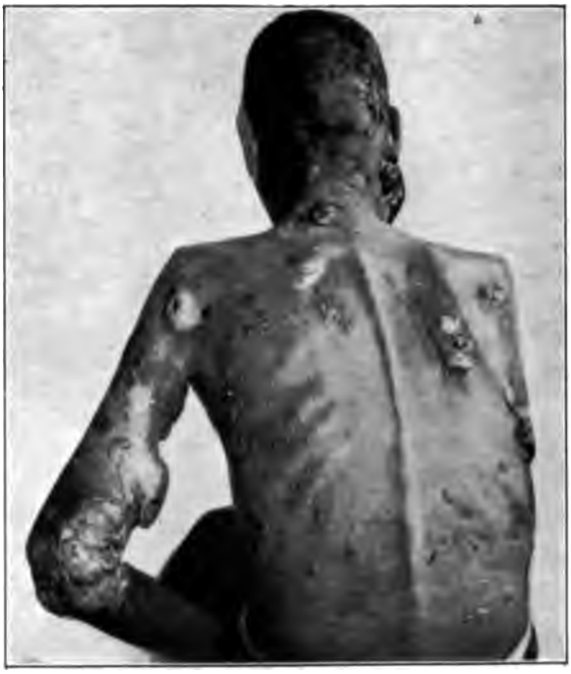
[1] Brit. Jour. Derm., 1900, xii, p. 153 and 187.

Fig. 324



Granuloma fungoides. (Gue.)

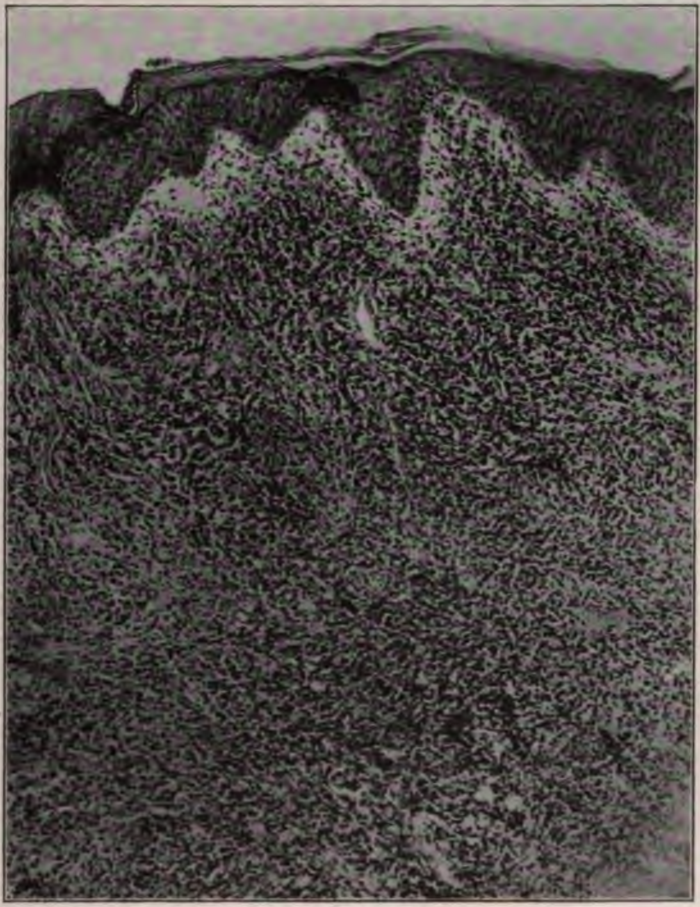
Fig. 325



Granuloma fungoides. (Gue.)

cate fibrous structure made up in part of elastin. Degeneration of the collagen and elastin occurs in the late but not in the early stages of the disease. The cells forming the infiltration are chiefly of the connective-tissue type, but in many cases they and their nuclei show the greatest diversity in size, shape and staining qualities. This multiformity of the cells is apparently characteristic of the disease. The types described by Galloway and MacLeod are, (1) large, oval, fusiform or roundish connective-tissue cells containing a granular protoplasm and large nuclei, and early show mytotic figures but later undergo amytotic division; (2) small round cells with nuclei similar to the first type.

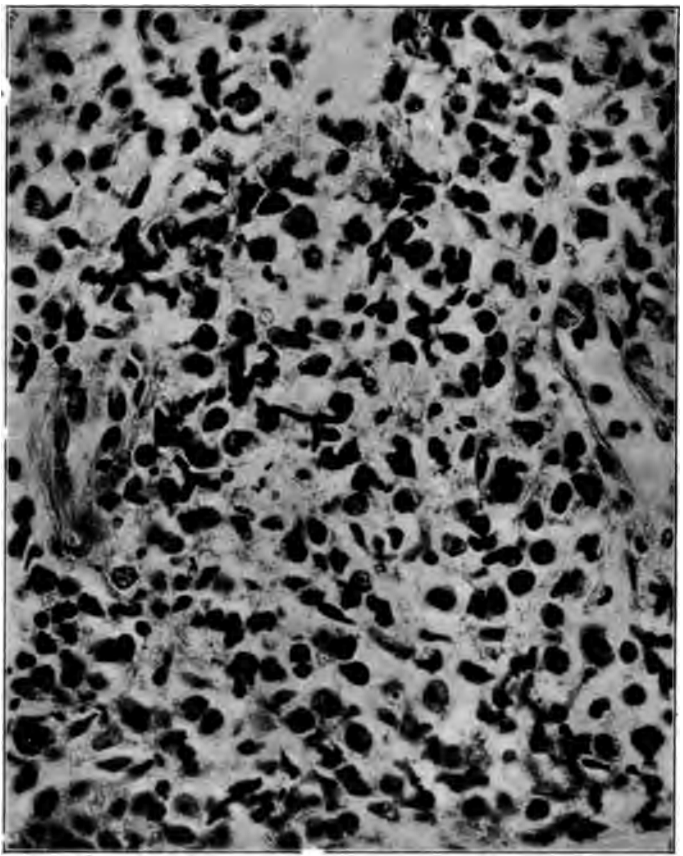
Fig. 326



Granuloma fungoides. (C. J. White.) The section shows absence of the stratum granulosum and parakeratosis of the horny layer, and also character of the cellular invasion and absence of all structures in the corium except bloodvessels and cells.

These were somewhat larger than leukocytes, varied slightly in shape and were the products of the rapid amytotic division of cells of the first type; (3) mast cells varying in size and shape but comparatively not increased in number; (4) a few plasma cells of the large type and (5) finally, imperfect giant cells containing eight to ten nuclei.

Fig. 327



Granuloma fungoides. (C. J. White.) Section of lesion from pubic region showing the nature of the cellular growth—*i. e.*, lymphocytes, large and small; also the minimum amount of collagen present and the dilatation of the vessels. (Histologically, a round-cell sarcoma.)

In the tumor stage they found a continued proliferation of the cells as described above with the additional tendency for them to break down. This was shown by their crenation, irregularity and fragmentation.

The epidermal changes consist in down-growth of the interepithelial processes, the cells showing active mytosis. Insets of corium tissue occurred in the rete. An interepithelial edema with the formation of reticular spaces, and in cases where scaling was present, a condition of parakeratosis was noted.

Hazen[1] states that the cells are of one type and in his case they were lymphoid. Furthermore, he found pathological changes in macroscopically normal skin.

Wolters, in summing up the results of all investigations, states that no specific changes peculiar to the disease have been recognized.

Tryb[2] says the cells in the early stages are of the connective-tissue type; later, there appear the transitional forms or connective-tissue round cells, chiefly of lymphoid variety, with horseshoe-shaped nuclei; and finally plasma cells in the third stage. He regards the disease as an inflammatory process that may, in the later stage, become a plasnoma. MacCormac[3] describes cells of indeterminate character, round, oval or irregular in shape, which were possibly lymphocytes and fibroblasts, and also a few plasma cells. Stelwagon[4] in his study, found the tumor cells to be lymphoid in type. Many of the tumors correspond closely in structure to sarcoma;[5] others show the histological formation of the granulomata. Bosellini[6] concludes that the disease represents a type of infectious granuloma of the skin, with visceral lymphoid changes. Nanti[7] regards the disease as an inflammatory cutaneous disorder, rather than neoplastic, approaching the granulomata in type.

Numerous microörganisms have been seen in the blood and tissues and some have been cultivated, but none has been demonstrated to have any pathogenic relation to the disease. Among them may be named streptococci in the capillaries of granulation-nodules, and staphylococci in cultures from blood. Other examinations of blood, of infected tissue, and of tumors have been wholly negative as to the discovery of causative organisms. Wolters agrees with other observers that the organisms recognized and cultivated are in general the result of secondary infections, and bear no etiological relation to the disorder.

The disease has been considered a form of sarcoma. The fact, however, that the fully developed tumor disappears spontaneously, and that in but few instances has involvement of the viscera been reported in granuloma fungoides, agree against the claim. The autopsy in the severe case pictured in Figs. 322 and 323 showed no visceral involvement. Changes in the deeper organs have been found similar to those that occur in leukemia and pseudoleukemia, but no definite relations have been recognized between these conditions and the disease under consideration. Pardee and Zeit[1] recorded a case terminating in lymphatic leukemia. Other examples are recorded of the coexistence of granuloma fungoides and leukemia.[2]

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[1] Loc. cit.

[2] Archiv, 1912, cxiv, p. 571.

[3] Brit. Jour. Derm., 1913, xxv, p. 415; histology in a case presented by Pringle before the Royal Society of Medicine, London.

[4] Loc. cit.

[5] Bowen, Jour. Cut. Dis., 1897, xv, p. 65. Breakey, Ibid., 1902, xx, p. 316; Autopsy and histological report; tumor-cells described as lymphosarcomatous.

[6] Archiv, 1911, cviii, p. 83; A careful clinical, hematological, and histological study of six cases.

[7] Annales, 1912, p. 572—three papers, October, November and December: Etude des Lymphodermies et des Myélodermies. Extensive review of cutaneous leukemia and its association with granuloma fungoides.

Various investigators have considered the disease to be a granuloma, a sarcoma, a disease *sui generis* midway between adenomatosis or granulomatosis and sarcomatosis and lymphoma.

Mackay and Boyd[3] found cells in the axillary glands that resembled the Hassel corpuscles of the thymus. They were flattened, elongated, and presented marked whorled or concentric arrangement.

While the disease, as a rule, attacks the skin alone, the internal organs occasionally become involved. Paltauf and Zumbusch[4] found in 2 cases involvement of the mucous membrane, general adenopathy and nodules in the viscera, and regard the disease as an infectious process.

In a study of the early lesions Hyde and Montgomery[5] found dilatation of the vessels and lymph capillaries often with some endothelial proliferation and a more or less dense cellular infiltration, which was limited usually to the upper part of the corium, except where it surrounded some of the deeper vessels in the form of sheaths.

Diagnosis.—In the early stages the disorder has to be distinguished from eczema, psoriasis, urticaria, multiform erythema, dermatitis exfoliativa and parapsoriasis. In some instances an immediate diagnosis is impossible, and a period of observation is necessary. In eczema the lesions will probably be more multiform and the weeping more evident. Psoriasis is, as a rule, unaccompanied by itching, the latter symptom usually being marked in psoriasiform complications of the disease under consideration. Urticaria is ordinarily more evanescent and there may be some discoverable etiological factor. While multiform erythema is simulated, it is usually not difficult to differentiate. In the cases beginning as dermatitis exfoliativa, observation for a certain time is essential, unless diagnosis is made by histological examination. The same is true of those cases resembling parapsoriasis. Wise says that the resistance to treatment in the latter disorder and its characteristic histology are the only distinguishing features. The infiltrated area, nodules and small tumors may at times closely simulate leprosy, but the history, the absence of areas of anesthesia, absence of nerve involvement and the histological examination, should clear up the diagnosis without difficulty. In the tumor stage little difficulty occurs other than the differentiation of the tumors from sarcoma, which is largely accomplished by the history and career of the tumors of granuloma fungoides and their final formation of characteristic fungoid, superficial, ulcerating masses.

Gaucher and Guggenheim[6] attempted to test the specificity of the complement-fixation test in the prefungoid stage of granuloma fungoides. They used as antigen triturated tissue of the disease. In the first case, the test was negative and the patient made a rapid recovery; in the second, the reaction was positive and the patient died later of bronchial pneumonia. The histology in each case was described as typical for the disease.

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[1] Jour. Cut. Dis., 1911, xxix, p. 7.

[2] Leviseur, Ibid., p. 556.

[3] Loc. cit.

[4] Archiv, 1914, cxvii, p. 679.

[5] Jour. Cut. Dis., 1899, xvii, p. 253.

[6] Soc. méd. de Hôp., 1912, p. 151.

Treatment.—Relief from symptoms is the only result to be obtained from treatment. The internal management depends upon the stage of the disorder. As a rule, no internal treatment is indicated early. Arsenic in full doses and by hypodermic injection has been of apparent service. Köbner reported 1 case cured by this treatment. We have treated several cases with arsphenamin. Temporary improvement occurred after each injection; no appreciable effect was noted in 1 instance and all the cases terminated fatally, as usual. Foster,[1] Foerster,[2] Fordyce,[3] Kingsbury,[4] and others, record similar results. Coley's fluid has been beneficial in certain instances. Gottheil[5] records such a case. Radiotherapy offers the best hope of relief. This agent was employed for five years by the author in a case of granuloma fungoides in both the prefungoid and tumor stages, with excellent results. The early plaques disappeared and the patient remained free from symptoms of the disease for months at a time. The later and more markedly infiltrated areas in tumor forms also readily responded. While each return of symptoms was successfully combated over a long period, eventually the patient succumbed. In all our patients so treated much improvement occurred, but the recurrence was the rule. In some cases lesions would develop in new areas more rapidly than they could be removed. The relief from itching, healing of foul, discharging ulcers, and the removal of fungating masses by this method is gratifying, even though relief be only temporary in most instances. Lesions involving the tongue[6] as well as those on the cutaneous surface, may be thus successfully improved.

Local treatment otherwise may be employed as found necessary. Ichthyol and salicylic acid or ammoniated mercury may be used in ointments in the early stages, or any of the various soothing and antipruritic applications described in the section on the treatment of eczema may be employed according to the indications in the individual cases. In the late stages the ulcerating masses require wet antiseptic dressings of boric acid or aluminum acetate. General baths are of great service when the disorder is extensive. These may contain mercuric chlorid in a strength of 1 to 10,000 or 1 to 20,000, or boric acid, or biborate or bicarbonate of sodium. It is occasionally necessary to use resorcin, thymol, boric acid, phenol, or some similar preparation in solutions or otherwise to overcome the fetor arising from the lesions.

Extirpation of the fungating masses has been practised, but without much benefit.

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[1] Jour. Cut. Dis., 1909, xxvii, p. 75.

[2] Personal communication.

[3] Jour. Cut. Dis., 1912, xxx, p. 683.

[4] Ibid., 1913, xxxi, p. 417.

[5] Progressive Med., September, 1909, p. 127.

[6] MacKee, Jour. Cut. Dis., 1919, xxxvii, p. 253.

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[4] Ibid., 1913, xxxi, p. 417.

[5] Progressive Med., September, 1909, p. 127.

[6] MacKee, Jour. Cut. Dis., 1919, xxxvii, p. 253.

Prognosis.—The prognosis is unfavorable except as to the prolongation of life by radiotherapy. The patient may survive after the beginning of the disease for from a few months to many years, the average duration being two to four years. After the development of tumors the patient may live for a few months, or, at the most, two or three years. A few cases of recovery are on record, one of the patients being relieved after an attack of erysipelas.

LEUKEMIA CUTIS.[1]

Cutaneous and subcutaneous lesions of various kinds occur occasionally in association with the general disease termed leukemia. They are found both in acute and chronic types of the disorder, as well as in the different varieties, lymphatic and myeloid.

Symptoms.—In acute lymphatic leukemia hemorrhages, both petechial and diffuse, occurring in the skin and mucous membranes are frequently noted, as are also ulcerations and necroses; the latter commonly in the mucous membranes of the mouth and nose, less often in the skin. In chronic lymphatic leukemia lymphomatous nodules and tumors are found in the skin and subcutaneous tissues as well as in the mucous membranes. In myeloid leukemia these manifestations may occur, but are less frequent than in the other variety. The skin in the latter variety is usually dry, lusterless, anemic; and may be the seat of a symptomatic erythema and urticaria, with secondary complications, and occasionally true lymphomatous tumors. Rolleston and Fox[2] describe nodular infiltrations in an atypical example of the myeloid variety.

In general, the cutaneous manifestation may appear to precede or follow the systemic disease, but at any time they are symptomatic of a grave disease of the blood. While the general symptom complex is often sufficient to place correctly the varieties of this disease from a clinical standpoint, the final diagnosis rests on the microscopic blood findings, the latter method being imperative in clinically mixed types. It is evident from a study of the literature that certain cases show characteristics that appear to be closely allied to granuloma fungoides[1] and sarcoma cutis; and opinions differ concerning their

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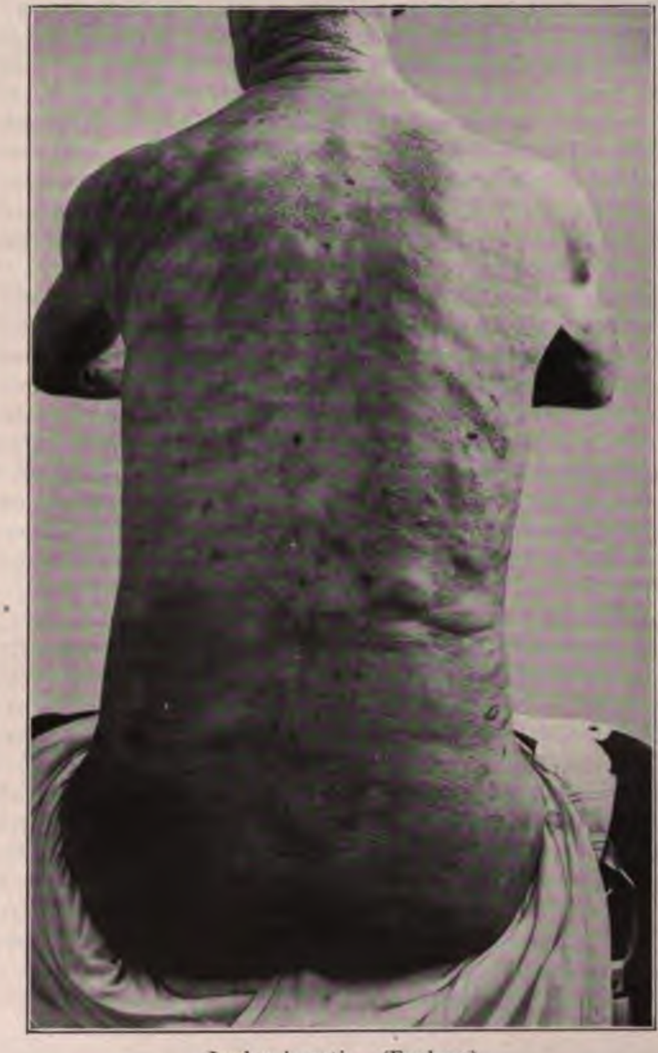
[1] LITERATURE: The following works, with articles referred to in their appended references, were consulted in the preparation of this article, in addition to special papers mentioned below: Nothnagel's Encyclopedia of Practical Medicine, American edition, 1905, volume devoted to diseases of the blood, pp. 539–637 (many references); Unna, Histopathology, pp. 618–624 (many references); Crocker, 3d ed., pp. 1036–1042; Nicolau: A Contribution to the Clinical and Histological Study of the Cutaneous Manifestations of Leukemia and Pseudoleukemia (Annales, 1904, p. 753, abstr. Brit. Jour. Derm., 1905, xvii, pp. 234 and 235); Wende: Leukemic Lesions of the Skin (Jour. Cut. Dis., 1901, xix, p. 479); Shattuck: A Case of Lymphatic Leukemia with Purpura (Jour. Cut. Dis., 1904, xxii, p. 118); Bosellini: A critical study of cutaneous manifestations found in leukemia, pseudoleukemia, and allied processes, with a clinical, hematological, and histological study of granuloma fungoides, and bibliography (Archiv, 1911, cviii, p. 83); Nanta: A study of the lymphodermias and the myelodermias (Annales, 1912, iii, p. 572—three papers, October, November and December, 1912); idem, ibid., 1914, v, p. 19: Two new cases of lymphodermia (in these articles an extensive study is made of the superficial and deeper lesions which occur in the lymphatic diseases); Fimmen: Lymphatic leukemia with cutaneous tumors (Zeitschrift, 1913, xix, No. 8, p. 705); Kreibich: Skin changes in pseudoleukemia and leukosarcomatosis (Archiv, 1908, lxxxix, p. 43); Bernhardt: Leukemia of the skin, a study of seven cases (Archiv, 1914, p. 17). Also see Batty and Loughlin, Brit. Jour. Derm., 1917, xxix, p. 36; Leukocythemia Cutis. Elliot, Jour. Michigan State Med. Soc., January, 1918, Leukemia Cutis.

[2] Brit. Jour. Derm., 1909, xxi, p. 377: A Case of Atypical Myeloid Leukemia, with Nodular Infiltration of the Skin (A clinical and histological study, with discussion. Five good cuts, one clinical and four histological).

FIG. 328



Leukemia cutis. (Fordyce.)

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[1] Pardee and Zeit, Jour. Cut. Dis., 1911, xxix, p. 7; Leviseur, ibid., p. 556; and others.

nosological position, as to whether they represent a single disease or a combination of two of the above disorders.

There are two types of cutaneous lesions: a superficial and a deep variety. Both may occur in the same patient, and a division is made merely for purposes of description. In the first are hemorrhages, petechial and diffuse; papular, vesicular, urticarial, and pigmented lesions; symptomatic erythema; diffuse, scaling erythrodermia; and, in rare instances, a moist or scaling dermatitis accompanied by itching.[1] Whitehouse[2] demonstrated a patient with lymphatic leukemia before the New York Dermatological Society, who had a generalized cutaneous eruption resembling dermatitis herpetiformis. The histology of the lesions showed leukemic infiltration. A similar case has been recorded by Parounagian.[3] Among the deeper lesions are ulcers, and areas of necrosis, especially of the mucous membranes, but also of the skin, induced by the breaking down of hemorrhagic or lymphomatous deposits.

Nodules and tumors of various sizes, shapes, and colorations also occur. All these lesions may develop on different parts of the body, but show a predilection for the extremities and face. The nodules vary in size up to that of a coffee bean, may be few or numerous, and occur especially on the extremities and face. They may be pale and waxy, reddish, brownish-red, or yellowish-red in color, firm or soft in consistency, movable with the skin, smooth or scale covered, oval, round or flat, or even have depressed centers, and may be often accompanied by telangiectatic vessels. When abundant on the face, especially in association with larger growths, a leonine expression may be exhibited.

The tumors vary in size up to that of a hen's egg or larger, and, like the nodules, may be few or numerous. They grow slowly but continuously, as a rule, and only exceptionally break down. Winfield[4] described multiple lymphoid tumors of the skin not associated with leukemia, and cites a similar recorded case in which at a later date leukemia developed.

Arndt[5] says the old names, lymphatic and myeloid leukemia, should be replaced by aleukemic, subleukemic, and leukemic lymphadenosis and myelosis. In the aleukemic form the blood is normal. In the subleukemic form the blood shows a relatively normal leukocytic count, but the proportion of lymphocytes is increased; while in the leukemic form the blood shows an increased number of leukocytes, with progressive increase of lymphocytes.

Under the title of *lymphadenids* (*leucémide*, Audry) are classed the cutaneous eruptions occurring with the disorder that are not characteristic in themselves, either clinically or histologically, and which occur in other diseases. These lesions may be purpuric (frequently terminating in necrosis), prurigo-like papules or papulovesicles; or the skin may present only the features of a traumatic dermatitis, due to scratching induced by severe itching; and, finally, universal exfoliative erythrodermia.[1]

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[1] Hasen, Jour. Cut. Dis., p. 521 (A report of three cases and extensive bibliography. One case presented marked hyperpigmentation, another vesicular lesions). Stillians, Ibid., 1918, xxxvi, p. 245. Scaling Dermatitis in Splenomyelogenous Leukemia.

[2] Ibid., 1912, xxx, p. 683.

[3] Ibid., 1916, xxxix, p. 126. Dermatitis Herpetiformis in Leukemia. Histology by Hermann.

[4] Ibid., 1913, xxxi, p. 246.

[5] Trans. Amer. Med. Assoc., Sec. on Derm., 1914, p. 103.

The lymphadenosis proper of the skin Arndt subdivides into two varieties: the universal and the circumscribed. The former is represented by a universal infiltrating erythrodermia,[2] with characteristic hematological and histological lymphadenotic features. The circumscribed variety is characterized by circumscribed patches and tumors, and may be observed in aleukemic, subleukemic, and leukemic lymphadenosis. The site of predilection of these lesions is the face, though in exceptional cases they may be generally distributed. A case in which leukemic nodules and tumors preceded the characteristic blood changes was recorded by Butler.[3] Atrophy in association with general erythrodermia was recorded in one instance by Wise.[4]

In a small proportion of cases of acute lymphatic leukemia green tumors (chloromata) occur. Children more frequently suffer, and the tumors are usually seen on the face, temples, and cranium. The osseous system is especially affected, the bone-marrow often being replaced by a peculiar greenish mass. The lymphogenous green tumors are deposited in the facial and cranial bones, in or under the periosteum or dura. In addition, practically every bone of the body has been described as the seat of these peculiar green tumors.

Etiology and Pathology.[5]—The cutaneous manifestations above described are a part of the general leukemic process which involves all portions of the human organism. The essential cause is unknown. The leukemic nodules and tumors are situated anatomically in the corium and upper part of the subcutaneous tissue and are made up of accumulations of lymphocytes. The infiltration of these cells is not limited to the nodules and tumors, but occurs throughout the skin, even where no clinical evidence of its presence exists. The process begins as a perivascular lymphocytic infiltration, especially about the coil-glands, and spreads upward toward the epidermis and downward into the subcutaneous tissue. The morbid growth is usually separated from the epidermis by a thin layer of normal corium. While the infiltration may be dense, it spreads around normal structures without destroying them. The lymphvessels in the region are full of lymphocytes, which may mean that these cutaneous lymphomata produce some of the specific cells found in the general circulation. In the cases resembling granuloma fungoides a multicellular infiltration similar to that found in this disease is described. Bruusgaard[1] records two cases with metastatic cutaneous tumors. The histological study revealed lymphocytes, plasma-cells, eosinophiles, and fibroblasts. This picture he interprets as an example of malignant granuloma.

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[1] Recent cases of this type are recorded by Hartzell International Clinics, 1918, iv, p. 88; Flat erythematous elevations, vesicles, bullæ, hemorrhagic lesions and pigmentations. Sequeira, Brit. Jour. Derm., 1919, xxxi, p. 148. Erythrodermia with Lymphatic Leukemia.

[2] Sweitzer, Jour. Amer. Med. Assoc., lxvii, p. 1511. Leukemia Cutis; Aleukemic type; Generalized infiltration.

[3] Archiv of Derm. and Syph., 1920, ii, p. 594; Lymphatic Leukemia with Primary Manifestations of the Skin.

[4] Jour. Cut. Dis., 1917, xxxv, p. 668.

[5] Symmers, Jour. Cut. Dis., 1919, xxxvii, p. 1. The association of certain cutaneous lesions with diseases of the hemopoietic system. See this article for discussion on etiology and pathogenesis.

Treatment.—The treatment is that of the general affection under consideration. Radiotherapy is advised as a temporary alleviative measure. Stengle and Pancoast[2] advise radiotherapy in chronic leukemia over the long bones, and in pseudoleukemia over the glandular enlargements, but say it is contraindicated in acute leukemia. Billings[3] used benzene in connection with radiotherapy with good results. He advises its use with caution and says care must be exercised to procure a pure preparation of the drug. Lawson and Thomas[4] report improvement in a case similarly treated.

Lymphodermia Perniciosa (Kaposi) (Fr., *Érythrodermie mycosique*).—A group of cases is described in which the true leukemic process occurs later, the early manifestations being exhibited as a moist, itching dermatitis, with redness and swelling of the skin, and in which finally nodules and tumors form, with a tendency to break down. These cases resemble in a high degree granuloma fungoides; and Kaposi's case is classed as such by Vidal, Hallopeau, Paltauf, Crocker, and others.

PSEUDOLEUKEMIA CUTIS.[5]

Synonyms.—Lymphadenoma, lymphogranulomatosis.

Symptoms.—The cutaneous manifestations occurring in this disorder correspond closely with those described in connection with lymphatic leukemia, though pruriginous papules and urticaria are of more frequent occurrence. Intense itching may occur without cutaneous changes other than those due to trauma. Pigmentation and scarring sometimes occur in the papular cases. General erythroderma, nodules and tumors occur less often. Ulcers on the leg resembling gummata are reported by Alderson.[6] A case presenting tumor-like infiltrations in the scalp, on the forehead and in the midscapular region was recorded by Fox.[1] Cole[2] suggests the term "*pseudoleukemids*" for the superficial lesions that occur in this disorder which are not characteristic clinically or histologically. This is similar to the "*leucémides*" of Audry, above mentioned in connection with leukemic lesions of like nature. In this group he places the purpuras, prurigo-like lesions, pruritides, urticarias, pigmentations, general erythrodermas and nutritional disturbances of the skin occasionally seen.

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[1] Archiv, 1911, cvi, p. 105; abstr. Jour. Cut. Dis., 1912, xxx, p. 113.

[2] Jour. Amer. Med. Assoc., September 28, 1912, lix, p. 1166.

[3] Ibid., February 15, 1913, lx, p. 495 (with bibliography of benzene therapy).

[4] Ibid., December 13, 1913, lxi, p. 2157.

[5] Literature, Sibley, Brit. Jour. Derm., 1915, 22, vii, p. 52; "Lymphadenoma with Cutaneous Lesions." Microscopic report by a committee of the Dermatological Section of the Royal Society of Medicine, and Corbett, ibid., 1917, xxix, p. 42. Schwarz and Wise, Jour. Cut. Dis., 1918, xxxvi, p. 435; "Hodgkin's Disease." Weber and Dove, Brit. Jour. Derm., 1918, xxx, p. 15, "A Case of Pruritus in Hodgkin's Disease (Lymphogranulomatosis pruriginosa)." MacLeod, ibid., p. 165; "Hodgkin's Disease with Prurigo." Levin, Jour. Cut. Dis., 1919, xxxvii, p. 635; "Hodgkin's Disease with Prurigo-like Papules, Pigmentation and Scars." Trimble, Jour. Cut. Dis., 1919, xxxvii, p. 336; Pseudoleukemia Cutis case report.

[6] Jour. Cut. Dis., 1917, xxxv, p. 481; "Cutaneous Metastases in Hodgkin's Disease."

Etiology and Pathology.—The cause of pseudoleukemia has not been discovered. Cole, and others quoted by him, believe that about 15 per cent. present cutaneous symptoms. A diphtheroid bacillus has been isolated from lesions in the disorder which was thought to be important etiologically.[3]

The histology of the pruriginous papule studied by Bowen[4] showed the structure of the papules of prurigo. In a metastatic tumor Brusgaard[5] found lymphocytes, plasma cells, eosinophiles and fibroblasts. Symmers[6] states that the pseudoleukemic deposits in the skin are similar to those seen in lymph nodes and viscera and consist of various numbers of lymphoid and plasma cells and multinucleated giant cells of the myeloid or bone-marrow type situated in a fibroblastic stroma.

Treatment.—Treatment has been of little avail. Improvement occurred in cases treated by Billings and Rosenow with a vaccine prepared from the above-mentioned diphtheroid bacillus.

SARCOMA CUTIS.

Definition.—Sarcoma of the skin is a rare disorder characterized by connective-tissue new growths.

A satisfactory grouping of sarcomata is not possible. The one followed here was suggested by de Amicis.[7] The three types are the following: (1) cutaneous sarcoma devoid of pigment and occurring as single or multiple lesions; (2) cutaneous melanotic sarcoma and (3) cutaneous multiple pigmented or hemorrhagic sarcoma.

Non-pigmented Sarcoma Cutis.—The lesions in this type may be single or multiple. The localized single tumor may develop on or beneath the skin, may follow an injury, or arise in the site of a nevus, scar or other cutaneous lesion. It may occur as a cutaneous or subcutaneous nodule or tumor, present a smooth surface and be elevated in varying degrees, and may be of the normal color of the skin or pale bluish or purplish-red. When admixed with vascular tissue it may be comparatively soft. When deeply situated it is indurated but does not present the hardness that characterizes the carcinomata. These lesions may grow slowly or rapidly and ulceration may occur and a fungoid process result. Usually, new lesions develop in the vicinity or in other regions, and these may be present in moderate number, or there may be hundreds of such tumors. The multiple lesions are small, firm, elastic and freely movable under the skin or adherent to it, and are of the normal color of the skin, bluish or dark-red, or purplish and at times are accompanied by telangiectasia. They vary in size from a pea to a walnut, a hen's egg, or larger, and only exceptionally ulcerate. At times, these tumors occur as the result of an internal

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[1] Archiv. of Derm. and Syph., 1920, ii, p. 578; Lymphogranulomatosis of the Skin in Hodgkin's Disease. Case report with histology and discussion of recorded cases.

[2] Trans., Sec. on Derm., Amer. Med. Assoc., 1917, p. 192; "The Cutaneous Manifestations of Hodgkin's Disease (Lymphogranulomatosis;" report of thirteen cases, discussion and classification; literature.

[3] Bunting and Yates, Arch. Int. Med., August, 1913, p. 236; and Jour. Amer. Med. Assoc., November 15, 1913, p. 1803. Negri and Mieremet, Centralbl. f. Bakteriol., 1913, lxvii, p. 292. Fränkel and Much, Zeitschr. f. Hygiene, 1910, lxvii, p. 159. Billings and Rosenow, Jour. Amer. Med. Assoc., 1913, lxi, pp. 21 and 22 (*Cf.* report for special technic for growing the organism).

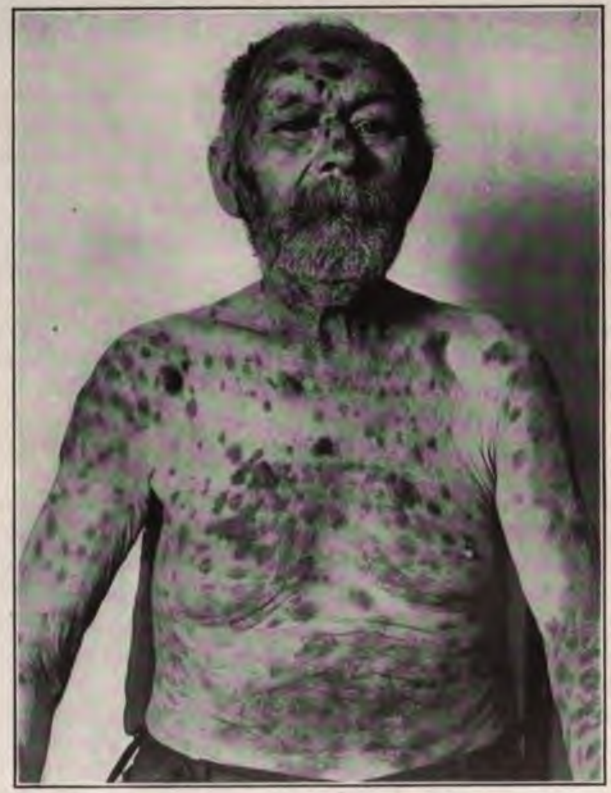
[4] Jour. Cut. Dis., 1897, xv, p. 72.

[5] Archiv, 1911, cvi, p. 105; abstr., Jour. Cut. Dis., 1912, xxx, p. 113.

[6] Jour. Cut. Dis., 1919, xxxvii, p. 1.

[7] Trans. XII Internat. Cong. of Med., Moscow, 1897; abstr., Brit. Jour. Derm., 1897, ix, p. 440.

Fig. 329



Sarcomatosis with cutaneous, conjunctival and membranous hemorrhages. (Dr. Potthoff's case.)

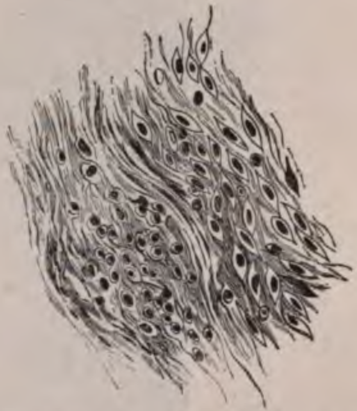
sarcoma, in which case they may be generally disseminated. The course of unpigmented sarcoma, whether single or multiple, may be slow or rapid. Usually new lesions continue to develop and general internal involvement occurs, resulting in a fatal termination in six months to two years. In some cases of generalized sarcomatosis of the skin it is difficult to determine whether the cutaneous tumors are primary or consecutive to internal involvement. In either case the termination is the same. Multiple sarcomata may in rare instances be congenital. Such a case is described by Crocker.[1]

Fibrosarcoma may occur as a single lesion,[2] or multiple tumors[3] may develop in the course of years. It is characterized by its slow growth and lack of malignancy.

Recurrent Fibroid of the Skin (Hutchinson) begins usually in the lower extremities and grows slowly at first. It recurs rapidly after removal and produces fungous tumors. Generalization occurs followed by a fatal termination. Crocker[4] describes this as a rare form of spindle-cell sarcoma.

Etiology and Pathology. — The cause is unknown. These new growths frequently have their origin in the site of a nevus scar or other cutaneous lesion. They usually occur earlier in life than the carcinomata. According to Johnston,[5] the growths are composed of variously shaped cells held together by a fibrillary network of connective tissue. This reticulum, he states, is the product of cellular activity, and is characteristic of the sarcomata. The cells may be large or small, round or fusiform. Giant cells may occasionally be seen with other types, but if present in any number indicate a secondary growth from the bone in most instances. Occasionally primary giant cell tumor growths have been described, examples of which are recorded by Unna, Perrin and Lereddé,[6] Zeisler, J. and E. P.,[7] and others. Fibrosarcoma are less malignant and are composed of fusiform cells with a greater amount of fibrous tissue.

FIG. 330



Sarcoma. Spindle-cells visible in sections of cutaneous nodule removed from a sarcomatous patient. (About × 300.)

Diagnosis.—General sarcomatosis at times closely resembles granuloma fungoides, many examples being on record in which neither a clinical nor histological study showed a distinction.[1] The tumors occurring in association with leukemia and pseudoleukemia also closely resemble those of this group. They have not infrequently been described as general sarcomatosis occurring with leukemia. In the latter disorder the blood examination is of importance, but in aleukemic cases a histological examination would have to be made. In rare instances fibromata and lepra are also to be distinguished.

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[1] Diseases of the Skin, p. 1025.

[2] Johnston, Jour. Cut. Dis., 1903, xxi, p. 23.

[3] Ionides, Brit. Jour. Derm., 1869, viii, p. 439.

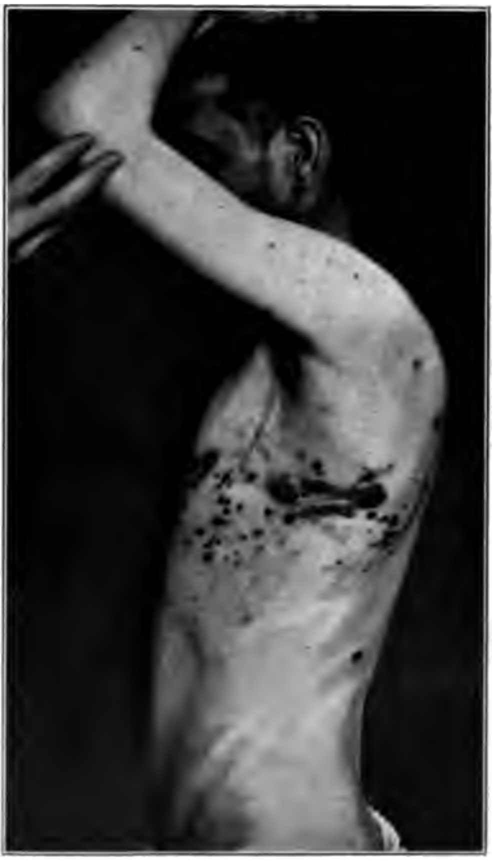
[4] Diseases of the Skin, p. 1023.

[5] Brit. Jour. Derm., 1901, xiii, p. 241.

[6] Annales, November, 1895; abstr., Brit. Jour. Derm., 1896, viii, p. 147; Generalized idiopathic sarcoma with giant-cells. This case did not appear to be metastatic.

[7] Jour. Amer. Med. Assoc., 1917, lxix, p. 111.

FIG. 331



Melanotic sarcoma. (Fordyce.)

Prognosis and Treatment.—The prognosis in fibrosarcoma is the least grave. Recovery after surgical extirpation usually occurs. In the single tumors above described recovery sometimes follows surgical removal. Not infrequently, however, recurrence takes place, rapidly followed by metastasis. If surgical removal is employed the operation should include tissue far beyond the supposed limit of the growth.

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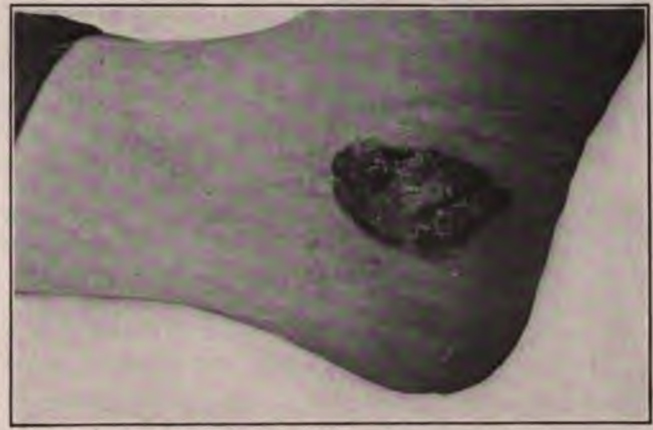
[1] Bowen, Jour. Cut. Dis., 1897, xv, p. 65.

Surgical treatment cannot be employed in generalized cases. In some varieties of these the patient may live for many years, while in others a rapidly fatal result occurs. Arsenic, as originally suggested by Köbner and Sherwell[1] is occasionally of value. Mixed toxins of erysipelas streptococcus and *Bacillus prodigiosus* (Coley's fluid) have been of some service. Radiotherapy will remove individual lesions, but the treatment cannot be said to be curative.

Melanotic Sarcoma (*Nevocarcinoma, Nevomelanoma, Melanoma*).—The disorder described under the above titles is the most malignant and rapidly fatal of the cutaneous sarcomata.

Symptoms.—The chief clinical characteristics of the affection are its frequent development from the mole or pigmented spot; the production of pigment; the marked regional and general metastases; and its malignancy. As a rule, the first lesion noted is a pigmented spot or a mole undergoing some change. In some cases, however, no such preliminary lesion is noted. When beginning in a mole, the latter increases in size; assumes a darker color; not infrequently ulcerates, the ulceration occasionally healing to a degree; or it may become surrounded by an area of deep pigmentation. These changes may occur independently or result from the irritation induced by trauma or caustics. When occurring in a black spot (*Hutchinson's malignant lentigo*), such as frequently develops on the foot, the lesion becomes elevated, and in the neighborhood new lesions, which may be macules, papules, or tubercles, all of a black color, develop. Not infrequently these occur subsequent to the operative removal of the primary lesion. The epidermis may be thinned but intact, fissured and hemorrhagic, or ulcerated. The floor of the ulcer is

FIG. 332



Pigmented or nevocarcinoma.

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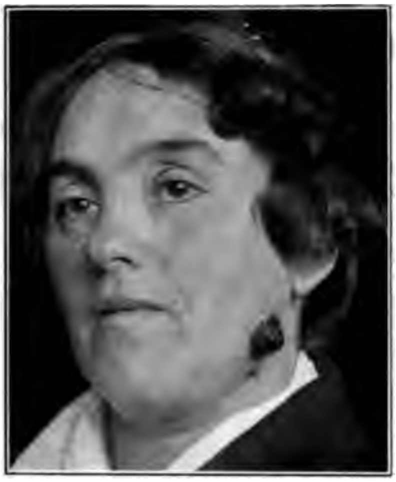
[1] Amer. Jour. Med. Sc., October, 1892.

black or brownish-black in color, and appears as though filled with ink, secreting a black, hemorrhagic fluid. As a rule, metastatic growths appear early.

Fig. 333

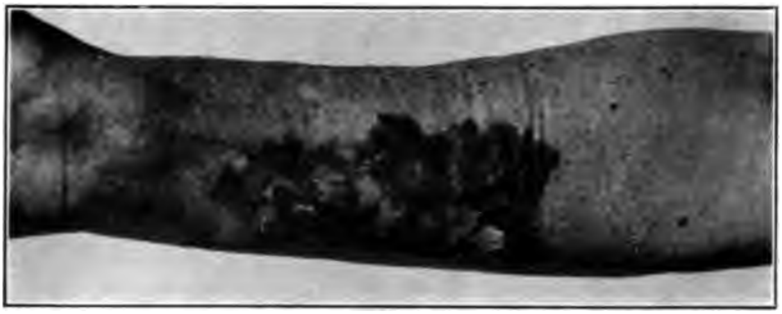


Melanotic sarcoma.

The lesion or lesions may remain stationary for a long time or they may be followed rapidly, (which is the rule) by generalization, as the result of local irritation or otherwise, producing lesions in the neighborhood; when near, by way of the lymphatics, and at a distance by way of the blood. Hartzell[1] recorded a case of two years' duration, apparently benign, showing the typical structure of pigmented sarcoma.

The disseminated cutaneous lesions are bean- to egg-sized, firm or doughy in consistency, spherical, lobulated, sessile or pedunculated, and vary in color from grayish-brown to inky-black. The lesions, as a rule, begin in the skin of the face and extremities, particularly about the foot. A common site, also, is in one of the pigmented structures of the eye. The lymphatic glands adjacent to the primary growth usually enlarge at an early date, and on microscopic examination show pigment- and tumor-cells similar to those of the original growth. In case they break down and ulcerate, they exhibit ulcers similar to those described above. The pigment present varies greatly in amount in different cases and in different tumors of a given case. The tumors may be of the normal color of the skin, or light-brown,

Fig. 334



Nevocarcinoma recurring after excision. No metastases present. (View of flexor surface of forearm.)

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[1] Jour. Cut. Dis., 1893, xi, p. 13.

brown, and even jet-black. In extreme cases streaks of black pigment may be seen extending from the primary lesion to the nearest lymphatic glands, or extensive areas of pigmentation may occur in the skin at some distant point. In others, by extension through the blood and involvement of the vascular walls, a general bronzing may occur. Pigment is usually present to a marked degree in the lymphatic glands and metastatic tumors. This may even follow what appears to be a non-pigmented primary lesion. In certain cases the pigment may be sufficient to cause melanemia and melanuria.

The course of the disorder varies, though it is usual for it to be rapidly malignant. A small, insignificant primary lesion may be followed within a short time by general visceral metastatic involvement, which proves rapidly fatal. It is possible that cases of this type, in which the primary lesion has passed comparatively unnoticed, are those in which the disease is supposed to have originated in lymphatic glands.[1] In the *melanotic whitlow of Hutchinson*[2] there occurs a chronic onychia, with the nail-fold swollen. Early there occur pigmented spots or streaks, suggesting silver-nitrate stains, at the edge of the nail-fold. In a short time ulceration occurs and a fungous tumor develops. Hyperpigmentation supervenes, the nail is exfoliated, and the process becomes generalized.

Pathology.—The pathogenesis of this growth is not clear. Early opinion placed the sarcomata as arising from some part of the connective tissue of the corium, the melanotic variety being distinguished by cells showing hyperpigmentation. Von Recklinghausen believed they originated in the endothelium of lymphatic vessels. Ribbert[3] believed the chromaphores, which were thought to be connective-tissue cells, the point of origin. Johnston[4] concludes that the commonest, and therefore the most important, melanomata are derived from soft nevi, which are endotheliomata of lymphvessel origin. Unna,[5] Gilchrist,[6] Schalek,[7] Whitfield,[8] and others[9] believe the growths are of epithelial origin, and to these growths the title nevocarcinoma is applied. Fox, W. S.,[10] concludes that the major portion of nevomelanoma are nevocarcinoma. Fordyce[11] believes there may be a twofold origin for nevi, and would class the pigmented malignant growths for the present as melanomata.

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[1] Eve, Practitioner, 1903, p. 165.

[2] Arch. of Surg., iii, p. 519.

[3] Geschwulstlehre, p. 225; quoted by Fordyce, Jour. Amer. Med. Assoc., 1910, liv, p. 91.

[4] Jour. Cut. Dis., 1905, xxiii, pp. 1 and 49 (Melanoma: an extensive study of pigmented tumors, with bibliography).

[5] Berlin klin. Wochenschr., 1903, xxx, p. 14.

[6] Jour. Cut. Dis., 1899, xvii, p. 117 (investigation of two cases and of several pigmented moles; bibliography).

[7] Ibid., 1900, xviii, p. 145 (histological study of five cases, with review of literature).

[8] Brit. Jour. Derm., 1900, xii, p. 267 (two cases, with references).

[9] Whitehead, Johns Hopkins Hosp. Bull., 1900, xi, p. 221 (two cases, with review of literature).

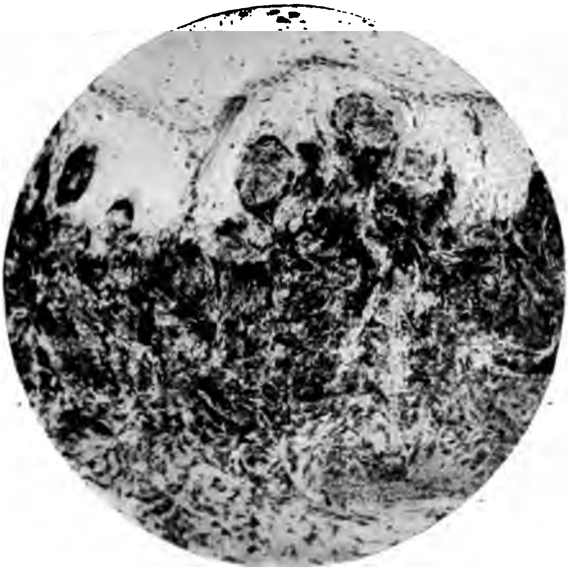
[10] Brit. Jour. Derm., 1906, xviii, pp. 1, 47 and 83.

[11] Jour. Amer. Med. Assoc., 1910, liv, p. 91 (Melanoma and some types of sarcoma of the skin).

The histology varies in different cases and in different parts of the same tumor; there being in some an alveolar arrangement of large or small cells in one part, and bundles of spindle or branched and stellated cells in another. The spindle-cells occur most frequently. The pigment, which is melanin, also varies. In some cases the component parts of the cell can hardly be distinguished owing to the dense pigmentation. In other cells no pigment whatever can be discovered.

Diagnosis, Prognosis, and Treatment.—The recognition of melanotic sarcoma is usually not difficult. An irritated and growing pigmented nevus should at once be suspected as the beginning of a series of grave events. Occasionally, this is insignificant and the disease has advanced beyond control before its nature is suspected. Early and complete removal may prevent dissemination, but frequently immediate generalization follows the removal of the local lesion; or recurrence may be delayed for several months.

FIG. 335



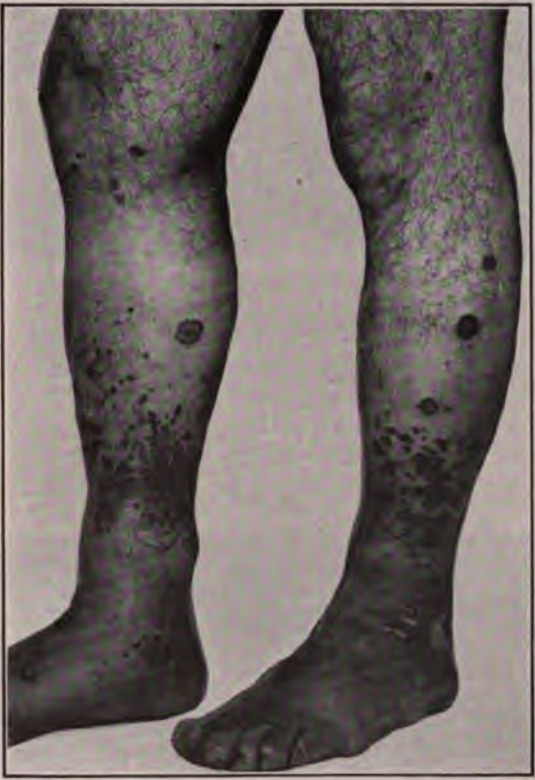
Melanoma of the foot. Zeiss 16 mm., C. O. 4. Showing the tumor situated directly beneath the epidermis and its extensive pigmentation. Clumps of chromatophores and pigment are also seen in the epidermis. (Fordyce.)

Arsenic has no influence on the progress of the disease, and other measures are usually equally inefficient.

Idiopathic Multiple Hemorrhagic Sarcoma (Idiopathic Multiple Pigment-sarcoma (Kaposi)).—This disease was originally described by Kaposi in 1897 under the second of the above titles. It is rare, but a number of examples have been recorded, both in this country and abroad. It occurs most often in men and its position among the sarcomata is unstable.

Symptoms.—According to Kaposi, who had a large number of cases under observation, the disorder begins on the hands and feet. The early lesions are reddish-brown or bluish-red nodules, varying in size from a pea to a bean or larger, discrete or grouped, forming infiltrations. Later observations show that in many instances the early lesions may be bluish infiltrations of varying size. The hands and feet may be diffusely involved with such infiltration without visible nodules. At a later date nodules and definitely infiltrated plaques develop. It is common for infiltrated pigmented plaques of varying size to occur on the lower extremities. The course of the disease is slow and usually presents new lesions in gradually increasing numbers, with the older lesions increasing in size or undergoing involution, leaving atrophic, scar-like or depressed pigmented areas. Marked swelling of a boardy character may involve the hands and feet, largely interfering with their function. In these same areas, by the multiplication of lesions and the progressively increasing infiltration with the resulting enlargement above-mentioned, an elephantiasic condition may

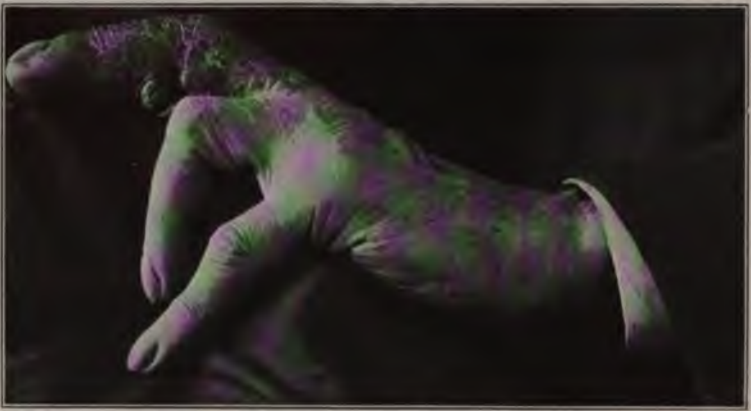
FIG. 336



Idiopathic multiple hemorrhagic sarcoma.

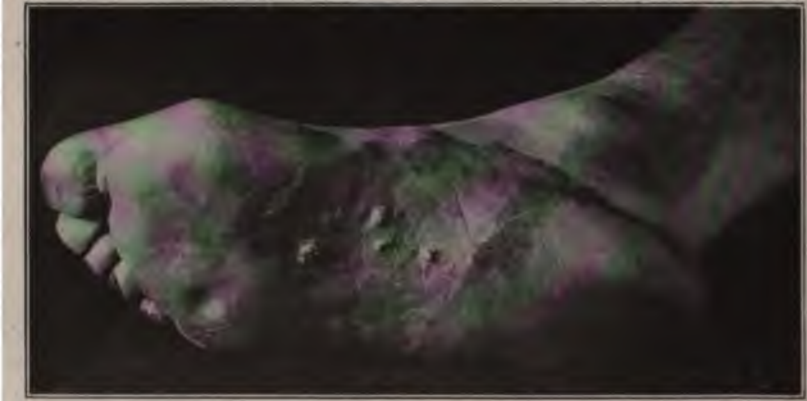
occur, when the leg, for example, becomes hard, bluish in tint, and more or less covered with nodules of various sizes. Not infrequently the nodules are covered with or surrounded by telangiectases and in addition soft tumors resembling angiomata are present. The lesions may be few or numerous, and in some instances the face and trunk are invaded. This usually occurs in the later stages. The mucous membrane of the nose (Kaposi) and of the mouth may participate in the process. Subjective symptoms may or may not be present. Commonly the lesions are tender and painful, the subjective symptoms being more manifest as the lesions are developing. Itching sensations have been described. According to Kaposi, the disorder usually runs its

Fig. 337



Idiopathic multiple hemorrhagic sarcoma. (Fordyce and Stewart.)

Fig. 338

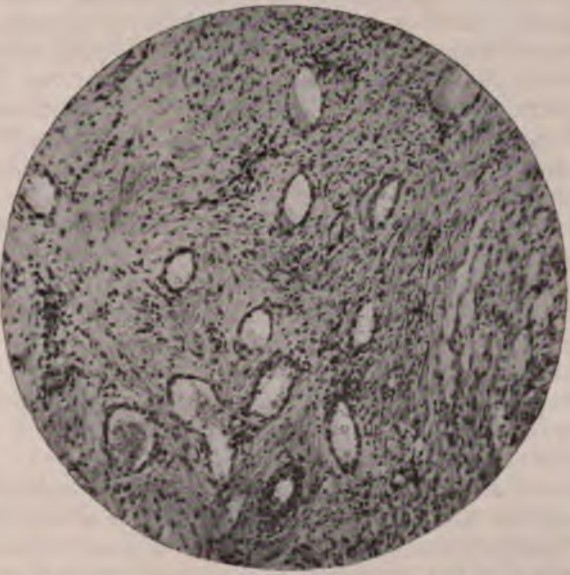


Idiopathic multiple hemorrhagic sarcoma. (Fordyce and Stewart.)

course to a fatal termination in four to six years. Many cases are on record in which the disease has continued for twenty or more years. In the case of a patient recently reported by Cole and Crump[1] leukemia developed during the course of the disorder.

Etiology.—The disorder occurs usually in males[2] past the fortieth year. In a resume of the recorded cases Sequeira found only 5 in females and all of these were not undoubted instances. He found 3 whose development followed severe chill, but believes gout and rheumatism, sometimes given as etiological factors, are incidental. Lieberthal observed trauma in 2 of his 4 cases and Jackson's[3] case followed a frost-bite.

FIG. 339



Idiopathic multiple hemorrhagic sarcoma. Zeiss 8 mm., C. O. 4. Showing the new-formed vessels and blood spaces in the sarcoma tissue. (Fordyce.)

Pathology.—Histologically, Kaposi found a round-cell sarcoma. In some instances features characteristic of spindle-cell sarcoma were seen. Capillary hemorrhages were noted, which explained the peculiar discoloration and pigmentary changes.

The histopathology has more recently been studied by Wende,[4] Bulloch,[5] MacLeod,[6] Lieberthal,[7] Sequeira and Turnbull,[8] Favera,[9] and

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[1] Archiv. of Derm. and Syph., 1920, n. s. i, p. 283; Report of two cases of idiopathic hemorrhagic sarcoma (Kaposi), the first complicated with leukemia.

[2] Little, Brit. Jour. Derm., 1918, xxx, p. 214; Case demonstration, woman of Irish descent.

[3] Brit. Med. Jour., October 2, 1897, p. 866; quoted by Sequeira.

[4] Jour. Cut. Dis., 1898, xvi, p. 205.

[5] Brit. Jour. Derm., 1901, xiii, p. 204; case report by Sequeira, with histology by Bulloch, with bibliography.

[6] Ibid., 1905, xvii, p. 173.

[7] Jour. Cut. Dis., 1909, xxvii, p. 522.

[8] Brit. Jour. Derm., 1913, xxv, p. 351.

[9] Archiv, Bd. cix, p. 387; abstr., Brit. Jour. Derm., 1912, xxiv, p. 82; a clinical and histological study, based on six cases.

Gilchrist and Ketron.[1] The histological changes occur in the corium, the epidermis showing secondary involvement. The latter is thinned and has the interpapillary processes flattened out over the tumor, but shows proliferation of the rete at the edges of the growth. In certain cases, where marked thickening has occurred, more extensive changes are described.

The condition has been considered a sarcoma by Kaposi and others, a chronic inflammatory process[2] and a granuloma. It has also been described as a growth of organizing connective tissue associated with marked vascular dilatation, edema and deposit of blood-pigment (MacLeod). The middle and upper parts of the corium are involved, showing a markedly vascular structure, with increased capillary loops, the latter being dilated and showing endothelial proliferation. Occasionally, the dilatation is sufficient to produce small sinuses. The lymphatics and lymph spaces contain pigment- and red blood-cells. Edema is found throughout the area, and the elastic tissue has disappeared from the center of the mass (MacLeod). The cells are of the connective-tissue type, both spindle and round. Mast cells are occasionally described in small numbers.

The chief changes are summed up by Favera as follows: New-formation and dilatation of the blood capillaries and lymphvessels; infiltration with spindle cells, either related to the capillary new-formation or of endothelial origin; and a small-cell and plasma-cell infiltration.

The pigment in these cases gives the iron reaction, and is derived from the hemorrhages occurring from the engorged capillaries. It differs from that found in melanotic sarcoma, where it is a product of cellular activity.

Gilchrist and Ketron concluded that the lesions begin in the skin as angiomata due to a proliferation and dilatation of the blood capillaries which are very frail at first and liable to rupture. This is followed by proliferation of the interstitial connective tissue and endothelium which gradually obliterates the blood spaces, forming solid tumors. In the early stages these resemble in some areas young connective tissue and in others sarcomata. As the lesions grow older they assume a more fibrous aspect and may undergo involution. Coincident with the formation of the tumors, there is a sclerosis of the small arteries supplying them, causing a gradual decrease in the amount of blood. To this is due, most likely, their later evolution as well as involution.

Treatment.—Arsenic was used with success by Köbner.[3] Wende,[4] Da Costa,[5] Leviseur[6] and others.

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[1] Jour. Cut. Dis., 1916, xxxiv, p. 429. Also see Sibley, Brit. Jour. Derm. and Syph., 1920, xxxii, p. 331. A case of so-called "Multiple Hemorrhagic Sarcoma" (Kaposi). Case report with histology. Kendal Frost, Archiv. of Derm. and Syph., 1921, iii. A case of multiple hemorrhagic sarcoma; a clinical and histological study.

[2] Bulloch, Turnbull and Skinner, Brit. Jour. Derm., 1919, xxxi, p. 28.

[3] Berlin. klin. Wchnschr., No. 2, 1883.

[4] Loc. cit.

[5] Festschrift (Unna), 1910, xx, No. 1, p. 212; abstr., Jour. Cut. Dis., 1911, xxix, p. 652.

[6] Jour. Cut. Dis., 1919, cxxxvii, p. 286.

Radiotherapy produced total involution of the lesions in a case recorded by Heiman[1] and improvement in one recorded by Lustgarten.[2] Much improvement in all lesions treated with x-rays was noted in a case under the observation of the author. Other observers have noted improvement or complete relief following the use of x-rays.

Prognosis.—The major portion of cases prove fatal in from two to five years. Spontaneous recovery has been reported, and more recently recovery following treatment has been noted. In several cases the disease has lasted for many years. Cases of this sort have been recorded by Hardaway,[3] Brayton,[4] and others.[5]

ORIENTAL SORE.[6]

Synonyms.—Mycosis cutis chronica, Lupus endemicus, Aleppo evil, Delhi boil, Oriental button, Oriental ulcer, Gafsa button, Afghan plague, Taschkat ulcer, Natal sore. Fr., *Bouton d'Orient, Chancre du Sahara, Clou de Biskra;* Ger., *Endemische Beulenkrankheit.*

The morbid condition known as oriental sore is one designated not merely by the synonyms detailed above, but by a series of names in the Arabic, Turkish, Persian, and Russian languages which refer to the same disease. It is an endemic cutaneous affection, recognized chiefly in tropical and subtropical countries, more particularly in those sections which have given titles to the disease, such as Biskra, Gafsa, Aleppo, Bagdad, and Delhi. It occurs in Morocco, Algiers, Tunis, Egypt, Crete, Cyprus, the Crimea, Syria, Mesopotamia, Arabia, Persia, Turkestan, India, in Brazil, and probably other portions of South America. Occasionally, from these centers infected patients are transported to various other countries, including England and America.

Definition.—Oriental sore is a specific ulcerative process occurring on exposed portions of the body, running a fairly definite course, and terminating in cicatrization. It is produced by *Leishmania tropica* (Wright).

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[1] Jour. Cut. Dis., 1913, xxxi, p. 424.

[2] Ibid., 1905, xxiii, p. 171.

[3] Ibid., 1890, viii, p. 21.

[4] Indiana Med. Jour., November, 1893 (quoted by Fordyce).

[5] Sequeira, Brit. Jour. Derm., 1901, xiii, p. 201.

[6] Firth, Vierteljahr., 1886, p. 805 (Zur Anatomie und Ætologie der Orientbeule). Cunninghan, Scientific Memoirs by the Medical Officers of the Army of India, No. 1, 1884, Calcutta, 1885 (On the presence of peculiar parasitic organisms in the tissue of a specimen of Delhi boil). Wright, Jour. Cut. Dis., 1904, xxii, p. 1 (Protozoa in a case of tropical ulcer). Scheube, Diseases of Warm Countries, 2d ed., 1903, p. 53. Darling, Jour. Cut. Dis., 1911, xxix, p. 617 (a clear exposition of the subject, including a historical sketch, geographical distribution, climatological factors, and clinical, pathological, and bacteriological description). Hutton, Jour. of Tropical Med. and Hygiene, 1912, xv, No. 1, p. 9; abstr. Jour. Cut. Dis., 1912, xxx, p. 376. Wenyon, Parasitology, iv, 1911; abstr. Brit. Jour. Derm., 1912, xxiv, p. 166 (Oriental sore in Bagdad). Manson, Manual of Tropical Medicine, p. 599. Howard Fox, Jour. Cut. Dis., 1912, xxx, p. 206 (four illustrations of oriental boil, furnished him by Dr. Adams, of the Assyrian Protestant College at Beirut). Hodara and Bey, Derm. Wochenschr., 1912, xliv, No. 1, p. 16; abstr. Jour. Cut. Dis., 1912, xxx, p. 229. Castellani and Chalmers, Manual of Tropical Medicine, 2d ed., 1913, p. 1548. McEwen, Jour. Cut. Dis., 1914, xxxii, p. 275 (Oriental sore in the Americas, with report of case).

Symptoms.—The disease begins, after an incubation period of days or months, as a circumscribed, itching maculo-papule, often appearing like an insect-bite, and having a firm, shot-like feel. It is situated on a hyperemic and infiltrated base. In the course of a few days, its surface becomes covered with furfuraceous scales, which later become agglutinated by the secretion from beneath of a thin fluid, forming a yellowish-brown, thick, adherent crust. On the removal of this crust there is exposed beneath a shallow ulcer, which extends peripherally and exudes a secretion, which tends to reproduce the crust. Beneath this crust the ulcer gradually spreads. In the vicinity, satellites composed of new papules and ulcers form. These often merge and produce a single sharply outlined, rounded or oval, punched-out ulcer, with a granulating floor, edematous base, outlying areola, and bulky crust. The dimensions of the ulcer vary from 6 to 12 or more centimeters in diameter.

After a period of from two to twelve or more months, repair takes place by the gradual drying of the lesion and the usual processes of granulation and cicatrization. The resulting scar is usually sunken, at first pigmented, and, about the face, is apt to be exceedingly deforming. The areas chiefly affected are the exposed portions of the body, such as the face, ears, hands, feet, arms, and legs. As a rule, the palms and soles, the scalp and trunk are not invaded. In some cases the primary lesion does not proceed to ulceration, but remains in the nodular stage, from which it undergoes involution. Instead of one, there may be several, in rare instances a large number, of separate sores. By some writers a verrucous type has been described, and by others mucous-membrane involvement has been recorded. In some instances an irregular fever has been noted occurring for some time before the development of the lesion. The course of the disorder is gradual extension for several months until a certain stage is reached, then gradual involution of the lesion, ending in scar-formation; the whole process requiring in the major portion of cases an average of from three to five to twelve or fifteen months.

Etiology.—Oriental sore is contagious, autoinoculable, transmissible by direct contact, and apparently also through the medium of insects and articles of clothing. It affects indiscriminately persons of both sexes and of all ages and nationalities, independently of the general health or occupation. It often attacks children in the second year of life, and seems at times to confer a species of immunity against second attacks, though many instances tend to disprove the possibility of such protection. Those exposed may develop symptoms in the course of two weeks, though in other instances it would seem that many months intervene before the infection is established. Nicolle determined experimentally that the incubation period varied from 16 to 166 days. Hutton recorded the case of a lieutenant in the British army, who contracted the disease in India, in whom the incubation period appeared to be eleven months. The direct cause of the disease is the parasite *Leishmania tropica*.

Pathology.—The organism accepted as the cause of the disorder was described in 1904 by Wright, who discovered in a smear preparation made from a tropical ulcer intracellular organisms, to which he gave the name *Helcosoma tropicum*. Previous to this time, Cunningham, whose observations were confirmed by Firth, described an organism termed by them *Sporozoa furunculosa*. It appears that most observers believe this organism differs from the one described by Wright. The latter organism may be seen in smears from ulcers or in films taken from serum expressed from nodules, and occurs as a small, oval, round or oblong body, situated in the cytoplasm of large mononuclear cells. It has a large, purple-staining mass placed at the periphery, and a smaller, more deeply staining rod or dot placed at a short distance from the trophonucleus and at different angles with respect to it and the long axis of the parasite (Darling). The organism varies from 2 to 4 microns in diameter, and the method of reproduction is by fission. Darling states that the pathogenic agent of oriental sore is morphologically indistinguishable from the bodies found by Leishman and Donovan in the spleen of patients dying from kala-azar.

The histology as described by various observers shows an extensive cellular infiltration occurring throughout the corium and in the papillæ, accompanied by atrophy and disappearance of the epidermis. The cellular infiltrate is composed of plasma-cells, lymphoid-cells, and, according to Wright, large cells with single vesicular nuclei and a relatively large amount of cytoplasm, in which are large numbers of microörganisms. In the latter, which composes a large part of the infiltration, the microörganisms are closely packed together and occupy most of the space between the nucleus and the cell-membranes, twenty or more microörganisms being found in one cell. In addition, giant-cells in varying numbers are described. The sweat-glands and perivascular lymph spaces show considerable round-cell infiltration; and coincident with the ulceration there is a secondary invasion of ordinary cocci, with accompanying leukocytic infiltration (Darling).

Diagnosis.—The diagnosis in localities where the affection is endemic is attended with but little difficulty; but among the classes in which the disease is especially likely to be encountered it is confused most often with syphilis. The strictly local character of the oriental sore, the course of the lesion, the duration of the disease, and, finally, the demonstration of the causative organism, make the distinction.

Treatment.—Treatment is unsatisfactory. Cauterization, excision, erasion, and local antiseptic treatment are the methods usually employed in the management of the disorder. By many local authorities the milder and soothing, rather than the more severe and drastic, measures are advocated. According to Castellani, salvarsan, atoxyl, and radiotherapy have not been successful. Jeanselme[1] reported improvement with salvarsan. Recently Castellani[2] says tartar emetic

[1] Bull. Soc. fran Derm., January, 1914, p. 4.

[2] Castellani and Chalmers, Manual Tropical Medicine, 1920, 3d ed., p. 2173.

is specific. He recommends intravenous injections of 5 to 10 c.c. of a 1 per cent. solution in saline or distilled water daily for five to ten days, then twice or three times weekly until fifteen to thirty injections have been given. Low[1] used a 2 per cent. tartar emetic ointment successfully. Lyster and McKenstry[2] employed chlorin ionization in twenty-minute sittings together with a hypochlorite dressing. The size of the electrode was 1½ by 1½ inches and the strength of current was 20 milliampères.

Prognosis.—The prognosis is generally favorable save in the matter of the deformity left by the resulting scars, which may by contraction result in considerable disfigurement of the face.

FRAMBESIA.[3]

Synonyms.—Yaws, Pian, Polypapilloma tropicum, Bouba or Boba, Tonga, Parangi. Fr., *Bouton d'Amboine.*

Definition.—Frambesia is an infectious and contagious disorder, existing endemically in certain tropical countries, affecting chiefly the colored races, characterized by peculiar raspberry or cauliflower-like lesions, and produced by *Spirocheta pertenuis*. The term frambesia was applied to the disorder by Sauvages, in 1759, but the disease has since been recognized under many colloquial terms, only a few of which are mentioned above. From a historical standpoint the disorder appears to have been recognized for a long period of time. It occurs in Northern Africa, Algeria, Mozambique, Madagascar, and the Comoro Islands, in Asia, Australasia, and the French and English West India Islands. Imported cases occasionally are recorded in England and America.[4] Goodman[5] observed examples of the disorder in Porto Rico and in Panama.

Symptoms.—The disorder is recognized by all authors as occurring in two stages; a number believe there is in addition a tertiary group of symptoms. These later symptoms are ascribed by some to an intercurrent infection with syphilis. According to Castellani, whose description is for the most part followed in these paragraphs, three stages occur. An incubation period, varying from two to four weeks, characterized by mild constitutional symptoms, including elevation of temperature, headache, malaise, and rheumatoid pains, is followed by the development of the primary lesion at the site of the inoculation, which is always extragenital. The primary lesion is a moist, crusted papule, which soon becomes surrounded by several others of similar type, and by fusion a single lesion is formed, which is covered by a thick crust. This later develops into an ulcer having a granulating floor and clean-cut edges. The subsequent career of this lesion varies. Occasionally, it heals, leaving a whitish scar, which later becomes pigmented; or it may develop into a lesion typical of the so-called secondary stage. The primary lesion (*maman pian*, "mother yaw") may be painful or present itching sensations, is not indurated, and is found most frequently on exposed surfaces—in men and children on the hands, arms, and legs; in women on the breasts, hips, and other areas. In from one to three months after the beginning of this lesion, the secondary manifestations occur. Their development is also preceded by constitutional symptoms. The lesions begin as small, reddish papules, irregularly disseminated, some of which show a yellow point or small, yellow apical crust. These remain for some weeks, when part of them disappear, leaving furfuraceous patches. Others become larger and develop into the characteristic nodules of the disease. These lesions vary in size from that of a large pea to a nut, and present a granulating surface, likened to a raspberry or cauliflower, secreting a thin, purulent material, which on drying produces a yellowish or brownish crust. This type of lesion occurs commonly on the upper and lower limbs and face. By coalescence rings may be formed about the mouth, anal region, and other areas ("ringworm yaws"). After some weeks the surface of the lesion becomes verrucous. This latter type of lesion occurs particularly on the dorsum of the foot and toes. On the palms and soles livid or dark brownish spots first appear; later typical frambesial nodules develop. In another type, hard, round, flattened papules, or small nodules having a thick, hard, epidermic plug in the center, are observed on the palms and wrists. Upon exfoliation of the plug, a deep, permanent depression remains.

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[1] Jour. Trop. Med. and Hyg., 1915, xviii, p. 258.

[2] Lancet, February 23, 1918.

[3] Castellani, Brit. Med. Jour., November 23, 1907, p. 154 (with cut showing spirochete), and Jour. Cut. Dis., 1908, xxvi, pp. 151–210; Castellani and Chalmers, Manual of Tropical Medicine, 2d ed., 1913, p. 1170; Scheube, Diseases of Warm Countries, 2d ed., p. 290; Manson, Tropical Diseases, 4th ed., 1910, p. 566.

[4] C. J. White, Jour. Cut. Dis., 1910, xxviii, p. 533; and White and Tyzzer, ibid., 1911, xxix, p. 138.

[5] Ref. at the end of this section.

The subjective sensations vary. There may be itching, and in certain situations, such as on the soles of the feet, pain is exhibited. A peculiarly offensive odor is often exhaled from these patients. The course of the disease varies. In the majority of cases—in children in from three to six months, and in adults in from six to twelve months—the lesions undergo resolution, leaving dark, hyperpigmented spots. In some cases they continue to develop in crops of nodules for several years, the individual lesion running its course in from two to four months. Occasionally, a much longer time is required for their evolution.

In addition to the above described typical lesion, scaling, papular, and ulcerative lesions occur. During the latter part of the secondary stage there may also be roundish or irregularly outlined, whitish patches, covered with hard, conical papules. A composite of an average case shows typical frambesial nodules, reddish papules with intact epidermis, other papules moist and crust covered, furfuraceous patches interspersed, and hyperpigmented areas developed from previous lesions. In the tertiary stage gummatous-like nodules and deep ulcerative processes are described. In career the former are indolent, and after softening and breaking down produce ulcers with clean-cut

In syphilis the plasma-cells are apt to be arranged in rows and clustered about the bloodvessels. In addition, there are multinuclear and incomplete giant-cells and intracellular hyaline degeneration. There also occurs an endarteritis, with proliferative changes in the vessel-walls.

Diagnosis.—The disorder is to be differentiated chiefly from syphilis. Some early observers, including Hutchinson, believed the diseases to be identical. All doubt concerning this matter has been dispelled by recent findings. Clinically, the following are important points of distinction: Syphilis often, frambesia rarely, attacks the mucous surfaces. The last-named disease much more rarely involves the lymphatic glands. There is usually itching in frambesia, which is not characteristic of syphilis. The characteristic lesion of frambesia is not seen in syphilis. The papules of the former are usually described as having a yellow, waxy crust, with honey-like serum beneath; those of syphilis, on the other hand, have a peculiar color and do not undergo this change. Clark[1] describes cases of ringworm yaws that resemble closely the annular, papular syphiloderm as seen both in the negro and other races. Syphilis is directly hereditary; frambesia, though common in children, is not inherited; healthy parents frequently contract the disease from their children. A very sharp distinction occurs in the early lesions of the two disorders. The Wassermann reaction is of no value in differential diagnosis as it is positive in both disorders. Goodman[2] reported a positive reaction in all of 7 cases he had tested.

Treatment.—The general treatment of the disease consists in the administration of tonics, the use of proper food, and in securing proper hygienic surroundings. Mercury and potassium iodid have been of value internally, but with the discovery of arsphenamin (salvarsan) a specific method of treatment has apparently been evolved. Many observers report complete eradication of the disorder with a single injection of this remedy. Among others, the disease has been treated successfully with arsphenamin by Castellani,[3] Rost,[4] and Cockin.[5] Recently Raoul de Boissiere[6] recorded the successful treatment of 182 patients with this remedy. The local treatment consists of the usual antiseptic surgical washes and dressings.

Prognosis.—The disease is not serious, relative to the life of the patient. In young children and debilitated older subjects it is of more consequence. Occurring in epidemic form on plantations, it interferes more or less seriously with labor conditions.

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[1] Jour. Cut. Dis., 1914, xxxii, p. 18.

[2] Archiv of Derm. and Syph., 1920, ii, p. 7; Frambesia Tropica (Yaws). A study of the literature with personal observations, a critique of its supposed identity with syphilis, and a bibliography.

[3] Loc. cit.

[4] Münch. med. Wochenschr., 1912, lix, No. 17, p. 924; abstr. Jour. Cut. Dis., 1912, xxx, p. 627.

[5] Jour. Tropical Medicine and Hygiene, September 2, 1912, xv, p. 277; abstr. Jour. Cut. Dis., 1913, xxxi, p. 65.

[6] Edin. Med. Jour., October, 1917, p. 226. On the Treatment of Yaws with Salvarsan and Allied Remedies.

VERRUGA PERUANA.[1]

Synonyms.—Peruvian wart, Oroya fever, Carrion's disease.

Verruga Peruana was described first in the sixteenth century by Zarate, of Lima, in his *History of the Conquest of Peru* (1543). The first scientific contribution to the subject was made by Tschudi, in 1845. The disease formerly had a more extensive distribution than at the present time. It is supposed to have occurred in Ecuador, Bolivia, and Chile. At present it is largely limited to certain narrow valleys on the western slopes of the Peruvian Andes, between latitudes 8 and 13 south, and at altitudes of from 1000 to 12,000 feet. The disease is never contracted in the lower coastal plain, but always in the valleys at the higher altitudes mentioned, not less than 16 to 36 miles from the sea (Darling). It does not occur on the eastern face of the Andes. In America a case has been recorded by Stelwagon.

Definition.—Verruga Peruana is an infectious disease, characterized by constitutional symptoms, the cardinal one of which is a severe grade of anemia, and a cutaneous eruption of warty nature, having a tendency to bleed.

Symptoms.—Two forms of the disorder are described, a mild and a severe. The incubation period varies from fifteen to forty days. In Carrion's case it was twenty-one days. During the stage of invasion, headache, pains in the extremities, fever, chills, anorexia, and insomnia are noted. The fever is of a remittent or intermittent type. The liver and lymph glands become enlarged; and the anemia is of such a severe grade that the red cells may become reduced to one million or even five hundred thousand per cubic millimeter. In severe cases a fatal result may occur within three or four weeks. In mild cases the constitutional symptoms are less marked, the anemia being of a milder grade, and after a duration of three or four weeks to several months cutaneous manifestations occur.

The eruption occurs chiefly on the extensor surfaces of the arms and legs, also on the face and neck. In addition, the mucous membrane of the lips, tongue, gums, palate, and pharynx may become involved, as well as the serous membranes of the internal organs. The cutaneous lesions begin as slightly elevated, pinkish or reddish spots, which later assume a dusky, bluish-red hue. There may be associated vesicles, bullæ, or pustules. These lesions rapidly develop into papules, and later into nodules, which are conical or hemigloboid in shape, and vary in size from that of a pea to a bean. When fully developed, they appear as irregular, wart-like lesions, usually discrete, of a red color, firm or soft to the touch, sessile or pedunculated, cylindrical, papillomatous, and hemorrhagic. In addition to these, a subcutaneous, nodular type of lesion occurs, which may develop into large size. These approach the surface, to which they become attached, and finally break down, producing deep ulcers, furnishing a fetid discharge, which may also be the seat of abundant hemorrhage. In these the lesion is transformed into a malignant-looking, grayish or blackish, spongy mass, covered with brownish crusts, and exhaling a putrescent fetor. The lesions vary in number from a few to a great many. During a period of several months new groups appear, each being accompanied by constitutional symptoms. The disorder, unless fatal, finally disappears by absorption of the nodules and cicatrization of the ulcers.

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[1] Tschudi, Arch. f. phys. Heilk, 1845, p. 378; Matas, Morrow's System, iii, p. 694; Scheube, Diseases of Warm Countries, 1903, 2d ed., p. 298; Manson, Tropical Diseases, 1910, 4th ed., p. 580; Stelwagon, Diseases of the Skin, 7th ed., 1914, p. 859; Darling, Jour. Amer. Med. Assoc., 1911, lvii, p. 2071; Gilton, ibid., p. 2074; Castellani and Chalmers, Manual of Tropical Medicine, 2d ed., p. 1196.

Etiology.—The disease is transmissible by inoculation, as was evidenced in the case of the physician Carrion, who, after inoculation in both arms with the blood of a patient, perished in fifteen days. His name added to the list of titles given above is a memorial to this sacrifice. The disease attacks persons of both sexes and all ages, including new-born infants. Persons working in the earth are especially liable to contract the disorder. It seems further to be aggravated by conditions of moisture and warmth. An organism, apparently a bacillus, described by Barton (quoted by Darling), is found in the red-blood cells during the febrile period, and also in the local lesions. These bodies have been the subject of much discussion, and Darling believes them to be a unique type of microörganism. The histology of the disease shows it to be a granuloma, similar in some respects to that of frambesia. A cellular infiltration, held together by a delicate fibrous stroma, caused early observers to compare the lesions to sarcoma.

Diagnosis.—The disorder chiefly resembles frambesia, and the most important factor in diagnosis is the residence of the patient. It is said that natives become immune to the disease, but that strangers going through infected districts are speedily attacked.

Treatment.—The treatment requires the removal of the patient to a climate where the disease is not endemic. Arsenic by hypodermic injection has been recommended. Castellani suggests arsphenamin and neoarsphenamin. In convalescent stages tonics should be employed.

Prognosis.—The disease may run its course in a few days or be prolonged for many months. The mortality varies from 10 to 40 per cent. Complications arising render the prognosis unfavorable. Among these are intestinal hemorrhages, hematuria, metrorrhagia, hemoptysis, epileptiform convulsions, and meningitis.

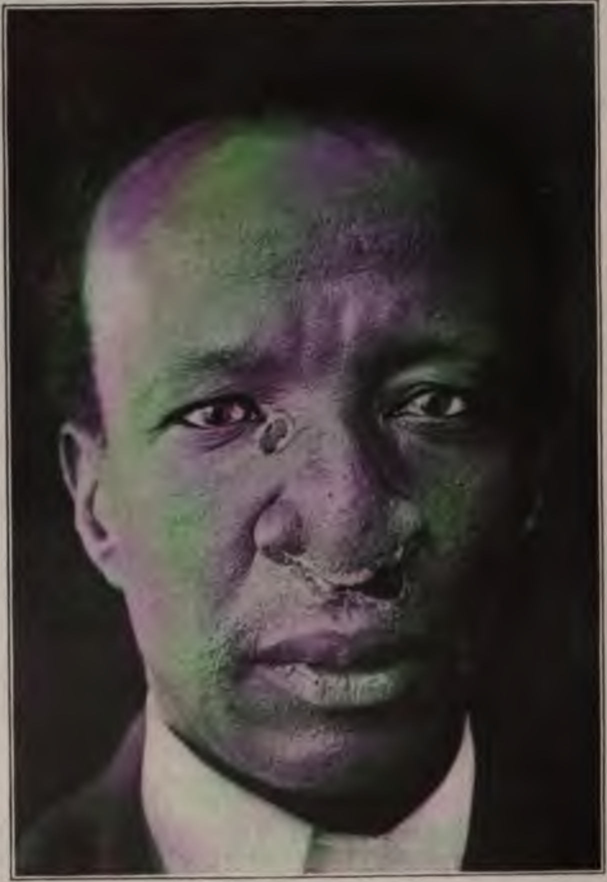
GANGOSA.

Gangosa is a disorder endemic in certain countries, such as Bolivia, the Philippine and Caroline Islands, British Guiana, Jamaica and other parts of the West Indies, but especially in the island of Guam, where it is said to have existed for the last one hundred and fifty years.

The disease in many of its features strongly suggests rhinoscleroma, a malady with which some authors hold it to be identical.

Symptoms.—Gangosa is characterized by a destructive ulceration, commonly beginning by attacking the soft palate, pillars, or uvula, and extending thence to the hard palate and the nasal cavity, downward to the larynx and upward to the face. The destructive process is either acute or chronic, and may terminate either by cicatrization or by extensive destruction of tissue with mutilation. Constitutional symptoms are wanting or developed in very mild forms.[1]

Fig. 340



Gangosa. (Fordyce.)

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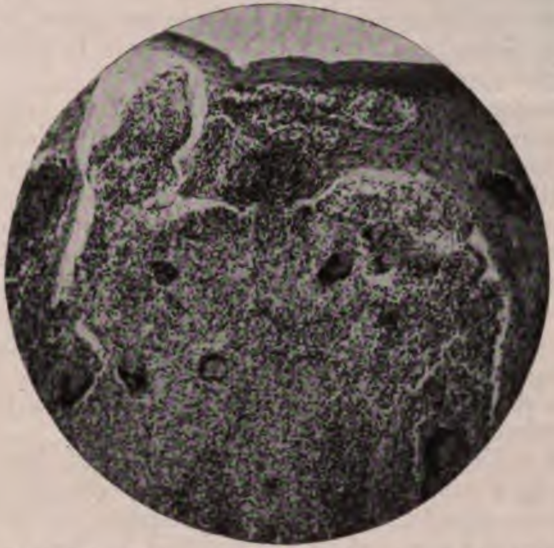
[1] Mink and McLean, Jour. Amer. Med. Assoc., October 13, 1906, p. 1166, and supplementary paper Jour. Cut. Dis., 1907, xxv, p. 503; Fordyce and Arnold, Jour. Cut. Dis., 1906, xxiv, p. 1; Leyes, Jour. Trop. Med., February 15, 1906; Senn, Jour. Amer. Med. Assoc., January 11, 1908, p. 116; Geiger, U. S. N. Med. Bull., January, 1908; E. R. Stitt, Jour. Cut. Dis., 1908, xxvi, p. 103; idem., U. S. N. Med. Bull., 1907, i, p. 96; Musgrave and Marshall, The Philippine Jour. of Sci., ii, No. 4, Section B, 1907, abstr. Brit. Jour. Derm., 1908, xx, p. 173 (Gangosa in the Philippine Islands. A case occurring in a Philippine, a native of the Batan Islands).

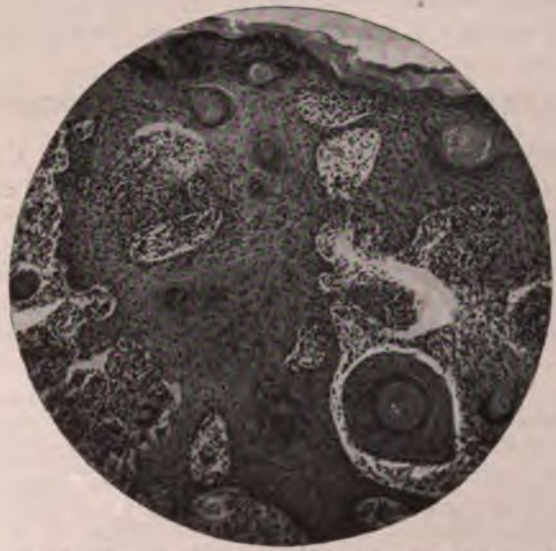
Geiger recognizes three types of ulceration of the upper air-passages common in the island of Guam: (*a*) septic forms due to the usual

Fig. 341



Gangosa. Showing the diffuse infiltration of the corium with round-, plasma-, and giant-cells. The vessels are the seat of an endarteritis, many of them being obliterated. (Fordyce.)

Fig. 342



Gangosa. Tropical ulceration involving nose, pharynx, and larynx. Hyperplasia and downgrowth of the epidermis, with cellular infiltration of round-, plasma-, and giant-cells in the corium. (Fordyce.)

pyogenic organisms; (*b*) ulcerations that are obdurate to antiseptic treatment, a group inclusive of most forms of gangosa; (*c*) distinct nodules or tubercles, involving the skin or mucous membrane of the nose, soft palate, pharynx, larynx, or lips. An organism closely resembling the bacillus of diphtheria has been recognized in every case examined by Geiger, and was found in pure culture in the conjunctival sac when the eyes were involved.

The disease is contagious, transmitted by direct contact, and is diminishing in those places where segregation is practised. It occurs almost exclusively among the natives of the countries mentioned, though Stitt's case was that of a sailor (white) who had been in Guam. No positive etiological factor has been identified, except that of Geiger above mentioned. The disease progresses indefinitely, but in itself is not grave as to the life of the patient.

The histological findings vary, but all agree that it is a form of granuloma. In Fordyce's case a granuloma with small lymphocytes, plasma-cells, mast-cells, epithelioid- and giant-cells was found; but tuberculosis was ruled out by the absence of the bacillus and negative animal.experiments and tuberculin tests.

Diagnosis.—The disease is to be distinguished from Vincent's angina by the absence of the causative microörganism of the latter disorder; from syphilis, by the lack of response to the treatment employed for the latter, and by the fact that in Guam, the home of gangosa, syphilis is rare. It is also to be distinguished from yaws and tuberculosis.

Treatment.—Treatment has only a palliative effect. Local antiseptic measures, with cauterization in early cases, is recommended. The general health is to be promoted by appropriate measures.

ULCERATING GRANULOMA OF THE PUDENDA.[1]

Synonyms.—Serpiginous ulceration of the genitals, Groin ulceration, Sclerotizing granuloma of the pudenda, Perforating granuloma of the thigh, Granuloma inguinale tropicum. Ger., *Das Venerische Granulom.*

In 1896 Congers and Daniels first recorded observations of this disease in negroes resident in British Guiana and among East Indians. The malady has since been observed among the natives of the Fiji and Solomon Islands and the New Hebrides. Contributions to the subject have been made by MacLeod, Manson, Grindon,[1] and others.

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[1] Congers and Daniels, Brit. Guiana Med. Ann., 1896, viii, p. 13. Crocker, Diseases of the Skin, p. 1076; Jour. Cut. Dis., 1908, xxvi. p. 61. Daniels, Brit. Guiana Med. Ann., 1898, x, p. 49. Galloway, Brit. Jour. Derm., 1897, ix, p. 133. K. MacLeod, Jour. of Trop. Med., 1899, p. 175. J. Maitland, Lancet, 1899, ii, p. 1624. Manson, Tropical Diseases, 1910, 4th ed., p. 583. A. Powell, Ind. Med. Gaz., 1899, p. 187. Scheube, Diseases of Warm Countries, 1903, p. 541. Sequeira, Brit. Med. Jour., March 7, 1908. Cleland and Hickinbotham, Jour. Trop. Med. and Hygiene, May 15, 1909, p. 143; abstr. Brit. Jour. Derm., 1909, xxi, p. 335 (On the Etiology of Ulcerative Granuloma of the Pudenda). Goodman, Archiv of Derm. and Syph., 1920, n. s. i, p. 151. Ulcerating Granuloma of the Pudenda: A review of the Literature with a Bibliography and some observations of the Disease as seen in Porto Rico.

Symptoms.—The disease occurs in both sexes after the puberal epoch, but chiefly in women. It is seen most often in the genital region and the parts provided with long hairs, but it has been observed on the cheeks, the lips, and inside the mouth. The lesions are vivid-hued, shining, verrucous, vegetating nodules of granulation-tissue. These are at first circumscribed thickenings and elevations. The thin overlying epidermis is excoriated readily, and exposes a hemorrhagic surface, which may ulcerate. The granuloma spreads both by autoinfection and peripheral extension, producing eventually, possibly after years of slow extension, a dense, contracting, irregularly nodulated scar-tissue, here and there sprinkled with islets of actively progressing disease. Unevenly pigmented areas are made up of excoriated or partly cicatrized and corded tissue, often with a narrow, serpiginous, elevated, glazed, pinkish or reddish border. The process is superficial, and as a rule unaccompanied by coincident adenopathy.

The parts most often invaded are the labia and the vagina of women; in men, the penis, urethra, and scrotum; in both sexes the ano-rectal region, pubes, groins, and rarely the bladder. Subjective sensations are not conspicuous; anemia and cachexia occasionally result. Offensive discharges are produced in advanced cases. The disease is aggravated in regions of pressure, friction, and moisture. Manson describes the affected surface as "an area of white or irregularly pigmented, perhaps excoriated, contracting, folded, and dense cicatrix, surrounded by a narrow, serpiginous, irregular border of nodulated, somewhat raised, red, glazed, delicately skinned or pinkish, superficially ulcerated or cracked new-growth."

Etiology.—The disease attacks persons of all races, but chiefly negroes; and individuals of both sexes, but mostly women. The subjects are, as a rule, young adults, though the disease is seen in aged persons. The affection is contagious, autoinoculable, and frequently venereal in origin, though not syphilitic. The precise character of its virus is not known.

Histologically, the disease is seen to be a granuloma. Galloway[2] found a more or less profuse cellular infiltration, beginning in the upper regions of the corium, extending into the papillæ, and also to a variable extent downward. The infiltration was composed of leukocytes, plasma-cells, mast-cells, and connective-tissue corpuscles. There were no multinuclear or giant-cells. In the areas of cellular infiltration the collagen and elastin had degenerated and were destroyed. The bloodvessels showed moderate dilatation. The epidermis over the infiltration showed edema and was stretched and thinned, and in it there was a certain amount of leukocytic infiltration. Donovan (cited by Manson) has recognized in scrapings from the lesions a gigantic short bacillus, 1 by 2 microns in length, with rounded extremities, abundant in mononuclear leukocytes. In addition to this, several microörganisms have been described: a spirochete (Wise, Cleland and Hickinbotham), and a capsulated, intracellular diplobacillus (Siebert, Flu, and others). Other observers have found no microörganisms other than contaminations (Grindon, Choyce and MacCormac). In one of Goodman's[1] cases a spirochete was demonstrated (*S. aboriginalis*) and in three others *Calimatobacterium granulomatis*.

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[1] Jour. Cut. Dis., 1913, xxxi, p. 236 (report of three cases, with histological study).

[2] Brit. Jour. Derm., 1897, ix, p. 133.

Diagnosis.—The disease is to be differentiated from syphilis by the absence of adenopathy, by the extreme chronicity of the process (at times extending over ten years with but few changes), and by the special features outlined above. It is not amenable to antisyphilitic treatment.

Treatment.—Treatment is by excision, which Manson prefers on account of the marked tendency to recurrence in many cases. Curettage and subsequent cauterization have been successful. Mercury and iodin salts are of little if any value. Salvarsan[2] also is disappointing. Radiotherapy has been employed with success.[3] Tartar emetic has been employed with success recently. It is administered by intravenous injection and by inunction (*Cf.* Oriental Sore for Technic of Injection, p. 830).

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[1] Loc. cit.

[2] Choyce and MacCormac, ibid., 1913, xxv, p. 65.

[3] MacLeod, ibid., p. 66.

CLASS VII.

NEUROSES.

PRURITUS.[1]

The term *pruritus* should be reserved to denote a disease of the skin characterized by itching, burning, or smarting sensations, primarily unaccompanied by lesional changes. Hebra was first to recognize the independent character of the disease as thus defined; but it is to be regretted that he did not give it a distinct name from that which is also applied to a symptom common to several maladies of the skin. It is a sensory neurosis, in which itching, burning, and smarting are the essential symptoms. It may be general or local. In either form it begins usually by a tickling, pricking, crawling, or itching sensation in the skin, which causes the sufferer to rub, press, scratch, or otherwise irritate the affected integument. Other symptoms are usually present, however, and are evidenced locally by the torn and injured skin, and systemically by varying degrees of malnutrition and lowered vitality of the patient through loss of rest.

General Pruritus (*Pruritus essentialis*).—General pruritus occurs usually in adults, a common example being the so-called *senile pruritus*. Although all parts of the cutaneous surface are liable to be attacked, it rarely attacks the whole surface simultaneously, but moves about from area to area. The itching may be intense. It is worse at night, but occurs more or less throughout the day, the morning hours being the least uncomfortable. The intensity and discomfort are sometimes relieved only by producing sufficient trauma to change the sensation from itching to one of burning and smarting. The paroxysms are of varying duration, and may be precipitated or accelerated by various influences. Thus, mental disturbance, a draft of cool air, the warmth perceived when in bed, the pressure of clothing, and often the substances applied externally for relief of the itching, suffice to determine a crisis. However firmly the sufferer may resolve to avoid injury to the skin, in a well-marked case the impulse to scratch becomes almost irresistible and in the highest degree tormenting. Many of these patients restrain themselves and avoid scratching during the waking hours, but while asleep inflict serious damage upon their skins. When the itching becomes intense, brushes, combs, coarse cloths, and even metal instruments, are employed for the purpose of assuaging temporarily the local distress.

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[1] For fuller discussion of the subject and bibliography, see Jacquet, La Pratique Dermatologique, iv, p. 341.

The objective cutaneous symptoms which may be present are all secondary, and invariably result from self-inflicted injury. In many cases they do not appear, the statements of the patient being the sole basis for the recognition of the disease. This absence may be the consequence of unwonted self-control, mildness of the malady, or the transitory character of the lesions produced. Thus, the skin may be reddened during a nocturnal paroxysm under the manipulation of the sufferer, and the transitory hyperemia disappear in the daytime, when the skin is submitted for inspection. Not rarely, however, the skin resents the treatment to which it is subjected, by displaying wheals, hyperemic blotches, reddened papules, excoriations, characteristic "scratch-lines," and minute blood-crusts; in other words, may present all the symptoms of a traumatic dermatitis, which in certain cases may develop into a true eczema, that still further adds to the subjective distress. Skins that for years have been the seat of a persistent pruritus, leading to traumatism of the epidermis, frequently show smaller or larger areas of deep pigmentation.

Senile Pruritus.—Senile pruritus is a term loosely applied to any form of the disease occurring in the aged, in whom it is very common. In this variety the pruritus is usually of a generalized distribution, with the trunk chiefly involved. Senile pruritus, properly, is that form of the disease due to atrophic and degenerative changes in the skin and other tissues of the aged, and is usually a serious disease. Patients of sufficient age to have this form, but whose skins do not show senile changes, are not infrequently attacked with the disease, and in these instances it is due to the causes operative in other generalized cases.

Local Pruritus (*Local pruritus essentialis*).—In local pruritus the areas attacked are the anal, vulvar, and scrotal regions chiefly, and occasionally the palms, face, and scalp.

Pruritus Ani.—This disorder may be limited to the anus and anogenital region, or be experienced over the neighboring parts (genital region, cleft of the nates, buttocks, and upper and inner parts of the thighs). It is a disorder of exceeding persistence when not properly treated, and accompanied by local distress (itching, burning) of a severe grade. In men the scrotal or anal region alone or in combination with the perineum may be attacked. The skin about the anus presents a whitish, sodden appearance, with increase in depth of the folds, in which there collects a foul-smelling secretion. Frequently, the intensity of the itching in these cases cannot be described. Naturally, in time local inflammatory symptoms develop, due to the scratching and rubbing, resulting in excoriations, fissures, crusting, induration, hyper- or deep pigmentation, and extensive thickening of the nates, as well as in a true eczema. The methods of obtaining relief employed by some of these victims of intense suffering testify strongly to its severity. The most severe types are seen in men, though both sexes are affected, and the subjects are commonly those in middle or later life. Instances of severe pruritus ani in children, however, are on record. When the disorder extends to and involves the scrotum in men, this becomes thickened and soggy, and may even become vitiliginous.

Pruritus Vulvæ.—This may be a disorder of mild grade or be productive of so much torture that after long deprivation from sleep and profound agitation of the nervous system, the unfortunate sufferers have been tempted to commit suicide.

Like pruritus ani, that of the vulva may be limited to the genital region (mucous, muco-cutaneous, or cutaneous surface adjoining the latter, the outer faces of the labia majora, the labio-femoral clefts, and the perineum), or it may extend to and involve the anus and neighboring parts. The tissues, from much scratching and rubbing, are usually tumid and dry, rather than moist, are reddened in various grades, and often excoriated, crusted, torn, and in extreme cases affected with marked deformity. All the muco-cutaneous tissues, the labia minor, clitoris, and vaginal membrane adjacent may be swollen several times beyond the normal.

Other cases of local pruritus are more rare. Those occurring on the palms, for anatomical reasons, are not accompanied by marked secondary lesions. The pruritus limited to the scalp and face is not common, but is seen occasionally.

Pruritus narium is a frequent symptom of irritation of the Schneiderian membrane. It is thus a common precursor or an attendant phenomenon of rose- or hay-erythema, and in some individuals announces the symptomatic effect of the ingestion of opium and its alkaloids.

We have seen cases limited to the nose, cheeks, and chin that have lasted for several years, each accompanied by a moderate traumatic dermatitis. Pruritus of the tongue is reported in a few instances. It is usually due to a central neurosis, to glycosuria, or other systemic disorder. At times one small area may be noted over the course of a special nerve. The author examined a case in which the itching extended in a narrow band from the vulva along the entire length of the leg, terminating at the heel.

Another important variety of essential pruritus, which might be properly classed as a seasonal neurosis, is that described by Duhring under the title of **Pruritus Hiemalis** (*Prurigo hiemalis*, "*Frost itch*," *Winter prurigo*). This disorder was first described by Duhring,[1] in 1874. It was later independently described by Hutchinson,[2] and was subsequently made the subject of study by Hyde,[3] Corlett,[4] Payne,[5] and others.

This disorder occurs in the autumn and winter, and becomes milder and clears up with the approach of warm weather. The chief and essential symptom is itching, which occurs upon disrobing and retiring. It possesses many features in common with the forms of pruritus already described, including variability in the subjective sensations awakened, nocturnal exacerbation, and the absence of primary eruption. The secondary results are also similar, being sequels of self-inflicted injury in the form of roughness, perifollicular redness and papulation, torn and fractured hairs, excoriations, blood-crusts, and, in severe cases, an induced dermatitis. The disorder is due to changes incident to cold weather, and is often aggravated by the coarse woolen underclothing worn at this season, though it occurs also in those whose personal habits as to clothing and cleanliness leave nothing to be desired. It abates in severity with a rise of atmospheric temperature, though there is occasionally noted persistence of distress after such weather changes. At times several members of a family are attacked simultaneously or successively, which fact has led often to the erroneous belief that it is contagious. The affection, moreover, is one which occurs in persons otherwise enjoying perfect health and in those of every social grade.

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[1] Philadelphia Med. Times, January 10, 1874.

[2] Brit. Med. Jour., 1875, ii, p. 773.

[3] Chicago Med. Jour. and Exam. March, 1885, and February, 1886.

[4] Jour. Cut. Dis., 1891, ix, p. 41, and Jour. Amer. Med. Assoc., 1902, xxxix, p. 1583.

[5] Brit. Med. Jour., 1887, i, p. 985.

The parts attacked are commonly the antero-internal aspect of the thighs, the calves of the legs, around the joints, and, to a lesser degree, the forearms and other parts of the body. It is without question a disease of northern climates, more particularly those where the variations of temperature between the summer and winter range between −30° F. and +100° F. The description by Duhring presents a picture with an accuracy verified by clinical observation, which justifies the recognition of the disease as a form of cutaneous pruritus.

Bath Pruritus.—Stelwagon[1] described a variety of pruritus occurring in patients immediately following a bath. The sensations are itching and burning of varying intensity. It usually occurs on the lower part of the body, from the hips down, but occasionally involves the arms also. It lasts from a few moments to an hour or so and gradually subsides. It attacks adults more frequently and those who habitually have an irritable, dry skin. The temperature of the water appears to have little to do with the condition, as any temperature produces the same result.

Prairie Itch.—This is a popular term applied largely in the Western, Northwestern, and Southern States of America to a cutaneous affection productive of itching sensations. It is supposed to be the disorder popularly described as "Texas mange," "Ohio scratches," "swamp itch," and "lumberman's itch." A parasitic origin has been claimed for it by several observers, who also insist upon its contagious character and its curability by parasiticides. In 1885–86, Dr. Hyde[2] published two articles on this subject, in which many letters of practitioners of medicine were quoted, showing the confusion and perplexity existing in regard to it.[3] From the titles above mentioned, it is seen at once that geographical distribution had to do with their naming. It is generally conceded by authorities on this subject that the bulk of the cases variously named belong in one of four categories: pruritus essentialis, pruritis hiemalis, scabies, or, as more recently suggested by Stelwagon,[1] a disorder of the skin now described as "straw itch," "grain itch," or "grain mite dermatitis," produced by the *Pediculoides ventricosus*. From the above, it is seen that prairie itch cannot be considered a distinct disease, but the term is descriptive of many diseases misinterpreted.

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[1] Philadelphia Med. Jour., 1898, ii, p. 863.

[2] Chicago Med. Jour. and Exam., 1885, lii, p. 187; ibid., 1886, iii, p. 116.

[3] Jones, Kansas City Med. Index, 1886, with views of several Western physicians; Clark, Med. Age, 1886.

Etiology.—In every case of pruritus a probable cause must be diligently sought. While pruritus cutaneous is dignified by a name and described as a disease *per se*, it is essentially a sympton of some disturbance, local or more or less general, of the nervous system; and this disturbance is further secondary to many and varied internal and local organic and functional diseases, and even to increased or decreased physiological activity, as exhibited in pregnancy and the menopause; and finally to abnormal mental states. Bronson[2] states that hyperesthesia is the chief predisposing cause in all cases. This hyperesthesia may be acquired by long-continued irritation of the cutaneous nerves in various itching dermatoses, such as eczema, lichen planus, or scabies, or it may be inherited. The disease may occur at all periods of life and in both sexes, but its aggravated forms are peculiar to middle life and advanced years. Functional or organic disease of the stomach, intestines (including the rectum), liver, kidneys, bladder, urethra, uterus, or ovaries may be causative. The intense itching accompanying icterus is well known, and this often appears only when the icterus is subsiding. An unaccountable pruritus may be the earliest symptom of an internal carcinoma. Malaria, tuberculosis, diabetes, and various forms of nephritis are all causative. Pregnancy is often accompanied by local or more or less generalized pruritus. At the menopause in women, when the organs of reproduction are undergoing organic changes, pruritus occurs, and may be due to reflex local irritation or to the changes incident to the general effect this state produces on the nervous system as a whole. Common among local causes of pruritus is constipation. The hard fecal matter lying in the rectum sets up a reflex irritation, as do also varicose veins and ascarides in this locality. Fistulæ, fissures, and local hyperidrosis are causative. In the genito-urinary tract vesical calculi, polypi, and stricture may be effective.

Reflexly, general pruritus may be caused by a local pruritus. For example, severe generalized cases have occurred in people the subjects of an anal pruritus, the whole disease clearing up by treating simply the local disease.

At times psychic disturbances produce pruritus. This may be seen when a class of medical students or nurses is suddenly attacked by real pruritus when viewing a patient covered with pediculi. Another example of this form occurs in patients who believe themselves to be infected with certain parasites. While a real mental disorder may be present in these patients, they suffer intensely with itching, and not infrequently declare that they not only feel the parasites, but see them emerging from the skin and are able to catch quantities of them. The moral emotions of a depressive character and mental distress occasioned by a variety of circumstances often find physical expression in the disorder.

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[1] Diseases of the Skin, 7th ed., 1913, p. 944.

[2] The Etiology of Itching, Med. Record, October 24, 1891 (a careful review of the subject).

Pathology.—The disease is essentially a functional disorder of the nerves of sensation supplied to the skin, and of itself is incapable of producing objective symptoms. This fact can, in some cases, be clinically demonstrated, as the seat of the pruritus, even though exhibiting artificially produced lesions, will, when protected from external injury, speedily regain its normal appearance, the pruritus continuing, nevertheless. It is probable, though not certain, that the nerves also in this disease undergo no structural change, but merely convey to the periphery a perverted sensation, that is often reflected from some centric point of disturbance.

Diagnosis.—The recognition of general pruritus is usually not difficult, though the secondary results of the disorder are apt to be less characteristic than its early phenomena. The complaint of the patient, the absence of cutaneous disease sufficient to explain the symptoms, and especially the discovery of an efficient cause in some visceral or systemic disorder, are all significant.

One of the most constant features of general pruritus is visible only when the clothing of the patient is entirely removed. It then becomes evident that the affected regions are, in order of frequency, those most accessible to the hands. The posterior body-surfaces are much less involved than the anterior. The small of the back and interscapular regions are usually untouched. The anterior surface of the legs shows more evidence of trauma than the calves, and the forearms more than the upper arms. The lower abdomen and the inner faces of the thighs are more severely attacked than the breast and outer faces of the thighs and hips. The clavicular regions are more excoriated than the back of the neck.

It must be admitted, however, that when the disease is localized, and complicated, as it frequently is, by an eczema or dermatitis, doubt may arise. Attention should then be paid to the history of the disorder, which may reveal the fact that the pruritus preceded for some time the cutaneous symptoms, and may disclose even more. Intelligent patients will often assure the physician of the real nature of the malady by voluntarily remarking that the skin symptoms disappear upon the region that is not scratched, though the itching continues. In all cases the influence of externally operating agencies should be carefully eliminated. The fact that pediculi, fleas, and bedbugs may induce severe itching should be borne in mind. The differentiation between scabies and pruritus hiemalis is important (*Cf.* section on Scabies).

Treatment.—The treatment of any case of pruritus is simple if the etiology is clear. In many cases this cannot be clearly determined, and then recourse to some of the general rules laid down must be resorted to. There are some general principles of treatment, both as to internal and local management, which are of great value, both in mitigating and completely relieving the disorder. As a rule, constitutional treatment is unsatisfactory, unless especially directed to some discoverable etiologic factor, such as diabetes, rheumatism, or the various hepatic, renal and other diseases concerned in its production.

All internal causes of cutaneous irritation should as far as possible be removed, and to this end attention should be directed particularly to any medication to which the patient may have been subjected, and which may have aggravated the complaint, and also to the diet, which should be regulated in accordance with the principles given under Urticaria.

In atonic conditions strychnin, iron, arsenic, and other tonics are indicated. The nutrition of the nerves and of the skin can often be improved by the judicious use of cod-liver oil and other fats.

The attempt to relieve pruritus by the internal use of sedatives is to be recommended only in extreme cases. The narcotics, while they may give temporary relief, tend to relax the skin and in the end to aggravate the disorder. This is especially true of the preparations of opium. The bromids, antipyrin, phenacetin, sulfonal, or even chloral, may be given for brief periods in extreme cases, but always with the understanding that any one of these remedies, after temporary relief, may aggravate the condition for which it was given. Furthermore, there are strong reasons for refusing to employ in pruritic disorders preparations containing opium, cocain, cannabis indica, conium, and other drugs intended to relieve the subjective sensations by internal medication. Many well-nigh incurable cases of the "cocain habit" have been begotten by treatment of this sort when the patient, especially if a nervous woman with an intolerable pruritus vulvæ, is in a condition peculiarly susceptible to the action of remedies of this class. These drugs should always be regarded as the last resort of the practitioner, and a confession of weakness in not discovering the special cause effective in the case.

Cathartics and laxatives and an abundant supply of pure water internally, employed as directed for relief of acute eczema, as well as diaphoretics and diuretics, are often of value in eliminating toxins to which pruritus may be due, in depleting the cutaneous vessels, and possibly, in a reflex way, by diverting irritation to other regions. Jaborandi and pilocarpin have thus been employed to advantage. In children full doses of quinin sometimes relieve pruritus, while in adults large doses of calcium chlorid[1] occasionally will accomplish the same result. Cannabis indica and gelsemium at times are effective, but should be prescribed with great caution. Acetylsalicylic acid is probably the least harmful and the most efficient of internal remedial agents.

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[1] Savill, London Lancet, August, 1906.

The indications for local treatment are to protect the skin from all sources of irritation and to relieve the itching. Hyperesthesia of the skin is common in pruritus, either as a predisposing cause or as a result of long-continued itching. In consequence, very slight external irritation may suffice greatly to aggravate the itching, and every precaution should be taken to protect the skin from exposure of all kinds. First in importance is the clothing. The garments worn next the skin should be of cotton, lisle-thread, linen, or silk, never of wool, and the meshes should be filled with an impalpable powder to reduce to a minimum the friction of the garments on the skin. All other clothing should be as light as possible and yet be warm enough for protection. If the patient live in a climate where sudden changes in temperature are common, the clothing should be regulated accordingly. The object is to keep the skin at an even temperature and to protect it from sudden changes. In cases in which the pruritus is due largely to the hyperesthesia, the itching may be entirely relieved by dusting the surface with a simple powder and completely covering it with a layer of cotton-wool or other protective dressing.

Hot baths, unless specially indicated, and the too free use of soap may render the skin unduly sensitive. The bran, oatmeal, alkaline, and other demulcent baths recommended in the chapter on General Therapeutics are those most generally useful. After the bath, the surface should be patted (not rubbed) dry and covered with a dusting-powder or other selected application. When the skin is free from excoriations and other lesions, the cold douche, alternate hot and cold douching or sponging, or even the cold salt-water sponge, may be used to improve its tone and vigor.

Scratching is a common source of irritation and one that is difficult to set aside. Until this is accomplished, however, relief cannot be obtained, as wherever the skin is scratched or rubbed there is produced a local hyperemia, or even a dermatitis, which adds to the cutaneous irritation, not only at the site of the rubbing, but also by reflex action in other regions of the body. It is not sufficient to tell the patient not to scratch; the surface must be protected by proper dressings, and the itching relieved by the use of antipruritics. Bronson suggests that patients be allowed to obtain relief at times by firmly pressing upon the surface or by gently drawing over it an oiled or a wet cloth.

The substances which have been employed topically for the relief of pruritus cutaneous are almost without number, a fact warranting the conclusion, corroborated in every wide experience, that each occasionally fails to afford the desired relief. That preparation, moreover, which is at one time of the highest value, at another period in the history of the same case will disappoint. Attempts to secure relief by such topical applications should, however, always be made, and will often be followed by gratifying results.

The sedative and antipruritic lotions, lead-water, lead-and-opium wash, liniments, and dusting-powders described elsewhere, together with their methods of preparation and application, are valuable and sufficient in most cases. They may be further modified by the addition of substances recommended in the following paragraphs. The dusting-powders are of special value in furnishing mechanical protection. When a decided antipruritic effect is desired, the Anderson, or a similar powder, may be used. In some localized forms of pruritus, more complete protection with ointments, pastes, or even the glycogelatins, may be secured.

Of all antipruritics, phenol easily takes first place. In most of the lotions recommended above it is used in strength of 1 to 5 per cent. In oils or liniments it may be used much stronger. Bronson uses it even to 25 per cent., stating that it is much more slowly absorbed than in aqueous solutions, and therefore less likely to produce systemic effects. A favorite formula with him is the following:

℞—Phenolis.,	ʒj–ij;	4–8	
Liq. potass.,	ʒj;	4	
Ol. lini.,	℥j;	30	M.

It is to be shaken before using, and may be scented with bergamot. These stronger preparations of phenol, even in the oils, should be used over only small areas, for fear of toxic effects. The possibility of producing gangrene by the long-continued application of even weak solutions of phenol should not be forgotten.

Other remedies that may be used in lotion, oil, liniment, ointment, or paste, in strengths varying from 1 to 5 per cent. or more, are: salicylic acid, hydrocyanic acid, menthol, camphor, thymol, salol, creosote, the tars, chloral, and chloroform. Two or more of these remedies may be combined in the same lotion. Morphin, atropin, and cocain may be added to lotions with occasional advantage.

Ointments and pastes are irritating to many pruritic skins, but at times are more acceptable than the lotions and oils. In abnormally dry skins and in some cases of bath-pruritus, a simple oiling of the skin often gives prompt relief.

Chloral-camphor, a pungent, syrupy liquid obtained by triturating an equal amount of the two substances to a fine powder, is an antipruritic remedy of value in certain cases if applied in a salve containing 1 drachm (4.) to the ounce (30.) of salve. Among other remedies occasionally of service are ichthyol, resorcin, and mercuric chlorid. Bronson speaks highly of hydrogen peroxid. In atonic cases, with diminution of the tactile sense, the use of electricity over the spine has been followed by good results.

In senile pruritus the progressive atrophy and degeneration of tissues may be checked or retarded by management proper to each case. Locally, electricity or hot and cold douches may aid in stimulating the skin to renewed vigor. Keeping the skin soft with daily inunctions of oil or a thin ointment is an effective measure in many cases. Lastly, many cases of intractable pruritus are best managed when the attention of the patient is diverted from the malady by the distraction incident to travel, aided by change of scene and climate.

Treatment of the regional forms of pruritus is that above described, with such modifications in the dressings as may be necessitated by the special location.

Locally, heat in the form of hot oatmeal-water or ordinary hot water gives prompt and sometimes lasting relief. This may be employed each evening before the medicament selected is applied. The following articles give most relief in the local cases: phenol, in oil or lotion; thymol or menthol, in an ointment; resorcin, in lotion or ointment; camphor, in an ointment or dusting-powder; chloral, in an ointment or lotion; and the tars, in lotion or ointment. Particular directions must be given concerning the proper method of applying these preparations and their subsequent reactions.

Fissures and areas of infiltration may be painted with compound tincture of benzoin, tincture of iodin, or solutions of silver nitrate containing gr. x to ʒj (0.66–4.) to the ounce.

For pruritus of the vulva, Wiltshire[1] recommends decoctions of almond-meal, marshmallow, slippery-elm, and rice; and in case of failure of the latter, an infusion of tobacco, 2 ounces (60.) to the pint (480.). Vaginal injections of hot water and tampons or cocoa-butter suppositories, medicated with opium, belladonna, or phenol, are also available. Mercuric chlorid lotions, Gr. ¼-j to ℥j (0.016–0.06 to 30.), are recommended by many writers.

Iodoform, cocain, oleate, and hydrochlorid, the latter in from 2 to 4 per cent. solutions; 1 ounce (30.) of the fluid extract of coca, to 2 or 4 (60.–120.) of water; and linseed oil (especially for pruritus ani), are also recommended. Jullien recommends in pruritus of the vulva:

℞—Zinc. oxid.,	ʒvj;	24	
Acid. salicylic.,	gr. xv;	1	
Glycerin.,	ʒvj;	24	M.
Sig.—Apply as required.			

Chéron, in pruritus of the vulva attending the menopause, has successfully used:

℞—Veratriæ,	gr. iij;	20	
Axung.,	℥j;	30	M.

He also administers in pill form $\frac{1}{120}$ grain of veratria rubbed up with licorice. Another useful formula is:

℞—Acid. tannic.,		℈j;		1 33	
Alcoholis,					
Glycerin,	āā	℥ss;	āā	15	
Aq. dest.,	ad	f℥iv;	ad	120	M.
Sig.—Apply morning and evening on a rag.					

On account of the peculiar predispositions of the sex, anodyne preparations are attended with danger as regards the future habits of the patients and should be used with caution.

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[1] Brit. Med. Jour., 1881, i, p. 327.

Surgical treatment of pruritus ani and pruritus vulvæ has been employed, even to complete ablation of the external genitalia.[1] Sir Charles Ball[2] produces anesthesia of the perianal region by dissecting away the skin in an oval about the anus and replacing the flaps after dissection. In several cases thus treated itching has recurred, and as a rule such measures are not to be recommended. Radiotherapy has proven to be the most valuable of all methods of local treatment. It should be employed cautiously, and a definite dermatitis should not be produced, on account of the subsequent telangiectasia.

In the treatment of pruritis hiemalis, it is essential to see to it that the patient has proper clothing. Cotton should be worn next to the skin, but extra clothing may be worn over this. Corlett advises resorcin as a local application, and believes internal treatment is of no value. Menthol and phenol are the two chief antipruritics employed. Bronson advises bandaging the limbs and smearing them with a 5 per cent. salol, superfatted soap. Owing to the harsh, dry condition of the skin, the following treatment has been successful: In the evening an ointment containing menthol, 1 part; salicylic acid, 1 part; pulvis. amyli. 30 parts; unguentum aquæ rosæ, 30 parts; and unguentum petrolatum to make 100 parts, is applied in thin coating and dusted with talcum powder. Upon rising in the morning, an oily preparation containing 25 parts of glycerin and 75 parts of olive oil may be gently massaged in and also dusted with the powder. In these cases warm weather is always curative.

Prognosis.—Pruritus senilis is usually an intractable disorder, and when dependent upon senile alteration of the cutaneous tissues is incurable. For all other forms of the disease a prognosis should be formulated with reserve. Under the influence of systematic and appropriate treatment the happiest results are often obtained. Other cases, especially those associated with hypochondriasis, may bid defiance to all medicinal measures. Relapse of the local forms of the malady, especially that of the ano-genital region, is common. In many of these patients the treatment serves merely to palliate.

ERYTHROMELALGIA.[3]

Definition.—Under this title Weir Mitchell, in 1872, described a chronic disorder in which one or more portions of the surface of the body, particularly over the extremities, became the seat of pain, redness, and local elevation of temperature. Since then Graves, Eulenberg, Senator, Mackenzie, and others have described cases more or less allied to the type originally depicted.

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[1] Illinois Med. Jour., 1905, p. 45 (Howard Kelly's and Borke's method).

[2] Brit. Med. Jour., January 21, 1905, p. 13.

[3] Mitchell, Weir, Phila. Med. Times, 1872, pp. 81 and 113; Amer. Jour. of Med. Sc., July, 1878, p. 17; Clinical Lessons on Nervous Diseases, 1897, p. 177. Graves, quoted by Crocker, p. 715. Mackenzie, Stephen, Brit. Med. Jour., 1879, ii, 704. Morrel-Lavellée, Bull. Soc. franc. de Derm. et Syph., Paris, 1891, ii, 354. Eulenberg, Verhandl. der Berlin med. Gesselshaft., Erster Theil, 1892, p. 239; Neurolog. Centralb., 1893, p. 657. Senator, Berlin klin. Wochenschr., 1892, p. 1127. Lewin and Benda, ibid., 1894, xxxi, pp. 53, 87, 117, and 144 (with references to date). Prentiss, Trans. Soc. Amer. Phys., Philadelphia, 1897, p. 303. Rolleston, Lancet, 1898, p. 783. Mitchell and Spiller, Amer. Jour. Med. Sc., 1899, cxvii, p. 1. Voorhees, Jour. Amer. Med. Assoc., June 1, 1907, xlviii, p. 1837. Barlow, Allbutt and Rolleston's System, 1911, vii, p. 149.

Symptoms.—The symptoms are developed usually in persons of early middle age, in men more often than in women, particularly in those who are engaged in physical labor requiring an erect posture. After a variable period of malaise or suggestions of ill-health, a burning pain occurs in one or more parts of the extremities, made worse by posture and movement, but commonly relieved in the recumbent position. The sensations are those of burning and pain, which recur with exaggeration after prolonged exertion in the erect posture and are soon followed by redness over the fingers, the toes, the heel, or the external or inner face of the foot. One or all digits and one or all members may be involved. The arterial pulsation becomes excessive and the veins distended, with throbbing of the part. The hyperesthesia to heat, cold, and pressure may be excessive; the reflexes are normal or exaggerated; the surface temperature of the parts affected rises two or three degrees above the normal; the swelling is slight; sometimes there is pitting on pressure. All symptoms are relieved by rest and cool applications.

In some cases the hands as well as the feet are involved, and pains occur in the head, neck, shoulders, elbows, and other parts. In other cases, instead of redness, the parts exhibit a special pallor. Mitchell lays great stress in diagnosis on the color of erythromelalgia—rosy-red, later purplish-red, as distinguished from the livid-red in Raynaud's disease. In some cases vesicles form; in others pinhead-sized nodules; in yet others the subcutaneous tissue is indurated; the finger-ends become tense and shining; or there are clubbing and thickening of the nails. In extreme cases the muscles of the limbs have become somewhat wasted, probably from disuse of the limb.

Etiology and Pathology.—The disorder occurs most frequently in middle life and in the male sex, in those engaged in heavy physical labor in varying temperatures. A few cases have occurred in patients between the ages of sixteen and twenty-one. There are two hypotheses concerning its causation: one connecting the disease with the nervous system, the other connecting it with the vascular system. Hysteria has been noted in association with the disorder, and it has followed certain severe fevers. Traumatism seems to have been effective in a few cases. Galloway[1] concludes that the disease is one dependent upon changes in the bloodvessels, of the nature of an obliterating arteritis. It has been noted to concur with paralysis, Graves' disease, tabes, multiple sclerosis of the cord, alcoholism, osteomalacia, and myelitis. A peripheral neurosis is recorded in a case studied by Mitchell and Spiller. Several instances of its occurrence in association with Raynaud's disease are recorded by Morrel-Lavallée. Lewin and Benda conclude that the morbid condition is rather a symptom complex than a malady *sui generis,* the external phenomena being due either to central or peripheral irritation, including either functional or organic change.

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[1] Allbutt and Rolleston's System, 1911, ix, 71.

Diagnosis.—The striking features of the disease are burning pain, at times intense, associated with swelling and redness, the latter being greatly increased when the part is in a dependent position, and largely relieved when the effect of gravity is removed. The rosy-red and later purplish-red hue of the affected parts differs from the livid-red seen in Raynaud's disease. In the latter condition, early in the disorder the affected part returns to normal; in the former it remains unchanged from the onset. Raynaud's disease, also, occurs at an earlier period of life.

Treatment.—The best results are secured by long rest in the recumbent position; faradization of the nerve-centers; high-frequency currents; cold applications during paroxysms, when required; and a nutritious dietary. Nerve-stretching and nerve-excision have been followed by questionable results.

Prognosis.—The patients exhibiting these symptoms are rarely improved by treatment, the disease often progressing for years with gradual aggravation of the symptoms. The few cases of recovery are important. Excision of a portion of the tract of the nerve and stretching in other cases have been found of questionable value. *X*-rays were used with improvement in the condition by Sutton and Kanoky.[1]

DERMATALGIA.

Synonym.—Neuralgia cutis.

Definition.—In this morbid state the integument becomes the seat of painful sensations, which may or may not be associated with a hyperesthetic condition. This disorder is much more frequently partial than general, being in the larger number of cases a local expression of some disease of the nervous centers or tracts. It is observed usually in middle life, and in women more than in men. Its symptoms vary in severity from a slight burning to a state of torture that defies description. In character the pain is variously described as comparable to that produced by friction, incision, penetration, contusion, or burning of the integument; as also to the passage over the part of streams of very hot or cold water, or the electric current. With this there is commonly associated an undue sensitiveness to contact with foreign bodies. The skin presents no objective signs of disease. The disordered sensations may be limited to the scalp, the region of the spine, or the palmar and plantar surfaces. In the latter situation it is often significant of some obscurely developed systemic disease, such as syphilis, rheumatism, or locomotor ataxia. In a middle-aged woman a persistent dermatalgia of the interscapular region was associated with confirmed gastric dyspepsia (Hyde). In other cases the disorder is dependent upon disturbance of the uterine function. It is occasionally observed as one of the rare signals of the occurrence of the menopause.

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[1] Jour. Amer. Med. Assoc., December 19, 1908, p. 2157.

It is to be noted that the severe dermatalgia associated with disorders of the uterus in women is occasionally succeeded by a cutaneous lesion. Buck[1] reports dermatalgia of the brows and wrists in a young woman who had frequently miscarried, followed by recurrent formation of a vesicle, which accomplished its career of rupture, crusting, and erosion in a stadium of from five to seven days.

Diagnosis.—The disease is to be differentiated from hyperesthesia of the skin, with which it frequently is associated, and from which it often cannot be distinguished with certainty, as it is not possible always to exclude slight sources of external irritation; and, further, the diagnosis must be based largely upon the observations and statements of the patient. Painful affections of deeper parts, muscular, nervous, aponeurotic, and visceral, must also be excluded. Severe pain limited strictly to the skin of the lumbar region, with hyperesthesia, may precede the occurrence of perinephritic abscess.

Treatment.—The treatment is to be directed to the disorder of which, in the great majority of cases, the dermatalgia is merely a local symptom. Quinin, the salicylates, iron, arsenic, and zinc phosphid are often indicated. Temporary relief, however, may be afforded by the local application of a rubber bag filled with very hot or very cold water; sometimes by an alternation of the two, each for a few moments at a time. Sponging the part with very hot water is also useful, continued for longer periods, and followed by swathing in cotton-wool covered with Lister protective. High-frequency currents over the cord are often of special value. In some cases anodynes may be used topically with advantage, especially cocain, opium, aconite, belladonna, or stramonium in oily combinations. In other cases relief is had by painting the parts with Squibb's mercuric oleate and morphin. The skin should generally, in the intervals of application, be protected by a dusting-powder; and the clothing worn next to the skin should be of an unirritating character.

Causalgia. Causalgia is a term, chiefly employed by Weir Mitchell, to designate a sensation of burning and pain, with tenderness, occurring in different regions of the integument affected with "glossy skin," and occurring as a complication or sequel to peripheral nerve injury. The sensitive area in these cases is supplied by the filaments of a single nerve.

Causalgia is distinguished from the pain of neuralgia or sciatica by the localized affection of the skin which is the seat of the abnormal sensation; and from the pains of rheumatism by the absence of the muscular soreness accompanying this last disorder. In the gouty state, with erythematous areas, often shining, over the painful joints, the recognition of any distinction may be attended with difficulty. In pruritus, the itching sensation is invariably the predominant symptom.

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[1] Phila. Med. and Surg. Reporter, 1881, p. 677.

Treatment.—In a group of three cases complete relief was obtained by the intraneural injections of 60 per cent. alcohol by Lewis and Gatewood.[1]

Various changes in the cutaneous sensibility have in the past been described as distinct diseases. They are important symptoms in many disorders of the nervous system, a few of them being also of importance in connection with the cutaneous system. The most important of these changes are the following:

Hyperesthesia, which indicates an exaggerated sensitiveness of the skin to external impressions; *hyperalgesia*, which indicates a condition in which the sense of pain is greatly exaggerated, while that of touch is diminished; cutaneous *anesthesia*, a condition in which one or all of the elements of cutaneous sensation are partially or wholly lost, an important symptom in determining the presence or absence of many nervous disorders, and which may be due either to central or peripheral involvement of the nervous system; and *paresthesia*, which denotes a perversion of cutaneous sensibility, as the result of which a given stimulus produces a sensation different from that which it would produce in health.

MERALGIA PARESTHETICA.

Under this title White[2] called special attention to this particular variety of paresthesia, which had been frequently recognized by neurologists, but had been apparently overlooked by dermatologists.

The perverted sensations are limited to the skin on the antero-external surface of the thigh, a region supplied by the external cutaneous nerve, a branch of the obturator. The disorder occurs usually in middle-aged men, but occasionally in women. It has been known to follow trauma, and flat-foot is given as a possible etiological factor. Holmes[3] states that the symptoms are probably due to compression of the nerve as it passes through the deep fascia when the latter is tense. In a small proportion of cases the condition is bilateral. The symptoms described represent various anomalies of sensation, such as tingling, numbness, coldness, pain of varying degrees, and formication. These are exaggerated on walking or standing, but are usually relieved when the patient is in a recumbent posture. In the major portion of cases, tenderness may be elicited below the anterior superior iliac spine, where the nerve pierces the fascia. In White's case, on the production of artificial hyperemia, the part became congested, the purplish hue receded more slowly than on the opposite side, and on testing with a needle-point the whole area was found to be less responsive than normal, and toward the lower portion, just above the patella, anesthesia was complete.

The disorder has proven intractable to treatment. Rest, massage, and faradism have been recommended. Resection of the nerve has been practised, with relief in some cases but with recurrence in others. Goldenberg[1] recorded the relief of a patient upon wearing a metal plate to overcome flat-foot.

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[1] Jour. Amer. Med. Assoc., 1920, lxxiv, p. 1; Treatment of Causalgia.

[2] Jour. Cut. Dis., 1906, xxiv, p. 160.

[3] Osler's Modern Medicine, 1910, vii, p. 490.

TRICHOTILLOMANIA.[2]

This term signifies a condition in which there exists in an individual an uncontrollable desire to pull out the hair. The hairs of the scalp, brow, and beard are the ones usually attacked. It occurs in neurotics, who, however, are otherwise normal.

Hallopeau (quoted by Brocq) has described under the name *trichotillomanie*, and Raymond under the name *tic de l'épilation*, a morbid state characterized by itching sensations of the body, exaggerated in the hairy regions, which leads the patient to seek relief by pulling out the hairs of the affected areas. Brocq states that the disorder consists essentially of an impulse (a true mania), which leads the patient to pull out the hair. He further states that no lesion has been demonstrated either in the hairs or in the affected areas, and that the condition should not be confused with that in which the hairs are rubbed off in areas characterized by itching. The hairs may be removed in small areas or quite extensively. Cases of this type have been recorded by Sutton, Pernet, Blaisdell and others.

The condition is to be differentiated from diseases associated with alopecia, and its management is directed toward the cure of the deranged mental condition.

Other conditions referable to the skin induced by mental derangement occur. At times patients are seen who believe themselves infected with insects. This peculiar mental disorder, mention of which was made in the section devoted to Pruritis, is termed "acarophobia" by Sutton.[3] A number of examples have been seen by the author, all presenting similar symptoms in varying degree. The subjects imagine themselves covered more or less extensively with insects or parasites, which they are constantly capturing and destroying. In one family three members were affected, the mother and two children; in another the husband and wife; and several isolated examples have been seen. Various substances, such as collections of epithelial débris, bits of cotton or wool admixed with dirt, and other extraneous matter collected upon the skin, are viewed by these subjects as the active and live cause of their troubles. Any portion of the cutaneous surface may be attacked, and in one patient imaginary insects were removed from the mouth and nose. In some cases intense itching is present, and then the lesions of a traumatic dermatitis occur.

The management of this condition presents a difficult problem, and the prognosis should be guarded.

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[1] Jour. Cut. Dis., 1906, xxiv, p. 163.

[2] Brocq, Dermatologie pratique, ii, p. 395. Sutton, Jour. Amer. Med. Assoc., 1914, lxiii, p. 2126: report of two cases. Pernet, Brit. Jour. Derm., 1915, xxvii, p. 55; Trichotillomania associated with other Factitious Manifestations. Blaisdell, Jour. Cut. Dis., 1916, xxxiv, p. 363: report of two cases.

[3] Loc. cit. (case report).

CLASS VIII.

PARASITIC AFFECTIONS.

DISEASES DUE TO VEGETABLE PARASITES.

FAVUS.[1]

Synonyms.—Honeycomb ringworm, Porrigo favosa, Tinea favosa. Fr., *Teigne faveuse;* Ger., *Erbgrind.*

Definition.—Favus is a parasitic affection, usually affecting the scalp, chronic in course, and characterized by yellowish, cup-shaped crusts, having a peculiar "mousy" odor. It is caused usually by *Achorion Schönleinii.* It terminates in atrophy or scar-formation.

Symptoms.—Favus affects the scalp chiefly, but it also occurs upon the glabrous portions of the skin and upon the nails. In the former situation it is usually first recognized by the development of minute, subepidermic, yellowish or reddish puncta, visible through the translucent stratum corneum at the site of implantation of the hairs. A circle of delicate vesicles may surround these spots. Puncture with a needle usually gives exit to puriform matter. In the course of a fortnight or more, these lesions cover themselves with pinhead- to pea-sized and somewhat larger, friable, circular, and elevated crusts, having the yellowish tinge of the lemon or of sulphur, and a concavo-convex shape, with the free concave face of the disk exposed. At the center of the umbilication thus presented to the eye one or several hairs usually make exit to the surface. The inferior surface of this disk or scutulum rests upon the scalp, which is either moist and deprived of its epidermis over a circumscribed area, or is smooth, dry, reddened, and tender. When the crust is removed by traction upon the hairs or otherwise, a minute cup-shaped depression is left at the point where the lowest level of the favus crust was in intimate connection with the epidermis.

The subsequent features of the crusts, the hairs, and the scalp are subject to variation. The crust may acquire a brownish or a greenish tinge by admixture with dirt or with dried pus; may coalesce, or may, by gradual desiccation, exchange the yellowish hue for the dirty-whitish

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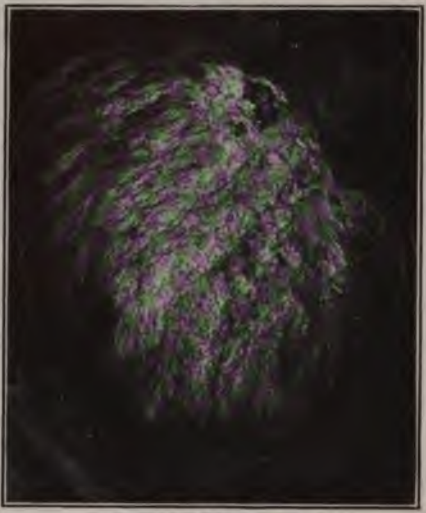
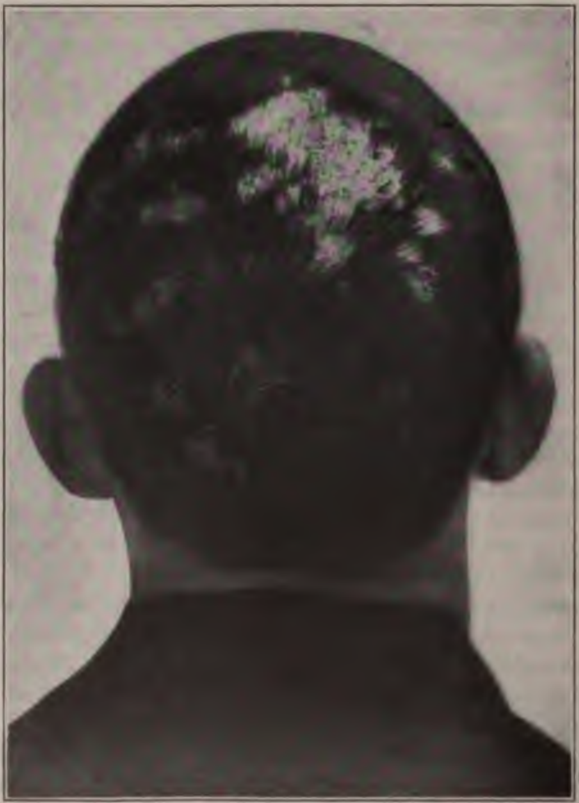
[1] For bibliography, see Bodin, La Pratique Dermatologique, ii, p. 617.

FIG. 343



Favus of the scalp.

FIG. 344



Favus capitis. (From G. H. Fox's Atlas of Skin Diseases.)

shade of old mortar, a substance which they then resemble in dryness and friability (*Favus squamosus*). The hairs, invaded both in the sheath and shaft, may lose their luster; become fragile; appear as fractured stumps; be readily extracted from their follicles; and finally be shed, leaving an atrophied hair-follicle. The scalp may first be the seat of an extensive hyperemia or exudation, going on to the formation of pus, when the fungus is a source of acute irritation in consequence of its active development. Later, when its destructive work may be said to have been accomplished, the scalp-surface is bald, irregularly atrophied, or disfigured with cicatrices, which at first are of a deep-red color, but which gradually fade, while here and there remain tufts of hair that have survived the attack. The lesions may be discrete or be confluent, and may vary in either case. Occasionally, but a few small and ill-developed crusts form upon the surface. The entire scalp is rarely covered with a confluent favus crust.

The disease is usually chronic in its course and develops slowly. Untreated, it may undergo spontaneous involution, after total destruction of all hairs and production of general follicular atrophy, but this is rare. It may last for fifteen or twenty years, or even longer. It is often accompanied by adenopathy.

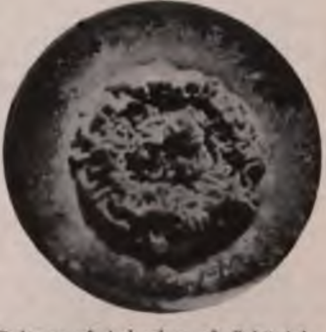
There is usually a moderate degree of itching, as the result of which the favus crusts may be torn and broken by the comb or the nails.

In favus of the body-surface, outside of the scalp, there is often a resemblance to ringworm in the production of circular patches, with an active border made up of vesicles or of papules, which may have a favus scutulum as a center; or several of these cups may be spread irregularly over circles of scaling patches. In these cases there is often an acuity of symptoms not observed in scalp cases and coincident gastro-intestinal signs of irritation, as vomiting, which Kundrat believes may originate in favus of the mucous surfaces of the esophagus and gastro-intestinal tract.

The lesions may occur on any part of the surface of the face, trunk, and extremities, being found most frequently on the hands, the upper and lower extremities, and the shoulders. It is a striking fact that favus may exist for years in the scalp without spreading to the trunk or elsewhere.

The lesions on the human skin produced by *Achorion Quinckeanum* ("mouse favus") have been of the erythemato-squamous type, resembling ringworm of the body. The odor of fully developed favus is so characteristic that by it alone a diagnosis has been established. It is usually compared to the odor of mice; also to that of the urine of cats. It should not be confounded with the

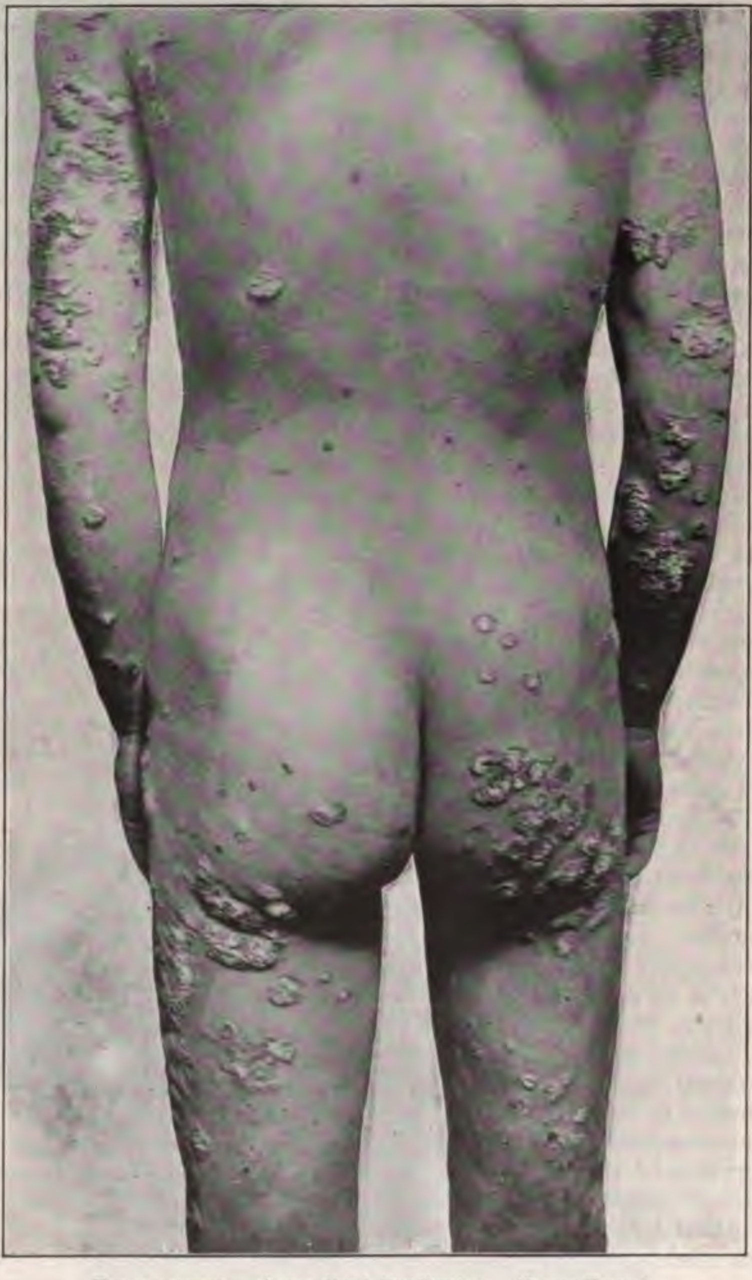
FIG. 345



Culture of Achorion of Schönlein. (Mewborn.)

peculiarly disgusting odor of neglected scalps affected with lice or covered with pustules and filth. The disease not infrequently co-

FIG. 346



Favus corporis. Generalized distribution. (James Dunn.)

exists with other cutaneous parasitic and non-parasitic diseases, as, for example, with pityriasis of the scalp, or eczema.

Etiology.—Favus is practically always produced by the presence and development of the vegetable organism which is named after its discoverer, *Achorion Schönleinii.* Sabouraud[1] states that 99 per cent. of cases are produced by this organism. Two other varieties, *Achorion Quinckeanum* (Bodin) and *Achorion gypseum* (Bodin[2]) are found, but such cases are rare.[3] The disease is contagious, and the parasite which produces it is capable of transmission from man to man and rarely from animals to man. It shares with other diseases originating from vegetable parasites the peculiarity of attacking certain individuals especially predisposed to such invasion, either by reason of physical peculiarities of organization or because of accidental and fortuitous circumstances. It is most common from infancy to the thirtieth year of life. It is less common in the United States, Austria and England than in France, Scotland and Poland. It is said by Bergeron[4] to be a disease of the country, while trichophytosis capitis prevails in the cities. This statement is corroborated by general experience. Favus is more common in public than in private practice, and the larger number of clinical patients with favus come to the city from the country. Little[5] recorded favus of the scalp and glabrous skin in a woman the wife of an army officer.

Evidences of contagion are exhibited in those cases in which several members of the same household are affected with the disease. Its contagiousness, however, is not marked. Géber,[6] in a study of twelve cases, seven of which appeared to have been contracted from a woman who had suffered with favus of the scalp for twenty years, fixes the incubation period at from six to ten days. Aubert[7] presents an argument in favor of the production of the disease by traumatism, the resulting wounds becoming by accident the seat of the disease.

Pathology.—*Achorion Schönleinii* may be found in the scutula, the hairs, and the surrounding scales. The fungus is found chiefly between the superficial and deeper layers of the stratum corneum, but it may at times invade the rete and even the corium. The scutulum is constituted chiefly of mycelium, spores, and granular débris. In the center spores are abundant, while externally there is a dense mycelial mass. The mycelial filaments are directed perpendicularly or toward the periphery of the cup. Surrounding the scutula there is an inflammatory reaction in the skin, evidenced by a leukocytic and plasma-cell infiltration, and beneath the cup there occurs a rarefaction of the connective tissue.[1] The process results finally in destruction of the normal structures, and scar-formation ensues.

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[1] Brit. Med. Jour., October 10, 1908, p. 1089.

[2] Annales, October, 1907, p. 586.

[3] Adamson, Brit. Jour. Derm., 1908, xx, p. 365; and ibid., 1911, xxiii, p. 49: Culture of *Achorion Quinckeanum* (mouse favus). (This was the third case of this variety seen) Sequeira, ibid., 1912, xxiv, p. 31. MacLeod, ibid., 1913, xxv, p. 1380.

[4] Étude sur la Géographie et la Prophylaxie des Teignes, Paris, 1865.

[5] Brit. Jour. Derm., 1909, xxi, p. 26.

[6] Archiv, 1912, cxiv, p. 101.

[7] Rôle de Traumatism dans l'Étiologie de la Teigne faveuse, Annales, 1881, s. ii, ii, p. 289.

Jackson and McMurtry[2] describe the development of the scutulum as follows: The parasite, after being deposited upon the skin, proliferates upon and in the horny layer of the epidermis, and upon encountering a hair-follicle the growth proceeds downward in the epithelial sheath toward the bulb of the hair. As the result of this, inflammatory reaction occurs, producing first the pustule and later the characteristic scutulum as above described.

The fungus in the hair-shaft occurs as polymorphic mycelial threads, which lie parallel with the long axis of the hair and terminate near the root in a fringe. The mycelia divide dichotomously at acute angles, the individual threads being 2 to 3 microns or more in width and the joints 4 to 15 microns apart. They contain no spores, but terminate in rows of spores, termed the *Favic tarsus*. The filaments may be replaced by air, which produces a characteristic appearance when the hair is examined in potassium hydroxid.

Diagnosis.—The clinical recognition of favus is based upon the presence of the characteristic yellowish, cup-shaped crusts, which in all typical cases are isolated, each pierced by a hair and each situated in a well-marked depression of the surface of the scalp. In the disseminated form the disks of conglomerated scutella, with defined and frequently festooned edges, friable, yellowish or yellowish-white in color, and greatly differing as to their bulk and contour, are commonly suggestive of the nature of the disorder. In yet other cases the affected area seems to be covered with a plaster-like mass of uneven thickness, irregularly distributed over an enormous patch of disease, which may be practically coextensive with the entire scalp-surface. Incidentally, there may be a history of contagion and a peculiar odor emanating from the scalp. The secondary effects upon the hairs, hair-follicles, and skin are also, when present, significant. In cases of long duration, the atrophy, scar-formation, and permanent baldness are characteristic. White, of Boston, in an essay on the "Vegetable Parasites, and Diseases Caused by their Growth upon Man," calls attention to the stage in which the disease is likely to be mistaken for ringworm. It exists before the formation of the crust, and may be characterized by hyperemia, vesiculation, or papulation, often unnoticed, beneath the hairs of the scalp. In doubtful cases a microscopic examination of the fungus or its cultural characteristics will usually establish the diagnosis.[3]

Aubert,[4] in the absence of the clinical features named above, lays stress upon an intense redness of the scalp where the hairs have been cut and the crusts removed, this color being limited to the portions attacked by the disease. The hairs also, as a result of disintegration of their elements, are infiltrated with air, and look opaque and black by transmitted light; by reflected light they appear polished and stratified, as if constituted of layers of tissue. It should not be forgotten that in exceptional cases favus-crusts coexist upon the body with other diseases of prior or of subsequent origin, as indicated. The disease should not be confounded with seborrhea, pustular eczema, or psoriasis of the scalp, none of which exhibits the special features of a parasitic fungus.

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[1] MacLeod, loc. cit., p. 365.

[2] Diseases of the Hair, 1912, p. 264.

[3] For literature on this subject see Mewborn, Jour. Cut. Dis., 1903, xxi, p. 11 (illustrations and bibliography, and the references tabulated with the introductory paragraphs on Ringworm).

[4] Annales, 1881, s. ii, ii, p. 34.

Treatment.—Radiotherapy offers the best method of treatment. Months and years are consumed in its relief without the employment of this method. As the disease produces scars and permanent alopecia, it is well to use any method that will insure prompt relief. The single-dose method, as practised by MacKee[1] and others, may be employed, or repeated dosage until epilation is produced.[2] If radiotherapy is not available, the first indication in the treatment of favus is to cleanse the affected surface from all crusts and scales that may be present. On the scalp the hair should be cut short, after which this area is thoroughly soaked with olive, cod-liver, or other oil, or with glycerin. After this treatment all the crusts are scraped off with a spatula, and the scalp is washed clean with hot water and soap, spirit of green soap being preferably used here. The scalp should then again be anointed with oil or be covered with a soft ointment. Once thoroughly cleansed by repeated soakings with oil and by ablutions, it is necessary to resort either to the topical employment of parasiticides or to epilation. Often both measures are required. Without further treatment the scalp, however completely freed from all evidences of the disease, will not fail to show fresh favus crusts in a fortnight or somewhat longer time.

Epilation is practised with the aid of epilating forceps. These forceps should be constructed with an easy spring that will not tire the fingers of the operator; with blades that are sufficiently broad to grasp a few hairs at once; and with smooth or but slightly serrated faces to the blades, as otherwise the hair is liable to fracture in the grasp of the instrument. The surface to be operated upon should previously be anointed with vaseline or with olive oil, and the hairs entirely removed, a sufficient number, covering a definite space, upon successive days.

The tediousness of this process has led to several devices by which it is sought to do away with its necessity. Originally the "calotte" was employed for the removal of the hairs; it was made by smearing a disk of leather with pitch, and applying it over the scalp. When the calotte was subsequently removed by a brisk twitch with the hand, the hairs which adhered were forcibly uprooted *en masse;* those remaining being adherent in their sacs in consequence of the fact that they had not been invaded by the fungus. As a substitute for this procedure, Bulkley[1] employed adhesive masses or sticks, which can be melted and made to adhere at once to large numbers of the hairs. When cold they can be withdrawn from the surface with the hairs attached. These sticks are from two to three inches in length, and from one-fourth to three-fourths of an inch in diameter. The hair is first clipped so as to be about one-eighth of an inch in length. The end of the stick is then heated in an alcohol flame, and quickly placed upon the scalp. It is thus left in place until cold, and is removed by bending it over and drawing upon the hairs successively with slight rotation. When free it is found thickly set with the extracted filaments, which may be burned off in the alcohol flame, thus destroying both the hairs and any adherent fungous masses. The stick is then carefully wiped clean with paper, after which it is again ready for use. The formula for the mass of which these sticks are composed is as follows:

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[1] Jour. Cut. Dis., 1912, xxx, p. 544.

[2] Geyser, ibid., 1909, xxvii, p. 321; Howard Fox, ibid., p. 356; Ormsby, ibid., p. 366, and ibid., 1913, xxxi, p. 353; Dally, ibid., 1911, xxix, p. 517, abstr. from Archives of Roentgen Ray, October, 1910.

℞—Ceræ flavæ,	ʒiij;	12	
Laccæ in tubulis,	ʒiv;	16	
Resinæ,	ʒvj;	24	
Picis Burgundicæ,	ʒxj;	44	
Gummi Dammar,	℥jss;	45	M.

The parasiticides in greatest favor are: mercuric chlorid in solution in the strength of from 1 to 4 grains (0.066–0.266) to the ounce (30.); formaldehyd (1 to 4 per cent.); sodium hyposulphite in saturated solution; pure or diluted sulphurous acid; spirit of green soap; chrysarobin, pyrogallol, tar, croton-oil; boric and salicylic acids; phenol, petroleum, chloroform, ether, creosote, and oil of cloves. The addition of acetic acid to liquid applications, or washing the surface with vinegar immediately before applying the parasiticide, favors penetration of the remedy. Ointments containing mercury (citrine ointment, yellow sulphate, or white precipitate), naphtol, benzol, thymol, sulphur, pyrogallol, phenol, and salicylic acid are also useful. Chrysarobin is effective in an ointment, though objectionable on account of the staining of the scalp, and almost inevitably of the face also. Lenzberg[2] generates sulphur-fumes in a dish of red-hot coals attached to a frame (made of wood or of pasteboard) close to the head of the patient. By means of a paper cap the fumes are collected and retained (from five to ten minutes) in contact with the patient's hair. During ten years' trial of this plan he has never been compelled to resort to epilation.

One or more of the methods may be needed, either at the same time or by repetition or alteration, until the fungus is entirely destroyed, the requisite period usually extending over three months. Treatment should then be discontinued in order to test the result by observation. If, in the course of a fortnight or more, a relapse occurs, treatment is to be promptly renewed. Upon the non-hairy portions of the body parasiticides thoroughly applied usually insure radical relief.

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[1] Favus and its Treatment by a New Method of Depilation, Arch. of Derm., 1881, vii, p. 1496.

[2] Der prakt. Arzt., February, 1881.

In general, it may be remarked that patients long affected with rebellious favus may need a roborant course of treatment and nutritious diet. Cleanliness here, as in all the parasitic disorders, is important.

Prognosis.—The prognosis is generally favorable to the ultimate termination of the disease in all cases; for even the most rebellious and untreated forms are relieved when the hair-follicles atrophy. Upon the non-hairy portions of the body the disorder is rarely severe if promptly and efficiently treated. Upon the scalp the prognosis is determined by the extent, severity, and duration of the disease. Early and vigorous treatment of the scalp in healthy children is usually followed by satisfactory results. In long-neglected subjects of the disorder the result may be remediless, with characteristic baldness, the affected surface being provided with scanty wisps of stunted and uncolored hairs. Neglect, filth, and systemic malnutrition are the most unfavorable elements in any case.

TRICHOPHYTOSIS.

Synonym.—Ringworm.

Definition.—Ringworm is a disease of the hair and hair-follicles of the scalp and beard, and also of the nails and the non-hairy portions of the body, and is produced by a vegetable fungus. Its parasitic nature was discovered by Gruby,[1] in 1842-1844. In 1891, Furthmann and Neebe[2] advanced the idea that there were two or more fungi responsible for the various manifestations of the disorder. In the following year Sabouraud[3] began his researches which have thrown so much light on the subject. As his work has been and is being confirmed in various parts of the world, his classification of the fungi will be adhered to in this text. As the several regions of the body, when invaded by the various parasites, display lesions which are more or less peculiar to themselves, it is usual to consider these separately. Ringworm of the body is therefore designated Trichophytosis corporis; of the scalp, Trichophytosis capitis; of the beard, Trichophytosis barbæ. For purposes of description, the symptoms and diagnosis are given in connection with each variety separately. The etiology, pathology, and treatment are considered as a whole.

Trichophytosis Corporis.—**Synonyms**: Tinea circinata, Herpes tonsurans, Ringworm of the body. Ger., *Scherende Flechte;* Fr., *Herpês circiné tricophytique.*

Symptoms.—Ringworm of the body displays different symptoms according to the temperature in which the vegetation flourishes and the various external irritants to which the skin where it has once been implanted is subjected.

In general, there are two forms, the dry and the moist. The dry or macular form of the disease is characterized by the occurrence of one or of several pea- to large-coin-sized, circumscribed, reddish circles, usually paling under pressure, often at the general level of the integument, occasionally slightly raised above it, forming then a flattened disk. The center of the circle may be paler, or, indeed, to the naked eye be unaffected, transforming the patch into an annular lesion, from which circumstance it originally received the name "ringworm." It develops within certain limits, rarely exceeding five or six inches in diameter, by peripheral extension; and is usually characterized at the outer border by slight, whitish, furfuraceous desquamation. This form of lesion is usually seen upon the exposed surfaces of the body, where there are less heat, moisture, and friction than upon other parts, as, for example, the forehead and neck, in moderate atmospheric temperatures. The disease may recur within the peripheral border, and in this way occasionally two, three, or more concentric rings or parallel bands of crescentic outline may be visible in a single patch of disease. Frequently, a tendency to a peculiar formation, often that of concentric circles, is found in every patch existing at the same time in a given case.

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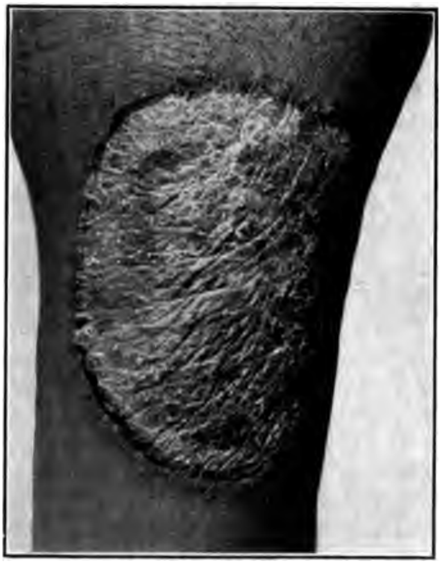
[1] Compt. rend. Acad. des Sci., Paris, 1842, xv, p. 512; ibid., 1843, xvii, p. 301; ibid., 1844, xviii, p. 583.

[2] Monatshefte, 1891, xviii, p. 47.

[3] Annales, 1892, iii, s. iii, p. 1061.

Fig. 347



Trichophytosis corporis. Note vesicles at margin.

The subjective sensations are a trifling degree of itching or burning.

The moist forms of the disease are represented by vesicular and pustular lesions. The vesicular lesions of ringworm appear as such at the onset, or they may rise from macular lesions. In the former case, pinpoint-sized, transitory, and superficial vesicles or vesicopapules spring from a central point or focus and spread peripherally.

The early lesions may speedily shrivel until they are represented merely by minute, whitish, branny scales. These are succeeded by others, always at the periphery, and these again by yet others, the rosy or the reddened base on which they rest being sometimes slightly in advance toward the outlying skin. The enlarging circlets of disease proceed in their course in an evolution similar to that observed in the macular forms. The difference, due to a more virulent fungus, is noted not merely in the type of the lesion, but also in the slightly exaggerated itching sensations that are awakened. Rarely, there may be acute symptoms and extensive development in multiple patches spreading over the face, neck, trunk, and extremities, accompanied by a slight febrile movement and moderate tumefaction of the affected surfaces. As a rule, the eruption is trifling, and may, indeed, be limited to a single ring, or to a few circlets about the neck, terminating in the branny desquamation described; but in the severe forms the evolution of the disease may persist for months, and crusts form, the fall of which leaves annular pigmentation of temporary duration.

The papular and rare pustular forms of the disease observe the same peculiarities with respect to the clearing of the center, the annular appearance of the advancing area of involvement, and the production finally of scales and crusts. They represent, however, a more actively developing fungus, to which may be added irritation of the affected part by friction and heat. These lesions are most commonly observed upon the back, the abdomen, the intermammary and inframammary regions, and the inner faces of the thighs and arms, in which localities they occasionally occur with chronic manifestations. The papules are light- or dull-reddish, pinhead-sized and larger, solid elevations, roundish, oval, irregular in outline, or confluent, forming individual bean- to coin-sized, raised disks, with a pale, exfoliating, or actively inflamed center—the so-called *nummular erythema*, or *discoid trichophytic erythema* of French authors.

Ringworm not infrequently occurs on the dorsal surfaces of the hands, and in this situation is commonly of a suppurating type, similar to kerion in the scalp; and, according to Sabouraud, these lesions are produced by *Trichophyton microïdes* of the types *gypseum* and *nibeum*.

Under the title, "Conglomerate or Agminate Folliculitis," Leloir[1] described a disorder, now known to be a variety of ringworm, which occurred in plaques, coin-sized or larger, elevated a quarter of an inch or more above the cutaneous surface, presenting a mammillated appearance, and perforated by numerous follicular openings, from which pus or dried plugs could be expressed. The openings of some of the follicles were covered by unruptured pustules. Later the patch became phlegmonous, fluctuation developed, the follicles became more patulous, and pus in large quantity could be expressed. The appearance at this time strongly suggested that of kerion of the scalp or of a flat carbuncle.[1] Still another rare form is that first described in 1883 by Majocchi as *Granuloma trichophyticum*. The lesions may occur upon the scalp, the bearded region, or upon the glabrous skin. The parasite invades the structures to a greater depth, making its way into the corium, and producing indolent nodules and plaques. The nodules are hard, and frequently discharge pus, blood, and a yellowish-red fluid. The reaction of the skin to the invasion of the parasite produces a granuloma. In a case recorded by Sequeira[2] the disease was of long duration. There were nodules, ulcers, and infiltrated plaques situated in the right axilla, about the umbilicus, and in the left groin. In the latter situation there were numerous flat, button-like nodules, of a purplish-brown color, with a smooth surface. The fungus as grown from scrapings of the epidermis was identified by Sabouraud as *Trichophyton plicatile*. The disease is rare; most of the cases have been observed in the Italian clinics. The above case is the only example recorded in England.

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[1] Annales, 1904, s. ii, v, p. 437 (with plates); and Quinquaud and Pallier: "Des périfolliculites suppurées agminées en placards," Thèse de Paris, 1889.

Hartzell[3] described an unusual clinical type presenting large patches composed of dark red, mostly discrete, hemp-seed to split-pea sized papules and nodules together with pigmented areas.

Ringworm of the glabrous skin is usually produced by one of the trichophytons, but in rare instances may be induced by a microsporon, chiefly that designated as *Microsporon lanosum*.

Eczema marginatum, Tinea trichophytina cruris.—Partly because of the controversy which the subject aroused, special attention was once directed to this variant of the disease, which Hebra was first to describe. It is most marked upon those portions of the body which come in contact with the saddle when a rider is mounted upon a horse; that is, the perineum and the inner faces of the thighs, the region marked by the reinforcing patch in the trousers of the cavalryman. The disease, as encountered here, occurs in both sexes. It is characterized by extensive exudation in bright or lurid patches, with a very distinctly defined, raised border, showing a sharp contrast with the healthy skin beyond, from which peculiarity it has its name. It may extend laterally over the groins, upward over the pubes, and backward over the sacrum, being generally defined at the periphery by a crescentic outline. The center may be paler and less involved, or actively irritated, while the periphery still extends in one or more annular festoons down the inner side of the thigh or upward over the regions indicated. It is aggravated by heat, the moisture of sweat, and the friction of apposed surfaces of the skin in contact with each other and the clothing. It is usually of symmetrical distribution, due to the circumstances of its development, and in this respect differs from the other manifestations of the disease. The condition may occur in milder or even severe forms in the axilla, about the breasts of women, or about the umbilicus. The course of the disease is obstinate, persistent, and subject in a remarkable degree to relapse in the same locality. The disorder is due to a special fungus termed *Epidermophyton inguinale* (Sabouraud), an organism similar to, but not identical with, the trichophyta.

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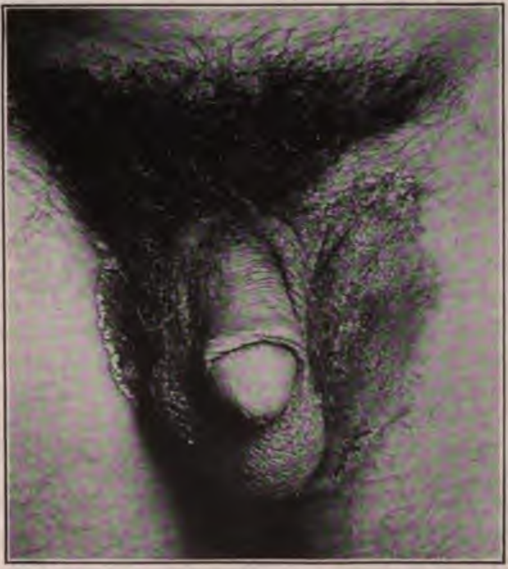
[1] Hartzell, Jour. Cut. Dis., 1895, xiii, p. 456; and Schamberg, ibid., 1902, xx, p. 410 (a review of published cases).

[2] Brit. Jour. Derm., 1912, xxiv, p. 207.

[3] Jour. Cut. Dis., 1920, n. s. i, p. 1. Two unusual cases of Ringworm, one of them due to a Fungus (Trichophyton rosaceum) Producing Pink Cultures.

Of much interest in this connection is a condition described as "*eczematoid ringworm of the extremities,*" by Djelaleddin Mouktar, Whitfield, and Sabouraud.[1] The disease was first described, in 1892, by the first-mentioned author, in 1908 by Whitfield, and in 1910 Sabouraud demonstrated the organism to be identical with that which produces eczema marginatum. The lesions in this variety occur, as a rule, on the feet, occasionally on the hands. The symptoms, as outlined by Whitfield, are as follows: The clinical types are divided into three groups: (1) the acute vesico-pustular; (2) the chronic intertriginous, attacking the toes; and (3) the hyperkeratotic of the palms and soles. The first variety is acute. The disorder comes on suddenly, in from 24 to 48 hours, and has all the characteristics of acute vesicular eczema or dysidrosis. In the second type (secondary to a more or less acute attack) there occurs a white, sodden mass of epithelium between the toes, with a more or less well-defined margin and slight vesiculation at the dorsal edge of the entire phalangeal skin. On the plantar

FIG. 348



Eczema marginatum of the thighs.

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[1] Brit. Jour. Derm., 1911, xxiii, p. 375.

surface the eruption spreads downward beyond the roots of the toes, to terminate in a well-defined, but somewhat irregular, line about opposite the head of the metatarsals. At this free edge the horny layer is in a constant state of desquamation, and the free edge of the scale is, as is always the case in ringworm, definitely turned toward the center of the eruption. The hands may be similarly affected. Intense itching is present, and occasionally soreness, from the patient rubbing the area. The third, or hyperkeratotic type, attacks the whole of the soles and occasionally the palms. The salient feature is the enormous and irregular massing of the overgrown horny layer. There are here and there small, indolent pustules, some of which only show staphylococci, while others bear the fungus in the roof. The dry type on the palms and soles is usually caused, according to Sabouraud, by *Trichophyton violaceum* and *Trichophyton acuminatum*. The duration of the disorder is variable, but in most of the intertriginous and hyperkeratotic cases seen by Whitfield the disease had been present for a number of years, the longest period being over twenty years. Sabouraud[1] believes the disease to be common, and states that in the major portion of the so-called cases of intertrigo of the toes the disease is caused by *Epidermophyton inguinale.*[2]

In a clinical study of 65 patients, in all of which the diagnosis was confirmed by the microscopic findings, the author with Dr. Mitchell[3] corroborated the above findings. In one of these cases a symmetrical keratoderma of the hands and feet had been present thirty-two years. In addition the author has seen the disorder occur in variously sized, well-defined scaling patches over the trunk and limbs coincident with the lesions on the hands and feet. In these the causative microorganism was demonstrated microscopically.

Dhobie itch of the tropics is eczema marginatum of other countries, and is due to *Epidermophyton inguinale.* Castellani[4] has isolated three different species of epidermophytons and one trichophyton that give rise to the eruption. In addition to the ones above mentioned, he describes *Epidermophyton Perneti, Epidermophyton rubrum,* and *Trichophyton nodoformans.*

The eruption in the tropics is exhibited in greater activity, and frequently involves the axillary regions as well as the groins. Owing to the increased local distress, a dermatitis is added through scratching and friction, which frequently induces secondary infections, such as boils and abscesses. Manson[5] believes many cases are due to various varieties of *Microsporon furfur* as well as to *Microsporon minutissimum;* and it is further thought that the organisms responsible for impetigo contagiosa induce some of the cases classed under this

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[1] Loc. cit.

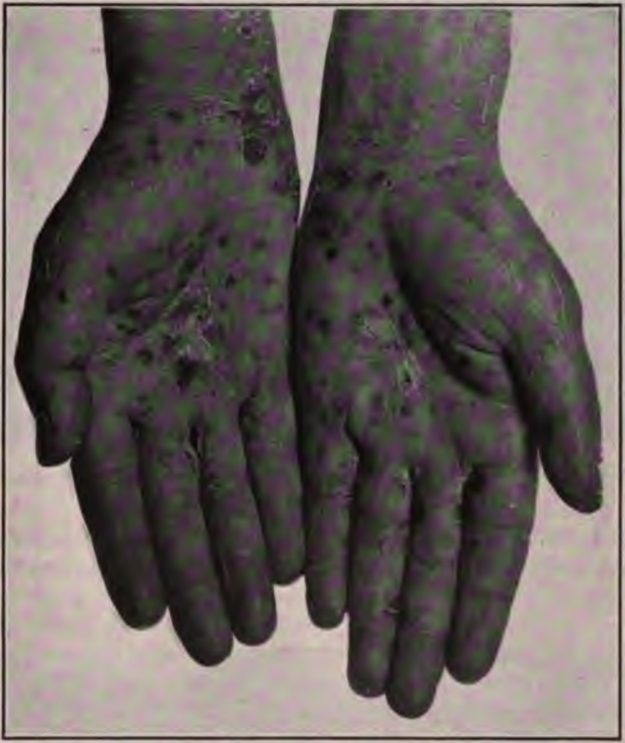
[2] Nicolau, Annales, 1913, iv, p. 65: A contribution to the study of eczema marginatum of Hebra, reporting among others six cases of interdigital infection by the Epidermophyton, in two of which no other sites were involved. Montgomery and Culver, Jour. Amer. Med. Assoc., April 4, 1914, lxii, p. 1076: Report of a case, with description of the parasite and the therapy employed.

[3] Jour. Amer. Med. Assoc., 1916, lxxii, p. 711. Ringworm of the Hands and Feet. *Cf.* this article for more complete description of the evolution of the lesions in the various types and cultured findings in 17 cases. Also see White, Jour. Cut. Dis., 1919, xxxvii, p. 501. The Question of Epidermophyton Infection.

[4] Castellani and Chalmers, Manual of Tropical Med., 2d ed., 1913, p. 1474.

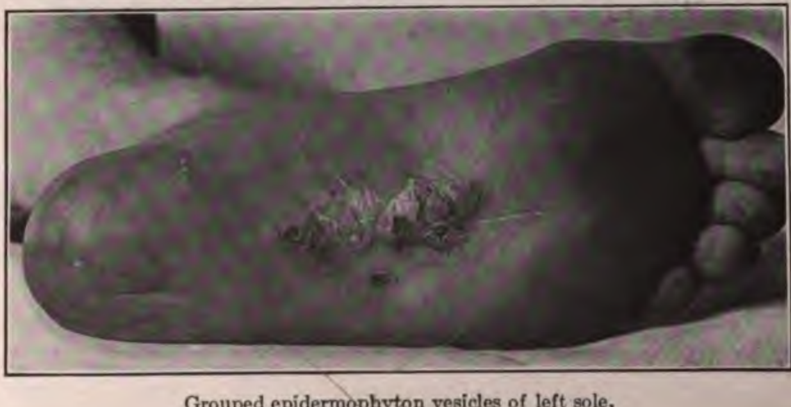
[5] Tropical Diseases, 4th ed., 1910, p. 770.

FIG. 349



Vesico-pustular epidermophyton lesions of hands and feet.

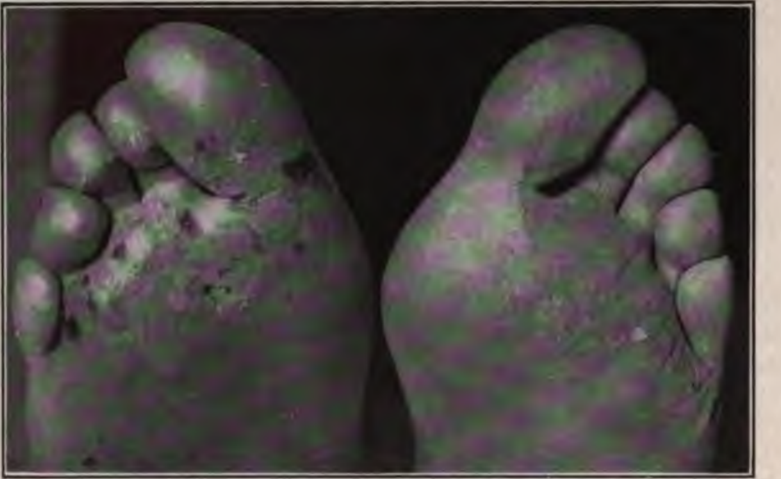
FIG. 350



Grouped epidermophyton vesicles of left sole.

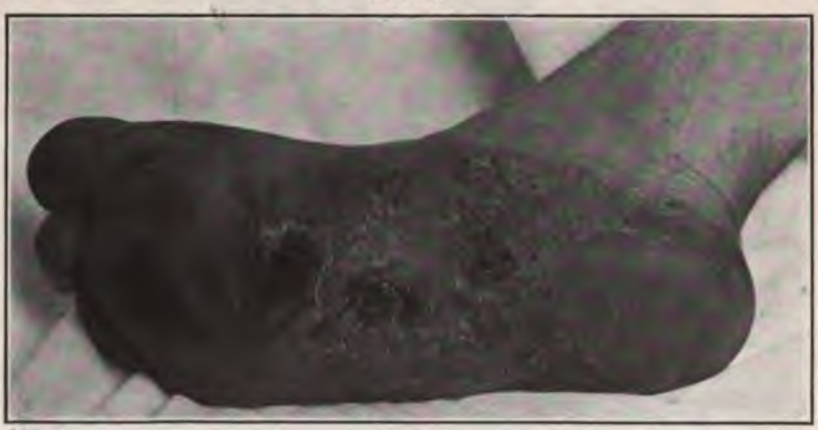
heading. The spread of the disease is attributed to the laundry, giving rise to the name "dhobie (washerman's) itch." Stitt[1] called attention to fulminating cases of dhobie itch due to a symbiosis of an infecting mould and coccus, such cases being much more virulent than those in which only the mould occurs. In the tropics the term is loosely used to denote practically all itching skin-affections.

FIG. 351



Chronic intertriginous type of epidermophytosis with extension to the plantar surface.

FIG. 352



Chronic keratotic epidermophyton lesions of plantar surface.

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[1] Jour. Cut. Dis., 1908, xxvi, p. 107.

Under the title of *Lichenoid trichophytosis*[1] (Jadassohn), a number of cases of ringworm infection of the glabrous skin have been reported, which presented symptoms differing from the types above described. The eruption occurred usually in association with kerion, and the lesions were small papules, generally follicular, bright-, bluish- or pale-red in color, and situated chiefly on the trunk and limbs below the knees. Guth described two varieties: one characterized by papules having horny spines, the other by patches resembling psoriasis or seborrhoic dermatitis. The disorder occurred only in children, and the fungus found was *Trichophyton gypseum*.

In addition to lichenoid, vesicular and scarlatiniform, eruptions occurring in association with kerion were observed by Rasch.[2]

Diagnosis.—Ringworm of the body is to be distinguished, clinically, from eczema, psoriasis, dermatitis seborrhoica, lupus erythematosus, herpes iris, and syphilis. All the varieties of eczema are noted for their greater degree of itching and infiltration, their much more poorly defined border, coarser scales, decided absence of a circular contour and of a history of contagion. Psoriasis does occur in circular and annular patches, often with a clear center and with insignificant subjective sensations; but its scales are lustrous and the tissue beneath them readily bleeds, showing deeper implication of the skin. The disease is often symmetrical in disposition; occurs by preference upon certain regions of the body where ringworm is relatively infrequent; and its history is that of a chronic disorder. Dermatitis seborrheica never presents vesicles nor pustules, and its redness is apt to be less acute, the scales and crusts more greasy and abundant. The subjective sensations are also likely to be less marked, and the disease is apt to be limited to special areas, such as over the sternum or between the shoulders. Lupus erythematosus is often symmetrical, generally chronic, and is characterized by the development of multiple annular patches, enlarging centrifugally from a clearing center and leaving more or less atrophy. Herpes iris can be distinguished, first, by its predilection for the extremities; second, by the color variegations which it displays and which are never seen in ringworm of the hands. Syphilis is multiform in its lesions, usually preceded by a history of infection; and its distinctly circular patches, enlarging at the periphery, exhibit either atrophy, or ulcerative or distinctly crusted lesions, which suffice for diagnostic purposes. Pityriasis rosea is not characterized by vesicles; is often symmetrical in development; occurs in oval rather than in distinctly circular patches; and exhibits a characteristic tawny-yellowish color not seen in ringworm.

Since the discovery that the disease may occur on the hands and feet as above described, much difficulty in diagnosis is encountered. It can only be suggested that in all cases of intertrigo that are of any duration and do not yield to the ordinary treatment for that disorder, a search should be made for the fungus. A history of a member of the family having suffered with eczema marginatum would be a valuable aid in diagnosis. In a number of the reported cases such a history was obtained.

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[1] Guth, Archiv, 1914, cxviii, p. 856 (a report of 15 cases); Herxheimer and Köster, Zeitschrift, 1914, xxi, p. 570 (case report). Rasch, Brit. Jour. Derm., 1916, xxviii, p. 9; Secondary Lichenoid Trichophytides in Association with Kerion Celsi (Lichen Spinulosus Trichophyticus). Translated by Oliver.

[2] Brit. Jour. Derm. and Syph., 1920, xxxii, p. 317; Clinical Remarks on Trichophytia Profunda (Kerion Celsi).

Finally, the microscopical discovery of the parasite is the chief, and, indeed, the essential method of diagnosis in trichophytosis corporis. Whitfield lays stress on a particular technique in examining the vesicular type of ringworm of the feet: The fungus, being ordinarily situated in the roof of the vesicle, is best seen by removing the structure and placing it upon a slide with the deep surface upward. A 10 per cent. solution of potassium hydroxid is then placed on the specimen and after five minutes a cover-slip is applied and the specimen examined with a one-sixth or one-seventh objective.

Trichophytosis Capitis.—Synonyms: Tinea tonsurans, Ringworm of the scalp, Herpes tonsurans, Tinea tondens. Ger., *Scherende Flechte; Teigne tondante.*

Ringworm of the scalp is a disease chiefly of childhood, and occurs most frequently among those congregated in public institutions. The gregarious habits of children, and the frequency and intimate character of contacts in their amusements and studies, greatly increase the chances of contagion when one of their number is affected with ringworm of the scalp. In consequence, the early recognition and relief of the disease furnish problems among the most important presented to the general practitioner as well as to the dermatologist.

Symptoms.—The symptoms usually first observed are circumscribed, small-coin-sized, roundish patches upon the scalp, wholly or partially covered with minute, whitish, slate-colored, grayish, or dirty-yellowish scales. Sometimes the formation of the scales can be observed as they develop upon a hyperemic and reddened area. Still more rarely, pinpoint-sized, transitory vesicles or pustules precede. The hairs upon such a patch seem irregularly clipped short near the surface, or, as it is frequently styled, "nibbled" off, thus producing the effect of partial baldness in the involved area. Among them may often be found lusterless, dry, long, and fragile hairs, which break upon slight traction or flexion. The patches may increase in number and spread individually in area until, in the course of weeks or months, the entire scalp is invaded. In the older patches young and downy hairs may here and there be seen pushing up the stumps left by those that have fallen. One or more of the various phases of the disease may be presented in its subsequent evolution. Thus, a single patch may extend to the size of a large coin or of the palm, and the disease be throughout limited to that area. Again, almost the entire scalp may be covered with relatively small or enlarging patches, or, even without the occurrence of any distinct patch, isolated hairs or tufts of hairs here and there over the entire scalp may exhibit evidence of impairment. The disease may be acute or be chronic in its course. Instead of assuming the dry and squamous type described, acute and exudative symptoms may develop, in which event the rare vesicular lesions are succeeded by the exudation of a gummy secretion and the formation of crusts. The condition known as "kerion" is described below.

Itching in various grades of severity, though usually mild, is induced by the disease; and often the patches are altered in appearance by the traumatisms produced by the finger-nails and the comb. When the scalp is very generally invaded by the squamous form of the disorder, its appearance is similar to that noted in diffuse seborrhea, chronic eczema, and psoriasis of the scalp, except that the hairs are less pasted to the surface; are more lusterless, friable, and contorted in shape; and much more often are represented by stubble or stumps.

Stowers,[1] Sangster,[2] and also Hutchinson, Hillier, Baker, MacLeod and Fraser,[3] Little,[4] and others have recorded cases in which the disease coexisted with alopecia areata. Géber asserts that after exfoliation of patches of ringworm the scalp may, in cases, become absolutely bald, smooth, and glossy. This condition may exist from the beginning in the *Bald ringworm* of Liveing, which is often mistaken for alopecia areata, an error readily corrected by the recognition of scaling patches, with hairs exhibiting under the microscope the presence of the fungus.

The *Disseminated ringworm* of Alder Smith affects isolated hairs or small groups of hairs scattered over the scalp, a broken stump, or a group, or a relatively small number of lusterless, dry, and friable hairs furnishing the only evidence of the disease. Fox[5] states that the prevailing pattern on the scalp is the formation of slightly scaling macules, on which diseased stumps may be found disseminated among some healthy hairs which have escaped infection. The diseased stumps on these macules may be single or in groups of two or three to a dozen or more. In children with long hair the disease often escapes notice. This variety is produced by *T. endothrix*. Occasionally, patches are formed which resemble those produced by the microsporon. The diseased hairs are described as being more or less pigmented. A striking feature of the diseased stumps is that the extrafollicular portion is so disorganized that it turns upon itself and becomes like a compressed corkscrew beneath the obstructing scales; and when the stump is pigmented the appearance of a black dot is presented. Most of the cases of the disseminated variety are due to *Megalosporon endothrix* (*Trichophyton crateriforme*). A few, however, are due to microspora.

Ringworm produced by *Microsporon Audouini* can often be distinguished clinically from that produced by the trichophyton. In the former the patches are single or few in number, are rounded or oval in outline, may be of considerable size, are usually slightly reddened and furfuraceous, and are more or less covered with hairs, which are lusterless, dirty-looking, broken off at irregular distances from the surface, and easily epilated between the thumb and finger in considerable numbers. In this form a grayish or whitish sheath (composed of spores) is seen encircling each hair and extending from 1 to 3 mm. above its exit from the follicle. In the case of ringworm produced by *T. endothrix*, the patches are much more numerous, but are very small and irregular in outline, and instead of being covered by hairs and broken stumps of hairs usually show a number of black dots at the mouths of the follicles, caused by the curling up of the hair beneath the scales; and instead of forming patches the disease may affect isolated hairs or small groups of hairs.

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[1] Lancet, 1881, i, p. 326.

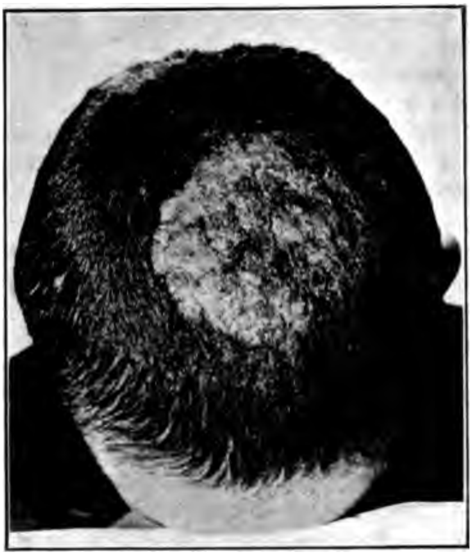
[2] Ibid., 1880, i, p. 425.

[3] Brit. Jour. Derm., 1909, xxi, p. 258.

[4] Ibid., 1913, xxv, p. 137.

[5] Ibid., 1909, xxi, p. 271.

FIG. 353



Kerion of the scalp in a boy aged ten years.

Tinea Kerion (*Kerion Celsi*).—The occurrence of active and usually circumscribed inflammation in a portion of the scalp affected with ringworm is at times followed by certain peculiar features. This complication of ringworm was recognized early in the history of medicine by Celsus, whose name has since been associated with its lesions. Tilbury Fox, in 1866, was first to recognize its identity with trichophytosis capitis; and it has since been the subject of a number of papers by Tanturri, Majocchi, Schilling, Barduzzi, Auspitz, and Wilson. In the United States, Atkinson[1] has made it the subject of a memoir.

The symptoms are the occurrence of acute inflammation, usually circumscribed, though occasionally diffuse,[2] in a portion of the scalp, where a tumor forms, which may project to a considerable height above the level. In time the appearance presented is suggestive of a carbuncle, since from the tumid orifices of numerous distended follicles a viscid, semitransparent, puriform fluid exudes. The latter is characteristic. The hairs loosen and fall. When the view of the lesion is not obscured by the pilary growth, it appears as a flattened, hen's-egg- to turkey's-egg-sized, boggy, semiglobular tumor, its surface congested, reddened, glazed, and often exhibiting other evidences of inflammation, with split pea-sized, pustule-like lesions distributed over its surface, or, when these have ruptured, exhibiting the gaping apertures described above, from which a gummy secretion is poured in varying quantities. Modification of this condition occurs, such as the production of a true subcutaneous abscess with fistulous sinuses. The sensations awakened are usually painful. The course of the disease is chronic. It may begin with the usual symptoms of ringworm of the head, though often there is no history of the latter. The parasite may or may not be found in patches of kerion, according to the acuity of the present or the precedent inflammatory process. If the latter has been of high grade, and suppuration has resulted, the fungus is destroyed, a result the attainment of which has been attempted in the production of "artificial kerion" by means of croton-oil for the relief of trichophytosis capitis. In the earlier stages, represented by deep-seated follicular inflammation, with pustulation of the hair-shafts, the latter may be seen microscopically to be invaded with the fungus.

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[1] Archiv. of Derm., 1881, vii, p. 47.

[2] Wallis, Jour. Cut. Dis., 1905, xxiii, pp. 428, 431. Ten cases with multiple small kerion are described occurring in girls from eight to seventeen years of age.

Diagnosis.—The recognition of a typical patch of ringworm of the scalp is simple. The branny scales, clumps of hairs, and distinct contour of the invaded area are always in the highest degree suggestive symptoms. It has been stated, however, that the general development of ringworm over the scalp produces a condition very like that seen in other diseases. In this case the microscope must be employed for a decision as to the nature of the process. In pityriasis there is a symmetry of involvement which even aggravated cases of ringworm of the head fail to assume; and even though pasted down, atrophied, changed in color, and loosened in their follicles, the hairs are rarely broken off near the scalp in pityriasis of the scalp. In pityriasis, psoriasis, and squamous eczema of the scalp there is, moreover, no history of contagion, and the scales in each disease are different in color and character. The hairs in the two affections last-named are firmly fixed in their follicles, and only in severe cases present nutritional changes. These diseases, moreover, are usually chronic in their course. In any doubtful case, apart from microscopical evidence, thorough removal of all scales from the scalp by shampooing with green soap and hot water will reveal the nature of the disease present.

Alopecia areata, as above noted, may coexist with ringworm, but it is pathologically distinct from it. The patches in the first-named disease are uniformly smooth, and the hair falls from them *en masse*, without scaling or other traces of previous involvement of the regions affected. Blackish points or dots, due to the accidental lodgment of particles of dust, are occasionally seen in alopecia areata, but should cause no confusion, as the other evidences of the so-called "black-dot" ringworm are usually absent.

In favus the cup-shaped crust will sooner or later betray the character of the disease to the naked eye. Confirmatory evidence as to the nature of the disease will often be furnished by a careful search for the source from which it was derived, and for obvious reasons this should always be attempted.

Complement-fixation tests have been employed by Kolmer and Strickler[1] and others with varying results.

Technique for the Microscopic Examination of Hairs.—To prepare a hair for examination, it may be placed between a slide and cover-glass in a solution of potassium hydroxid. Sabouraud uses a 25 to 40 per cent. solution, which is admirable for rapid work, but which quickly disintegrates the hair. Adamson employs a 5 or 10 per cent. solution, which clears the hair slowly in the course of one or several hours. By making frequent examinations of the specimen, the observer can arrest the destructive action of the solution at any stage desired, and thus better preserve the relative position of the fungus to the hair. Many attempts have been made to stain the fungi, which unfortunately show an affinity for the same stains as does the cortical layer of the hair. A satisfactory method has been devised by Morris and his laboratory assistant, Calhoun. It is a modification of the Gram and Weigert stain for bacilli, and gives good results. The hair is first washed with ether, to remove fatty débris; it is then put for one or two minutes in the Gram-iodin solution, and after drying is stained for from one to five minutes in gentian violet and anilin-water. It is again dried and treated for a minute or two with the iodin solution, and for an equal length of time in anilin oil containing pure iodin, after which it is cleared with anilin oil, washed in xylol, and mounted in Canada balsam. Coarse, dark hairs and spores within the hairs require more time for staining than do fine, light-colored hairs and the fungus elements situated without the hairs.

While microscopical examination will often suffice to distinguish the microsporon from the trichophyton, or even for recognition of some of the varieties of the latter, the finer, and often disputed, points of difference can be appreciated only by means of culture-experiments, for details of which see Pathology and Bacteriology.

Trichophytosis Barbæ.—Synonyms: Tinea sycosis, Hyphogenous sycosis, Tinea barbæ, Sycosis parasitica, Mentagra parasitica, Ringworm of the beard, "Barbers' itch." Ger., *Parasitäre Bartfinne;* Fr., *Trichophytie sycosique.*

Symptoms.—The disease is best studied at its onset in the beard of a blond subject with relatively fine, downy hairs, where are presented the typical features of trichophytosis corporis, or ringworm of the body. One or several, reddish, pea- to small-coin-sized rings become visible, with pinpoint-sized vesicles, branny scales, and often, indeed, no other lesion save a hyperemic, scarcely elevated margin. The hairs over the patch may be fragile, and clusters of them here and there betray evidences of change. With proper treatment the disorder may not progress beyond this point.

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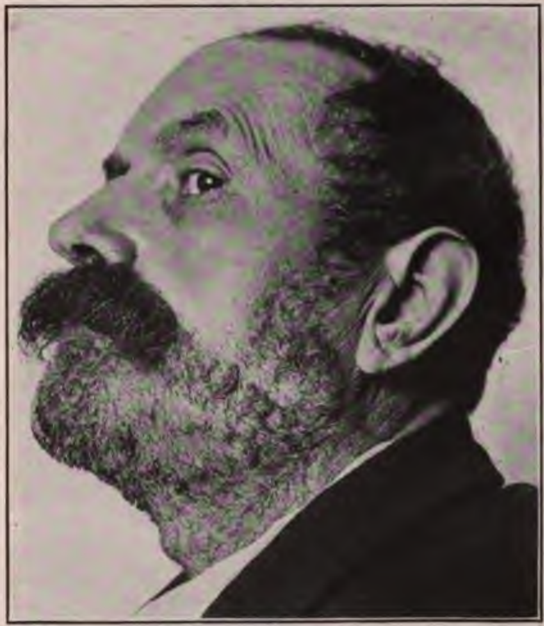
[1] Jour. Amer. Med. Assoc., March 6, 1915. Complement-fixation Tests in Ringworm and Favus.

In some cases the very slight degree of itching awakened by the process just described may be intensified, and large plaques form, a portion of which may extend from the region of the beard over the face and neck, or *vice versâ*. When fully developed, a phlegmonous disorder is produced, which bears some analogy to the kerion just described, and which may so actively progress that it is first seen in typical development. The skin is congested and reddened, has subepidermic pustules (or débris of those already ruptured) at the orifice of the hair-follicles, and is studded irregularly with firm, pea- to nut-sized papules and tubercles. The tubercles are usually aggregated in masses or lumps, which involve the skin and subcutaneous tissue, and they are firm, often tender and painful, rarely boggy and furuncular. When pierced, they give exit to a characteristic muciform, gluey, yellowish, and sticky fluid, puriform yet differing from pure pus, that rapidly dries into crusts. These composite lesions are usually circumscribed in a given area of involvement, very rarely covering the region of the

FIG. 354

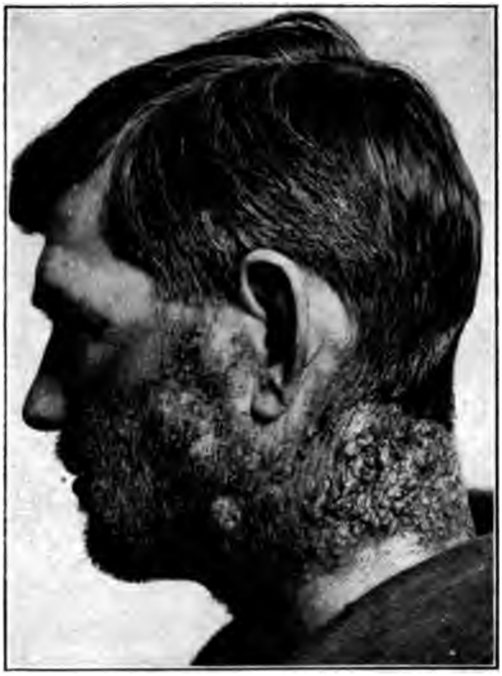


Trichophytosis barbæ.

beard in symmetrical disposition, more often limited to one cheek or to the cheek and chin.

The hairs in the invaded region are involved as in ringworm of the scalp. These filaments break near the surface of the integument, leaving ragged stumps; or they spontaneously fall after being loosened in their follicles. The ease with which they may be epilated is one of the most characteristic features of the disease; they are slipped out of their follicles as readily as if they had been oiled; or, as Anderson writes, "as easily as a pin can be pulled out of a pin-cushion." They are then often whitish, because enveloped in the fungus producing the disease. In either event, the resulting gradual thinning or removal of the hairs renders the disease of the surface more conspicuous and deforming. At the edge of a patch thus exposed, deformed, lusterless, contorted, flattened, twisted, or split hairs may be found. Occasionally, the features of the patch are changed in consequence of the unusual degree of suppuration excited. In this case the pustules burst and their contents concrete into dry crusts about the stumps of shafts of surviving hairs, from which circumstance the disease has received its name. Rarely, a conglomerate crust covers the entire region, with an excoriated, inflamed, and secreting surface beneath.

FIG. 355

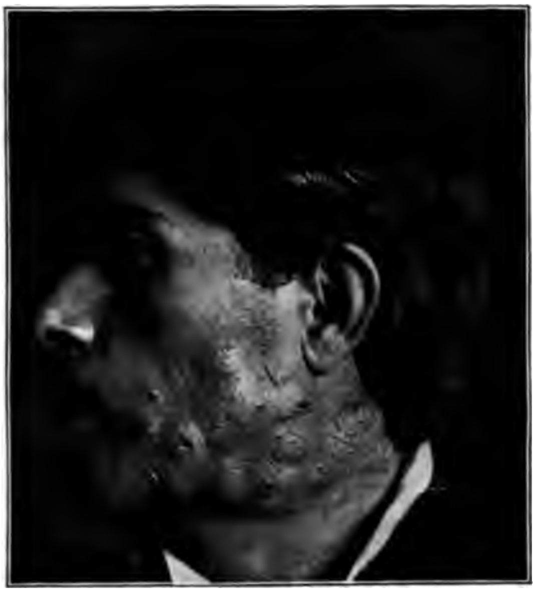


Trichophytosis barbæ. (Kerion.)

Formidable cases of trichophytosis barbæ have occurred in the persons of farmers, where the disease was long untreated and unrecognized. Some severe types of the disease have been produced after shearing sheep having diseased pelts. In these cases the cheeks, lips and chin are the seat of nut- to fist-sized and larger cutaneous and subcutaneous, soft, boggy, and pus-filled tumors, accompanied by excessive soreness of the entire throat and neck, and the hairs fall from the follicles in large masses, and as if lubricated to facilitate their escape.

The major portion of cases of ringworm of the beard observed in this country are of the suppurative type and due to the ectothrix fungi, which are of animal origin. In London the endothrix fungi, which are of human origin, appear to be responsible most frequently; and, according to Fox, it is the endothrix of the type *violaceum*. Clinically, the lesions have the general characteristics of the other *Endothrix trichophyta*. Small macules with black dots are present. Adamson[1] saw twelve endothrix ringworms of the beard in one year. The symptoms in his cases were described as scaling rings, leading to black dots and curled-up stumps, often becoming inflammatory and producing peculiar inflammatory nodules, but no pustules.

Fig. 356



Trichophytosis barbæ.

Diagnosis.—Ringworm of the bearded region is to be differentiated chiefly from coccogenous sycosis; and, necessarily, the microscope must be employed to settle the question definitely. The diseases, however, differ in their clinical features. The coccogenous form always fails to exhibit the nodules, tubercles, and composite cutaneous and subcutaneous agglutinations of the disease produced by the fungus. The process in the former is more superficial, and it exhibits a more vivid redness as a result of the cutaneous hyperemia. Owing to the same cause, the frequent pus-containing lesions are developed and elevated above the general level of the integument; they are less commonly subepidermic crypts filled with characteristic mucoid, puriform contents. The region of the bearded upper lip, so often involved in cases of nasal catarrh, is often spared by the trichophyton. When this parasite is present, the hairs are characteristically loosened, distorted, and otherwise changed. This condition is not seen in the coccogenous disease; exception, however, in this particular is to be noted in some long-standing cases of the latter. When the affection has persisted for many years (and one may often see patients thus affected), the thinned and starved condition of the pilary growth is a striking symptom, the scanty, lusterless hairs often scarcely sufficing to conceal the deforming redness and pustulation of the surface from which they spring. The diffuse, symmetrical affection of the hairy face, extending over both cheeks and chin, is more frequently connected with the presence of pus-cocci. Lastly, the hyphogenous, as a rule, is less painful and tender than the other form of sycosis, and, furthermore, is, without question, of rarer occurrence.

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[1] Brit. Jour. Derm., 1909, xxi, p. 287.

With respect to syphilis, it is to be noted that the papular or the pustular syphiloderm developed in the region of the beard is, almost without exception, to be discovered also in other parts of the body, especially the scalp. Ringworm of the scalp and the beard existing at the same time in one individual is rare. In syphilis there is usually an offensive odor to the abundant crusts; shallow ulcers are also prone to form beneath the pustules; and there is often a history of infection or a hint of the nature of the disease in its polymorphic character.

Eczema of the bearded region may extend to or from other portions of the face, as in a case in which it sweeps down from the ear. The presence of a stalactitic crust depending from the lobe of the ear of an affected side would at once furnish a clue to the nature of the disease in the beard. In eczema the interfollicular region is invaded, not deeply as in ringworm, but superficially as in coccogenous sycosis; the itching is severe; the hairs are not involved; the infiltration is diffuse; the outline is indeterminate; and a halo of redness spreads from the affected part to the non-hairy surface in the vicinity.

Etiology. — *Trichophytosis corporis* is caused by the various fungi before mentioned. The disease is more contagious than favus, and hence is much more common. Occurring upon the non-hairy portions of the body, it is often spontaneously removed by the desquamative process which it excites in the skin. The development of the fungus is favored or retarded by external influences. Heat and moisture are important factors, and it is therefore much more severe and rebellious in tropical countries. It occasionally occurs in epidemic forms. Gerlier[1] gives the details of such an epidemic in Ferney-Voltaire, France, where twenty-six cases of the disease came under his observation. In some of these instances the lesions were pustular, in others tuberculo-pustular. Aggravated forms of the disease often originate in the lower animals, and a severe and rebellious type is frequently derived from the horse. Trichophytosis corporis occurs more frequently in children than in adults, presumably from the relatively tender condition of the epidermis in these subjects. A small epidemic occurring in several members of four families was recently observed by the author. The origin of the disease in this instance was traced to a litter of kittens which had been distributed among these families. A fungus was isolated from these patients and also from the kittens, and these appeared to be identical. The disease is particularly liable to occur in men whose skins are especially moistened, as in those who work in atmospheres saturated with steam. Several members of a single household will often display ringworm of the body at the same time, having transmitted it the one to the other. The need of an appropriate soil for the germination of the fungus is shown by the fact that some individuals are predisposed to its invasion. It is, however, encountered in both sexes and in all social conditions.

Trichophytosis capitis is produced usually by *Microsporon Audouini,* quite a large per cent., however, being produced by *T. endothrix* (see paragraph on Pathology and Bacteriology). The disease is observed frequently in children of both sexes, especially in those gathered together in schools and public charities, where it may spread very generally from one to another, and require months and years for its extermination. It is a highly contagious disease, but yet requires unquestionably a suitable soil for its development. Ringworm in the scalp of the adult or aged is indeed among the rarest of cutaneous accidents (Hyde). MacLeod[2] demonstrated a patient, aged forty-three years, with ringworm in the scalp, due to a microsporon.[3] Pernet,[4] Rothwell,[5] Oliver,[6] and others describe adult cases of trichophytosis capitis. Among the methods of transmission is the use of contaminated brushes, combs, wearing apparel, sponges, or towels. The disease is one which prevails rather in the cities than in the country, in which respect it differs from favus.

Trichophytosis barbæ is produced by the trichophyta (see paragraph on Pathology and Bacteriology), usually the ectothrix, less often the endothrix. J. C. White, of Boston, has called attention to the frequency of its appearance in the barber-shop, a fact which common experience verifies. It is usually the irregular visitor to these establishments who is first to supply the germs of the disease. No individual proprietorship of cup, soap, brushes, and razor can secure against the danger of infection the person whose razor is drawn over a common strop, whose cheek is handled by unwashed fingers which have recently been passed over an infected face, or whose beard is combed, brushed or rubbed with the implements and towels in common use at these establishments.

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[1] Lyon méd., 1881, xxxvi, p. 599, and xxxii, p. 7.

[2] Brit. Jour. Derm., 1909, xxi, p. 90.

[3] Idem, ibid., 1911, xxiii, p. 84 (a small-spored ringworm in the scalp of a woman aged twenty-three).

[4] Ibid., 1912, xxiv, p. 141 (tinea tonsurans in a woman aged sixty years; fungus, *Megalosporon endothrix*).

[5] Jour. Cut. Dis., 1914, xxxii, p. 712.

[6] Brit. Jour. Derm., 1915, xxvii, p. 119, and ibid., 1917, xxix, p. 34. Adult cases of trichophytosis capitis produced by a microsporon.

It is difficult to determine the frequency of the disease from statistics. The affection is certainly relatively rare, yet more common than is often supposed to be the case. It is of somewhat irregular occurrence, months often passing without a case coming under observation, after which several may be noted in rapid succession.

The disease, being contagious, is one affecting men in all stations of life, and these rather under than over the fortieth year. More men with light hair and eyes, and light-brown, reddish or sandy beards, are affected than those having darker shades of hair and eyes.

Pathology and Bacteriology.—Microscopically, there are two distinct and unrelated forms capable of producing ringworm: *Microsporons*, or small-spored fungi; and *Trichophytons*, or large-spored fungi. The *Trichophytons* are divided into two main varieties: *Megalosporon endothrix*, which grows entirely inside of the hair; and *Megalosporon ectothrix*, which grows chiefly on the surface of the hair-shaft. Occasionally, the latter variety appears to penetrate the hair to a certain extent, in which case it is termed *Megalosporon endo-ectothrix*.

The *Megalosporon endothrix* is further subdivided into two varieties: the resistant and the fragile, these names being given to varieties either unaffected or disintegrated, respectively, by the action of potassium hydrate. *Microsporons* are differentiated from *Megalosporons* more by their arrangement than by the size of the individual spores (Morris). There are certain members of the former family which are larger than certain ones of the latter.

From a cultural standpoint, Sabouraud's classification of the ringworm fungi, as given in his work, "Les Teignes" (Paris, 1910), is as follows:

Microsporons are divided into two groups: first, those of human origin, which include *M. Audouini*, *M. umbonatum*, *M. tardum*, and *M. velveticum*. In Paris, only the first and third of these are important. The second group is of animal origin, and produces in man lesions of the more inflammatory type. Of this group *M. lanosum* (*canis*, Bodin) is the only one of importance in Paris. In addition there belong to this group the *M. felineum*, *M. fulvum*, *M. villosum*, *M. pubiscens*, and *M. tomentosum*.

The *Megalosporons*, or *Trichophytons*, are divided into two main groups: *endothriches* and *ectothriches*.

The *endothriches* are still further subdivided into, first, the pure endothrix type, with the following members: *T. crateriforme*, the most important; *T. acuminatum*, the next in frequency; *T. violaceum*, the third in frequency. The following of this type are rare in Paris: *T. effractum*, *T. fumatum*, *T. umbilicatum*, *T. regulare*, *T. sulphureum*, *T. polygonium*, *T. exsiccatum*, *T. circumvolutum*, *T. pilosum*, and *T. glabrum*.

The second variety of endothriches Sabouraud terms neo-endothrix. In this variety the organism is of slower growth and does not penetrate the hairs with the same rapidity that the pure endothrix types do. The two members of this group are *T. cerebriforme*, of which quite a number of cases occur in Paris; and *T. plicatile*, which is rare in Paris.

The ectothriches occur in two forms: those with small spores and those with large spores. The small-spored variety, termed the microid type, is still further subdivided into the *gypseum* and *niveum* types.

Members of the ectothrix microid, *gypseum* type, are as follows: *T. asteroides*, with a fair representation in Paris; and *T. radiolatum*, *T. lacticolor*, *T. granulosum*, *T. farinulentum*, and *T. versicolor*. All of the latter members of this group are rare.

The ectothrix microid, *niveum* type, is represented by two members: *T. radians* and *T. denticulatum*.

The megalospora ectothriches are divided into two types: first, those with cultures having a velvet-like surface; and, second, those with cultures resembling the cultures of favus.

The ectothriches megaspora, with cultures of velvet-like surface, are represented by *T. rosaceum*, *T. vinosum*, *T. equinum*, and *T. caninum*.

The ectothriches megaspora, with cultures resembling the cultures of favus, are represented by *T. ochraceum*, *T. album*, and *T. discoides*.

The above outline indicates that only a few of the large number of the organisms occur with any frequency in Paris, and there the microsporons of human origin (*M. Audouini* and *M. tardum*) are most common, the former being in the great majority. Among the microsporons of animal origin, *M. lanosum* is the only one of importance in Paris.

Of the megalospora endothriches, *T. crateriforme*, *T. acuminatum*, and *T. violaceum* are of importance.

Of the megalospora endothriches, neo-endothrix type, *T. cerebriforme* is the important one.

Of the megalospora ectothriches microid, *gypseum* type, *T. asteroides* is the most common one.

Of the megalospora ectothriches megaspora, with velvet-like-surface cultures, *T. rosaceum* is the most important.

Of the megalospora ectothriches megaspora, with cultures resembling the cultures of favus, *T. ochraceum* is the most common.

In London, Colcott Fox,[1] in a study of 639 cases of endothrix trichophytosis, made findings similar to the above. *T. crateriforme* was present in 38 per cent., *T. acuminatum* in 26 per cent., *T. violaceum* in 15 per cent. This agrees with Sabouraud's findings. In addition, he found a primrose-colored culture in 21 per cent. There were also a few that could not be identified.

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[1] Brit. Jour. Derm., 1909, xxi, p. 271.

According to Fox, *T. violaceum* is the common cause of beard ringworm in London. This particular organism induces the black-dot, superficial variety. Nicolau[1] found *T. violaceum* in 38 out of 45 cases of ringworm of the scalp.

Bang,[2] in a discussion of ringworm found in Denmark, states that *M. Audouini* is rare, but that there was an epidemic in that country in 1906 which affected fifty children. Of the endothrix trichophyta, he describes nine cases produced by *T. violaceum* in the scalps of children between the ages of three and nine. The trichophyton neoendothrix *plicatile* is the most common in Denmark, according to this author. It produces most of the ringworm of the beard, and occurs in four forms: (1) round, dry, scaling areas; (2) impetiginous lesions; (3) infiltrated, sycosiform, suppurating lesions, often associated with impetiginous ones; and (4) kerion-like plaques. Of 55 cases, 39 affected the beard. Of the ectothrix trichophyta, the common varieties are: *T. gypseum asteroides*, which correspond with Sabouraud's findings; and *T. faviforme discoides*.

The former was found in the scalp, producing kerion lesions and scaly patches. *T. faviforme discoides* was found in cases derived from the horse, and occurred in the scalps of children, once in the beard of a man, and twice in the glabrous skin.

Among the small-spored fungi only two need special mention: *Microsporon Audouini* and *Microsporon lanosum*.

Microsporon Audouini is the more important, and produces the major portion of the cases of ringworm of the scalp. In London it is responsible for from 80 to 97 per cent., according to different observers. Morris[3] found it in 92 per cent. in 126 consecutive cases; Fox and Blaxhall[4] in 80 to 90 per cent. in 400 consecutive cases; and Adamson[5] in 97 per cent. in 178 cases. In Paris, according to Sabouraud, it causes slightly less than 50 per cent. of the cases. In America, in Boston, White[6] found this organism in 88 per cent. of the scalp cases; Corlett[7] in 90 per cent. in Cleveland; Wende[8] in 89 out of 90 cases in Buffalo. In Scotland, Norman Walker[9] found it in 18 out of 20 of Jamieson's cases. A similar preponderance of the microsporon in ringworm of the scalp has been noted by us in Chicago. In a series of 100 cases Beeson[10] found this microörganism in 89 per cent. This variety is found fairly commonly in Paris, seldom in Italy, Spain, Denmark, Sweden, Germany, and Austria. Pasini[11] states that in Italy, until recently, only four sporadic cases of small-spore ringworm had been seen, and one of these was not the common microsporon of London, but that of the dog, and the other three were that of the horse. In 1906, Pasini found three cases of the common English microsporon in Como, and subsequently in an institution in Como, where there were 200 poor children, he found 27 cases. He also found three other children in other parts of the province. In each case he isolated the microsporon of Audouini.

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[1] Annales, November, 1909, p. 609.

[2] Verhand. des ersten Nord derm. Kongress, p. 9.

[3] Ringworm in the Light of Recent Research, London, 1898.

[4] Brit. Jour. Derm., 1896, xviii, pp. 241, 291, 337, 377.

[5] Quoted by Morris.

[6] Jour. Cut. Dis., 1899, xvii, p. 1.

[7] Jour. Amer. Med. Assoc., March. 18, 1899.

[8] Cited by Corlett.

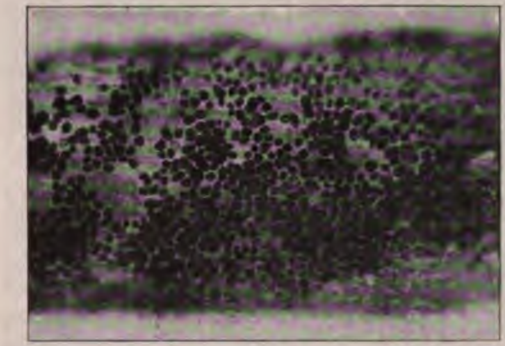
[9] An Introduction to Dermatology, 1899, p. 150.

[10] Jour. Cut. Dis., 1915, xxxiii, p. 781. Ringworm of the Scalp in Chicago, a bacteriological study in 100 cases.

[11] Atti. della Soc. Milanese di Medic. e Biologia, ii.

This organism is of human origin and produces the epidemics. seen in schools and public institutions. In this type the hair is enve oped by a sheath of closely packed spores, which under the microscope appear in the form of a mosaic. The hair is denuded of its cuticle and eroded, and upon its surface and within its structure are found threads of mycelium running in a longitudinal direction. According to Fox and Blaxhall,[1] branched mycelium occurs first in the epidermis about the mouths of follicles, whence it gradually spreads down into the follicle encircling the hair. In the lower third of the follicle, above the bulb, the cuticle becomes frayed and allows the mycelium to make its way beneath it. The mycelium then spreads upward beneath the cuticle and for a short distance downward, forming a terminal fringe (Adamson's fringe). Immediately beneath the cuticle, it breaks up into short segments, which are arranged in rows or groups, and which, being large and swollen, are known as giant-spores. The latter divide into smaller spores, which coalesce to form the mosaic. The sheath extends upward to just beyond the intrafollicular portion, while the aerial portion of the hair-shaft is eroded and has only a few broken mycelial threads.

FIG. 357



Portion of a hair showing *Microsporon Audouini*. (From a photomicrograph.)

Microsporon lanosum (*canis*, Bodin) is of animal origin, of rapid growth, and produces epidemics in families. This form of ringworm is extremely contagious at all ages. It produces ringworm of the scalp in children and of the body and beard in adults. The organism is found in cats, dogs, and horses.

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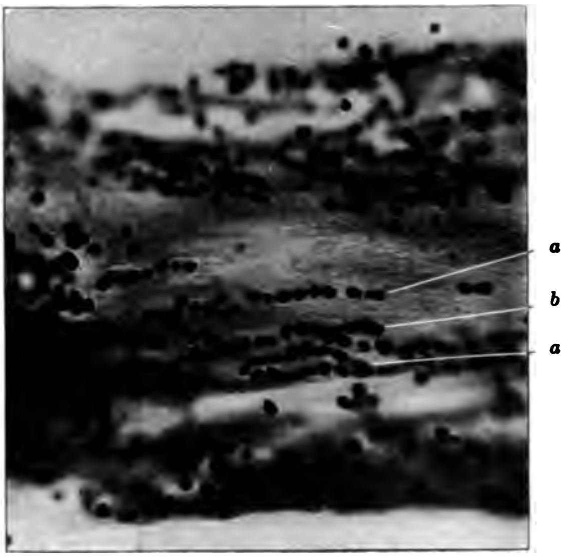
[1] Loc. cit.

Of the more than thirty species of trichophytons only a few need be more specifically described.

Trichophyton endothrix with resistant mycelium (*T. crateriforme*): This organism produces the yellow, crateriform cultures, and is responsible for the majority of large-spored ringworms of the scalp (*disseminated ringworm* of Alder Smith), some of the beard, a few of the nail, and many of the body ringworms. The mycelia entirely fill the hair-substance and appear as regular, straight, ribbon-like strings running downward parallel to the long axis of the hair and branching dichotomously. The spores are double-contoured and of quadrangular shape, and vary from 4 to 6 microns in diameter. At an early stage the spores and mycelia may occur outside of the hair, later entirely within.

FIG. 358

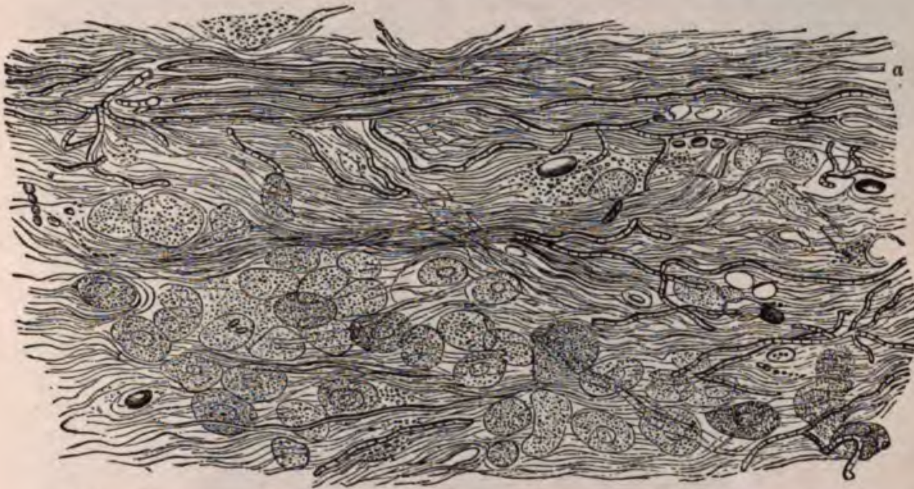


Portion of a hair invaded by the trichophyton endo-ectothrix. × 500. *a*, *a*, chains of spores in focus; *b*, a chain situated farther within the hair, and hence not in focus. (From a photomicrograph.)

Trichophyton endothrix with fragile mycelium (*T. acuminatum*): This organism is characterized by yellow, acuminate cultures, and is clinically indistinguishable from the form described above. It is responsible for a certain number of the black-dot scalp ringworms. Under the microscope the spores are seen like nuts in a bag. When a row of spores is seen it resembles a string of beads, and it is never the ladder-like arrangement of the crateriform spores. Their size is from 4 to 7 microns. The mycelium is fragile and should only be treated with weak solutions of potassium hydroxid.

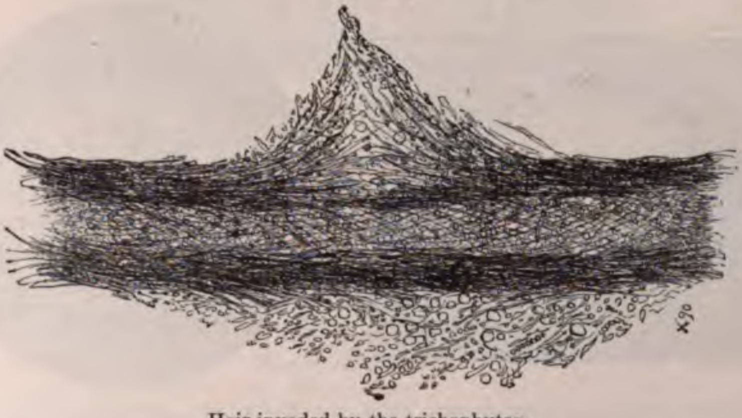
Trichophyton endothrix (*T. violaceum*) is found commonly in Italy, Roumania, Poland, Algeria, and Argentine. Fox found it in 15 per cent. of cases of ringworm of the scalp in London. This variety affects the scalp, beard, glabrous regions, and nails. The lesions produced are small black dots, and the organism is more active than the acuminate variety. During the stage of invasion, this trichophyton appears as an endo-ectothrix.

Fig. 359



Epidermis invaded by trichophyton: *a*, inferior portion of the stratum corneum; *b*, superior portion of the rete. Both exhibit long mycelial threads, with a few ramifications and a small number of spores. (Kaposi.)

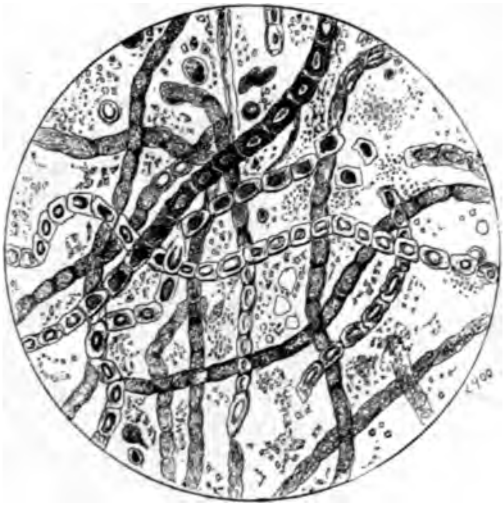
Fig. 360



Hair invaded by the trichophyton.

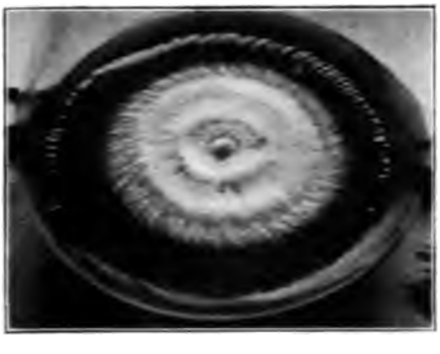
The ectothrix trichophytons, as a rule, are of animal origin, and are productive of markedly inflammatory lesions, resulting in suppuration, the disease produced being, however, of much shorter duration than that induced by the endothrix. Epidemics of ringworm induced by the microsporon and endothrix are common; those by the ectothrix uncommon. There are two main subdivisions of the ectothrix: a small- and a large-spored variety, or the microid trichophytons and the mega-

Fig. 361



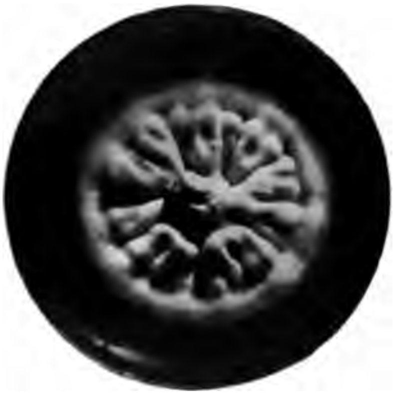
Filaments and spores of the trichophyton from the beard of a patient affected with trichophytosis barbæ.

Fig. 362



Culture three weeks old from ringworm of cat, contracted from ringworm of girl's face. (Mewborn.)

Fig. 363

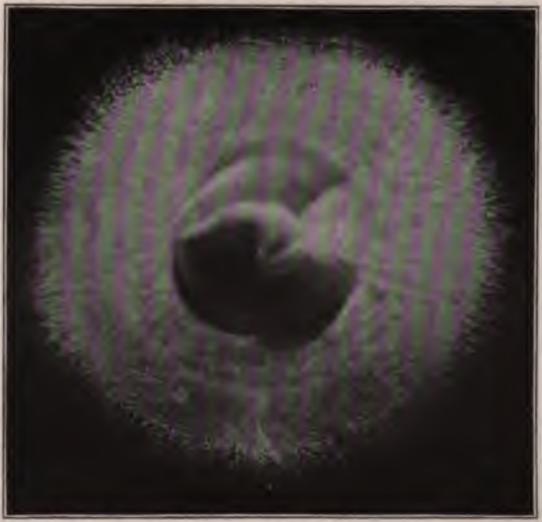


Trichophyton ectothrix culture (rose-pink in color), from a case of trichophytosis barbæ. (Mewborn.)

spores. The microids produce pyogenic lesions, and are therefore usually the cause of kerion. Their spores are small, usually about the same size as the microsporon, and there is a sheath about the hair. The chains of spores make the differentiation. An example of this group is *Trichophyton asteroides*. The lesions induced by this organism occur on the hands and wrists of adults, occasionally in the beard, and in the scalps of children. The lesions are kerion-like. It is said that the small-spored kerion is induced by this fungus.

Ectothrix trichophyton megaspore, type *T. rosaceum:* This was found by Sabouraud only eight times—in only 1 per cent. of all his cases. In the beard it produces characteristic lesions. Each hair occupies the summit of a little cone resembling the lesion of keratosis pilaris. The lesions are small and disseminated. The affection outside of the beard is uncommon.

FIG. 364



Trichophyton ectothrix culture three weeks old, from a case of trichophytosis barbæ. (Mewborn.)

As changes in culture media induce variations in growth of fungi, Sabouraud recommends the use of a common standard, as follows: Water, 3 liters; crude maltose (Chanut), 120 grammes; granulated peptone (Chassaing), 30 grammes; gelose, 54 grammes. In the above, glucose (Chanut) may be substituted for the crude maltose and in the same quantity.

Histology of the Skin when Affected with the Fungus.—The involvement of the skin by the ringworm fungus produces a certain amount of inflammatory reaction, which causes dilatation of the superficial bloodvessels of the corium and a hyperplasia of the thick cellular elements. In certain cases where the fungus penetrates more deeply, a true granuloma is formed, and in this plasma-cells, multinuclear cells, and other cells characteristic of this condition may be found. Edema and marked dilatation of the vessels may occur, with the accompanying changes which follow this process.

Treatment.—The indications in the treatment of trichophytosis corporis are the removal of the superficial layers of the epidermis, by which means the spores and mycelia are thrown off from the surface; and, if possible, the simultaneous destruction of the latter. Upon the delicate skins of infants and children the simpler remedies are first to be employed. Scrubbing each patch with spirit of green soap, or merely soap and water, will often suffice for its obliteration. The topical application of tincture of iodin is a common and usually an effective remedy. Sabouraud[1] states that tincture of iodin diluted with five times its volume of alcohol gives better results than the pure tincture of iodin. Dilute acetic and boric acids and phenol, or a 1 to 2 per cent. solution of formalin, are available. A solution of acetic acid used with or immediately before other parasiticides is said to favor penetration of the latter. Morris' solution of thymol,[2] ½ drachm (2.) to 2 drachms (8.) of chloroform and 6 drachms (24.) of olive oil, is equally available. One may also use thymol in ointments, ½ drachm (2.) to the ounce (30.) of simple unguent, with good effect. Of the mercurials, ammoniated mercury, 1 scruple (1.33) to the ounce (30.) of ointment; corrosive sublimate, 1 to 2 grains (0.066–0.133) to the ounce (30.) of solution; and the ointment of mercuric nitrate, 1 drachm (4.) to the ounce (30.) of vaselin, are valuable. Sulphurous acid, from a freshly opened can, and saturated solutions of sodium hyposulphite are as effective as any of the parasiticides, and are often used with advantage as lotions, to be followed by an appropriate unguent, always providing against chemical decomposition of the ingredients of the latter. Sulphur- and tar-containing lotions and unguents are useful in more obstinate cases.

Chrysarobin and pyrogallol, in ointment, from 5 to 10 grains (0.33–0.66) to the ounce (30.), are brilliantly effective in all these cases, subject, however, to the disadvantage incidental to the staining and irritative effects they produce. They should be used with caution upon the skins of children, and always tentatively at the onset. Chrysarobin used in the strength of 1 to 5 per cent. in traumaticin is a valuable application and has the advantage of being retained in the area applied. In cases of ringworm of the face of male adults close to the beard or the scalp, one may employ these remedies to prevent invasion of the hair-follicles by the fungus, the prompt destruction of which may then become a matter of urgency. Wilkinson's ointment, recommended by Kaposi, is also useful in the treatment of aggravated forms of ringworm of the body, but it should be restricted to such forms. For other and more urgent reasons potassium hydroxid solutions should be reserved for exceedingly intractable cases. Sometimes a combination of several of the simpler remedies named above may be serviceable, as in the following formulæ:

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[1] Brit. Med. Jour., October 8, 1908, pp. 1089 and 1094.

[2] Lancet, 1881, i, pp. 164 and 241.

℞—Sulphur præcip.,		ʒijss;		10	
Liniment sapo. mollis,					
Tinct. lavandul.,	āā	ʒvj;	āā	24	
Glycerin.,		ʒss;		2	M. (Kaposi.)

℞—Iodin. pur.,	℥ij;	60	
Ol. picis (sp. gr. 0.853),	℥j;	30	M.

Mix with care, gradually.

℞—Creasoti,	♏xx;	1 33	
Ol. cadini,	fʒiij;	12	
Sulphuris præcip.,	fʒiij;	12	
Potass. bicarb.,	ʒj;	4	
Adipis,	℥j;	30	M. (Van Harlingen.)

To be used in obstinate ringworm of adults.

R. W. Taylor applies mercuric chlorid, 4 grains to the ounce (0.266–30.) in tincture of myrrh. Perry, of California, uses the bichlorid, in one-half the strength last named, dissolved in sulphuric ether. Foulis, of Edinburgh, recommends iodin dissolved in oil of turpentine or benzin, as these fluids penetrate with greater ease the deeper portions of the skin.

Other articles advised are oleates of mercury and copper, croton oil, glacial acetic acid, cantharidal collodion, petroleum, and pyroligneous acid (Thomas).

The thorough application of the remedy selected for use upon the skin freed from scales by scrubbing with soap and water, is a matter of importance. When a solution of sodium hyposulphite is employed, the previous application of vinegar and water by sponging renders the agent more effective, for evident chemical reasons. Over-treated skins, or those to which too strong a parasiticide has been applied, require subsequent relief of the induced irritation by the simpler bland dressings. The inert dusting-powders, even when not thus indicated, are often useful when there is distinct vesiculation; and in simple cases they may be the sole remedies required, as then the disease is self-limited in duration.

For the eczematoid ringworm of the feet, Whitfield suggests an ointment or oil containing 5 per cent. of benzoic acid to 3 per cent. of salicylic acid, or an ointment of chrysarobin. Sabouraud recommends for the same condition the application of a 1 per cent. solution of iodin and chrysophanic acid. Chrysarobin in the strength of 5 per cent., in either traumaticin or an ointment base, has been uniformly successful in many cases treated by the author involving either or both the hands and feet.

The indication for the relief of trichophytosis capitis is the destruction of the parasite, and there can be no question that this can be accomplished in some cases without having recourse to epilation. Various parasiticides are employed. Prominent among these may be named formaldehyd (1 to 5 per cent. in aqueous solution), pyroligneous, sulphurous, acetic, salicylic, and boric acids, saturated solutions of sodium hyposulphite, acetum cantharidis, tincture of iodin; Crocker's ointment containing thymol, 1 part to 4 of salve-base; Morris' solution of thymol in chloroform and olive oil; and ointments of boric acid and sulphur, of each 1 drachm (4.) to the ounce (30.) of vaseline; and chrysarobin, the action of the latter being carefully limited to the patch of disease by the aid of a skull-cap.

Epilation, however, is a valuable and often an essential method of treating the disease, and it may be practiced as recommended when considering the treatment of favus. The scalp in each case should first be oiled and then cleansed by the soap shampoo, and after the epilation is performed an appropriate parasiticide should be employed. The calotte, made by spreading pitch-plaster upon leather or muslin, is a clumsy substitute for epilation in order to remove the hairs, but the sticks recommended by Bulkley may be employed, the formula for the preparation of which has already been given. In each case the epilation should remove a zone of sound hairs encircling the diseased patch, that the encroachments of the fungus may in every possible way be limited. It should not be forgotten, however, in the treatment of trichophytosis capitis by both epilation and parasiticides, that in chronic cases these methods in the hands of the most expert have failed for consecutive months to relieve radically the disease; that even the most inveterate cases, in the course of time and as adult years are reached, are relieved spontaneously without permanent alopecia; and that no remedy or procedure is ever justifiable which is capable of either producing follicular atrophy or an effect worse than that wrought by the disease itself.

Jackson recommends an ointment containing a drachm (4.) of iodin crystals to an ounce (30.) of goose-grease. This is rubbed into the scalp twice a day until swelling is produced. An alopecia follows, but the hair returns. Levan[1] applies oil of turpentine on linen twice a day for a week, or until inflammation occurs which produces exfoliation. The subsequent treatment consists in the application of a simple ointment. Hodara[2] applies daily, after shaving the hair, from 5 to 10 per cent. of chrysarobin in equal parts of glycerin and chloroform. On the production of erythema and edema the treatment is suspended until the irritation has subsided, and then is renewed. Four or five months are necessary for a cure. Sabouraud[3] prescribes the following method: The scalp is shaved and the hair epilated from the diseased area and from a zone 4 or 5 mm. wide surrounding it. Every second evening the entire scalp is rubbed thoroughly with 60 per cent. alcohol containing 25 per cent. of pure iodin. Beginning areas of the disease not visible to the naked eye are stained by the iodin and can be recognized easily. On alternate evenings an ointment containing pyrogallic acid, 15 grains (1.); oil of cade, 1 drachm (4.); and vaseline, 5 drachms

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[1] Jour. Mal. Cut., 1901, xiii, p. 241.

[2] Monatshefte, 1903, xxxvii, p. 118.

[3] La Pratique Dermatologique, iv, p. 508.

(20.) is applied. The scalp is washed each morning with soap and hot water. If this treatment fails to produce a follicular inflammation, croton oil is added to the ointment.

Coster's paste is popular among English practitioners, including Stowers, Fox, Liveing, and others. It contains 2 drachms (8.) of iodin in crystals, dissolved in 1 ounce (30.) of oil of tar; and is painted over the part at intervals of a few days. It is most useful in circumscribed patches of the disease. Williams[1] recommends the following formula: picric acid, 7 grains; camphor, 4 ounces; alcohol, 4 ounces; to be applied all over the scalp twice daily.

Among other remedies suggested for use on the scalp may be named mercuric chlorid, ammonio-chlorid, red oxid, oleate, and ointment of mercuric nitrate; pure phenol and carbolated glycerin; sulphur, chloroform, ether, tar in ointment, and Wilkinson's salve.

To be effectual, the treatment must be persistent and thorough, and always be accompanied by frequent washings and soapings of the affected part.

The induction of suppuration in the hair-follicles (or a species of artificial kerion), by the aid of electrolysis and croton-oil liniment, has been praised by Alder Smith and Wyndham Cottle, of London, and later, in a modified form, by Magee Finny, of Dublin. By the process of Finny, 100 parts of the oil are mixed with 50 each of cocoa-butter and white wax. Sticks are made of this compound, which can be rubbed thoroughly into the part affected. By both methods it is claimed that no pain is produced, nor is permanent alopecia the result. A solution of salicylic acid is applied after each treatment, and a subsequent poultice may also be needed. In these cases the parasite is destroyed presumably by the suppuration excited. As in the case of ringworm of the body, trichophytosis capitis is not remediable by internal treatment. Such internal medication, however, may be indicated by the systemic condition of young patients, and should be in each instance such as the condition suggests.

The treatment of kerion is either by the milder parasiticides or by the methods proper for the relief of ordinary phlegmonous inflammation of the scalp, according to the stage of the kerion. The pus-cocci present in some of these cases require boric-acid lotions and bichlorid washes. Engman[2] employs typhoid vaccine intravenously with excellent results.

Strickler[3] obtained good results by vaccine treatment.

When properly controlled, the treatment by means of x-rays gives best results. Sabouraud[4] revolutionized the ringworm situation in

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[1] Brit. Jour. Derm., 1912, xxiv, p. 216.

[2] Demonstration of results of treatment of large spored ringworm of the scalp of children and the beard of men with typhoid vaccine before the St. Louis Dermatological Society, November, 1920, showed several cases of cure. The vaccine was used intravenously, from fifty to one hundred million of the prophylactic typhoid suspension, every four days.

[3] Jour. Cut. Dis., 1915, xxxiii, p. 181, and Jour. Amer. Med. Assoc., July 15, 1915.

[4] Brit. Jour. Derm., 1906, xviii, p. 199 (A complete discussion of the subject, with interesting details as to technique and apparatus and results of treatment).

Paris when he introduced his method of treatment of the disease with *x*-rays. The method of Kienboch-Adamson[1] is also largely used. By this method a single exposure is given, which causes epilation of all the hairs, and with them the fungus, with consequent relief of the disorder. About two weeks after the exposure the hair falls, and, as a rule, within two months begins to regrow. The dosage is measured by Sabouraud's pastille, and other measures are taken to prevent undue exposure. In many thousands of patients treated very few cases of permanent alopecia have resulted. The treatment, however, should not be used except by an experienced operator, and cannot be recommended for general use.[2]

The treatment of trichophytosis barbæ is conducted generally as in trichophytosis capitis. It is customary to begin by anointing the affected surface with an oily or fatty substance, and to follow this with a shampoo of soap and warm water for the removal of crusts, after which shaving and epilation are practised on alternate days, and parasiticides employed locally. For softening the crusts the spray of an atomizer may be used.

Epilation of the male beard is often essential for removal of the disease, but the results of the treatment suggested below may be satisfactory.

The patient for two successive days keeps the affected part macerated with almond or olive oil. On the evening of the third day the shampoo with soap is employed, and the skin is washed free from crusts and scales. The part is then cleanly shaved. This operation is at first painful, but gradually becomes less distressing. After shaving, the affected surface is bathed for ten minutes with borated water as hot as can be tolerated, by which means the inflammatory condition of the perifollicular tissues is, in a brief time, considerably reduced. While the bathing is in progress all subepidermic pustules or points where a mucoid fluid is coming to the surface are opened with a fine, aseptic needle. A solution of sodium hyposulphite is then sponged freely over the affected surface for several minutes and allowed to dry. This solution may contain 1 drachm (4.) to the ounce (30.), or even more. After a thorough and final washing with hot water, the tender skin is carefully dried and gently smeared with a sulphur ointment containing 1 to 2 drachms of sulphur (4.–8.) to the ounce (30.) of vaseline, often with the addition of from ¼ to ½ (0.016–0.033) grain of mercuric sulphid. The patient then retires to bed. In the morning the unguent is washed off with soap and water, the sodium solution is reapplied, and a borated or a salicylated powder is thoroughly dusted over the part and kept on during the day. In the evening the shaving may be repeated or not, according to the vigor with which the beard is reproduced, but on the second day shaving is imperative.

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[1] Lancet, May 15, 1909.

[2] For technic and discussion see MacKee and Bemer, Med. Record, August 7, 1915; also Hazen, Jour. Cut. Dis., 1919, xxxvii, p. 307. The Roentgen-ray Treatment of Tinea Tonsurans.

As soon as the pustulation ceases and the tubercles have manifestly diminished in size, the ointment at night is superseded by the use at that time of the dusting-powder. Whether the shaving is practised nightly or on alternate nights, ablution with very hot water and with a solution of sodium hyposulphite is continued nightly until the inflammation excited by the fungus is practically limited to the follicles that are invaded. The dusting-powder is to be thoroughly and constantly employed after the ointment is discontinued. In many cases good results may be obtained by following the above technique, but substituting a 1 to 2000 solution of mercuric chlorid for the boric acid bath and sodium hyposulphite solution, after which an ointment is applied containing ½ to 1 drachm (2.-4.) of hydrargyrum ammoniatum to the ounce (30.) of adeps in place of the sulphur ointment. With care and patience these measures may save many patients the annoyance of epilation; and they should be continued for several weeks after apparent relief of the disease.

The treatment may be varied to suit the needs of individual cases. Kaposi highly recommends, for example, 1 per cent. solutions of mercuric chlorid locally; and the other parasiticides considered heretofore in connection with the treatment of ringworm may also serve a good purpose. In some cases an ointment of thymol may be used with manifest advantage; in others, a substitute may be found in Morris' solution of the same in chloroform and oil (the formula for this has already been given). In still other cases spirit of green soap with sulphur, finely powdered sulphur, boric and acetic acids and phenol, or other topical applications of recognized value may be employed.

When resort is had to epilation (and this is essential in all severe cases), the hairs should be thoroughly removed from their follicles over every lumpy nodule, and even over every suggestive patch covered with scales. A zone should be cleared about each such papule. The results are prompt and in the highest degree satisfactory. Beck[1] obtained good results by the hypodermic injection of tincture of iodin into the affected areas. Radiotherapy used in moderation, just sufficient to produce epilation, is a valuable addition to other methods.

Precautions to be Observed in the General Management of Tinea Favosa and Trichophytosis.[2]—The physician consulted in the case of a patient affected with either of the diseases thus far considered as resulting from the presence of a vegetable parasite should bear in mind that they are the most contagious of their class. He may not only suffer himself from the disease which he is attempting to relieve in another, but may also convey it to others, or be consulted by others of his patient's family actually infected during the course of the treatment pursued.

It is essential that the hands of the physician should be carefully washed after each manipulation of the part, and preferably with a weak disinfecting solution. In the case of children the lining of all caps, hoods, and other coverings of the head should be removed and destroyed by burning, and fresh linings, made of tissue-paper, renewed daily; while caps of the same or of similar material should be worn when indoors. Brushes, combs, towels, and articles of clothing should never be used in common by two or more individuals. When practicable, infected individuals should occupy separate beds; and the bed-covering, clothing, toilet-apparatus, and dressing or other materials which have been in contact with a diseased surface should be immersed in boiling water before they are again employed for any use in common. Thin recommends covering every diseased patch, after the treatment appropriate to itself, with an adhesive and impermeable dressing, for the sake, not of the patient, but of those with whom the latter may be brought in contact; and the suggestion is both wise and practicable. A man who has been infected with ringworm of the beard in a barber-shop which he has visited but once will often, when directed by his physician to shave, resort to some other establishment where he has more confidence in the cleanliness of the operators. In this way he often thoughtlessly spreads the disease of which he is the victim. It is well to send patients who cannot shave themselves to a particular barber, who has been instructed in the manner of shaving so as to insure immunity.

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[1] Jour. Cut. Dis., 1916, xxxiv, p. 606; Treatment of Tinea Barbæ.

[2] Corlett, Ibid., 1900, xviii, pp. 315 and 360. Lane, Jour. Amer. Med. Assoc., 1915, lxv, p. 1717. The Prevention and Treatment of Ringworm and Favus of the Scalp.

Tinea Imbricata.—Synonyms: Tokelau ringworm; Burmese, Chinese, or India ringworm; Bowditch-Island or Scaly ringworm; Lafa tokelau, Pita, Cascadöe, Gune, Herpes desquamans, Tinea circinata tropica, Gogo. Fr., *Herpès tonsurans desquamatif.*

This disorder was portrayed first by Alibert in 1832, and described first in 1844 by Fox, and has since been studied by Turner,[1] Manson,[2] MacGregor,[3] Königer,[4] Roux, and Castellani.[5]

Symptoms.—The eruption may appear in the form of patches made up of concentric rings, or be diffuse, resembling in the latter case ichthyosis. The scales in both cases are identical. After artificial inoculation, the disease first appears as a series of semicircles, made up of minute reddish points, which soon develop into papules, accompanied by intense itching. The growth thenceforward is reported to be at the rate of from 5 to 10 mm. each week. In a brief time, lamellæ of epidermis are detached, their free border being directed to the center of the circular disk, the patch or patches when fully developed being represented by concentric rings, about 5 mm. broad, suggesting a resemblance to watered silk. The scales may be as large square as half a centimeter, with curling edges, which later become horny and much darker in color. It is said that the hand passed over such patches from the circumference to the center recognizes a smoothness of the surface; but when the motion is reversed, from the center to the periphery, the scales are raised and resist the fingers. The appearance of the older patches suggests a skin covered with clay. The process of production of the concentric rings is reported to be, first, the elevation of a central point of epidermis and the formation there by the fungus of a brownish mass; then the separation of the epidermis at the central point, with persistence for a time of attachments at the border; next, the liberation of the attached edge by friction or otherwise; and, finally, the exposure of the corium. Just beyond this line, a brownish rim declares the line of advance of the fungus beneath the epidermis. When the ring thus formed has attained the diameter of about 5 mm., a brown point again appears centrally, and there is a repetition of the process originally observed in the primary ring.

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[1] Glasgow Med. Jour., 1870, p. 502.

[2] Tropical Diseases, p. 628; China Imp. Merit. Cut. Med. Rep., 1879, xvi, p. 1; Med. Times and Gazette, 1879, ii, p. 342.

[3] Glasgow Med. Jour., 1876, p. 343.

[4] Virchow's Archiv, 1878, lxxii, p. 413.

[5] Brit. Jour. Derm., 1913, xxv, pp. 377–401.

All portions of the body and large areas of the general surface may be affected. According to most observers, the scalp, face, palms and soles, axillæ, and nails usually escape, but Castellani has often observed the eruption in these situations. He states further that the nails may be affected and become much thickened, presenting a rough surface and deep fissures, scrapings from which show the causative fungus. The hair-follicles, according to all authorities, are spared, though the hairs are said to fall when the glabrous skin of the hairy region is involved. When the disease spontaneously disappears from any portion of the integument, there are left persistent, deep-colored rings or circles where the scaling originally occurred. The itching is commonly marked. A somewhat characteristic piebald appearance is produced in places where the scales have been removed and the resulting pigmentation is partial. The scaling is most marked in parts contiguous to healthy skin. The disease is chronic, as a rule.

Etiology.—Tinea imbricata is a contagious disorder affecting persons of both sexes and all ages, and is produced by a vegetable parasite. The disease in certain localities is endemic. It has been produced a number of times by experimental transmission from a diseased to a healthy skin.

Pathology.—Much confusion has existed relative to the causative organism, although all authorities have agreed that it is some sort of a vegetable parasite. Sabouraud and Nieuwenhuis believe it to be a variety of the large-spored trichophyton. Tribondeau reports that it is not a trichophyton, but an aspergillus, termed by him *Lepidophyton*. In Castellani's recent work, he reports that he has succeeded in growing the fungus and reproducing the disease by inoculating pure cultures of this fungus. To identify the organism, he has created a new genus, *Endodermophyton*. Two species have been separated, one of which he terms *concentricum*, the other *indicum*. He states further that the fungi belonging to this genus are characterized by their growth between the superficial and deep strata of the epidermis, forming an interlacing belt of mycelia, which detaches the horny and granular layers from the rete Malpighii. Botanically, they are closely allied to the achorions. Microscopically, the fungus presents interlacing mycelial tubes. The segments are rather regular in shape, somewhat square, and usually straight. Fresh preparations from young cultures show abundant septate mycelia. Reproduction, apparently, is by sprouting, branching taking place. No spore-bearing hyphæ are present.

Diagnosis.—The disease is to be differentiated from ringworm, which is readily done by the complete absence of all signs of inflammation; and by the large, flaky, firmly attached scales, and the peculiar concentric circles. In ichthyosis, a microscopic examination readily settles the question.

Treatment.—The scales are to be removed with soap and water or by alcohol baths, after which various medicaments may be rubbed in. Manson recommends strong linimentum iodi. Castellani endorses this recommendation and advises also resorcin 8 in tincture of benzoin 30; or chrysarobin in ointment in a strength of 5 to 10 per cent. Many other remedies have been employed with varying results. Dubreuilh[1] treated a patient intensively with the usual local applications employed for parasitic affections without result.

Prognosis.—Temporary removal of the lesions with treatment occurs, but recurrence of the disorder is the rule.

TINEA VERSICOLOR.

Synonyms.—Pityriasis versicolor, Dermatomycosis furfuracea, Mycosis microsporina, Chromophytosis. Ger., *Kleienflechte.*

Symptoms.—The eruption in this disorder occurs in the form of few or of many, irregular, roundish, circumscribed, or reticulated macules, pinhead- to small-coin-sized, rarely occupying an area the size of the palm or larger. In color it varies from the most delicate buff or fawn shade to a reddish, deep-brown, or even blackish hue. The surface of each lesion, when closely inspected, is usually seen to be covered with furfuraceous scales. If the scales are not visible, slight erasion with the finger-nail or the curette will demonstrate the fact that the superficial layers of the stratum corneum are, in the site of each lesion, readily separable from the tissues beneath. The eruption is most common upon the anterior surface of the thorax; but it is displayed also upon the neck, the dorsum, the abdomen, the other regions of the trunk, and the flexor aspects of the upper extremities (the hands only excepted). It rarely is seen upon the lower extremities; still more rarely on the face and on the hands and feet.[2] The eruption is either unproductive of sensation or is accompanied by mild itching. Patients usually declare that after profuse sweating, bathing in warm water, or brisk friction of the surface, minute epidermal rolls separate from the affected area. The disease may linger for years upon the surface of the body. It has a special tendency in susceptible individuals to recur after removal.

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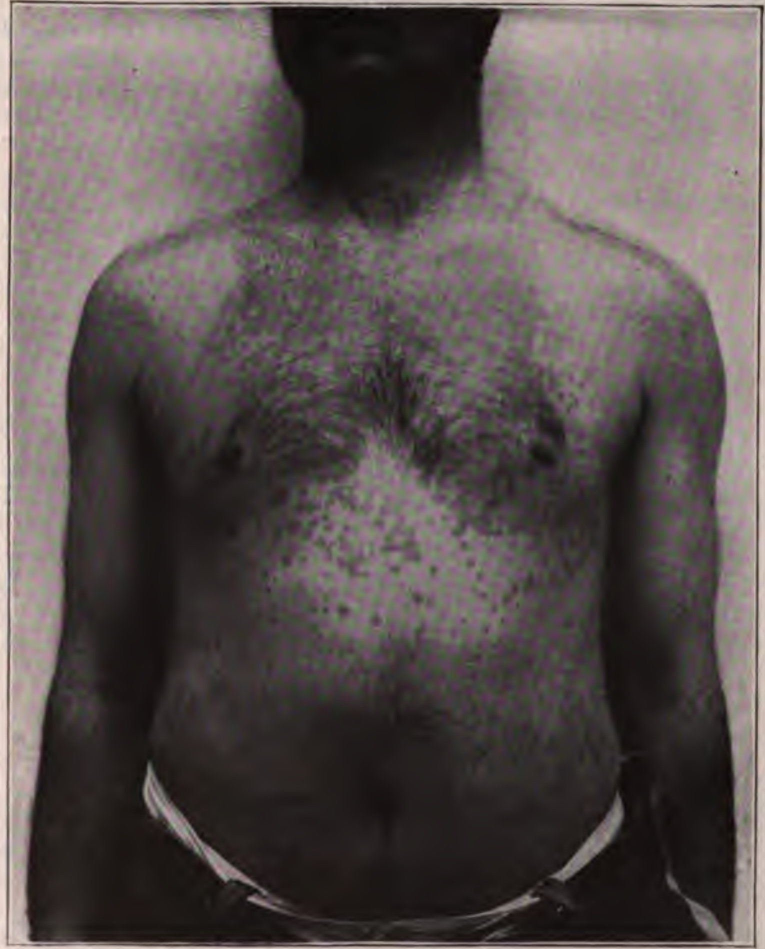
[1] Bull. Soc. franç. de dermat. et de syph., 1929, Nos. 5 and 6. A case of Tokelaw (Tinea Imbricata). Reviewed in Practical Medicine Series, 1919, vii, p. 65.

[2] E. O. Smith, New York Med. Jour., 1896, lxiv, p. 583, reports a case in which the disease was limited to both soles; and Gottheil, New York Med. Rec., 1899, lvi, p. 15, a case in which the left palm was involved.

The eruption is occasionally encountered in extreme development. In a young married woman who had been the subject of the disease for many years, the entire trunk, the axillæ, the groins, the upper portion of the thighs, the neck up to the level of the high collar worn, and the upper extremities down to the wrists, were encased in a uniform sheet of chocolate-tinted epidermis in a condition of exfoliation in finger-nail-sized, lamellated flakes. Even in these extreme cases the tendency of the disease to avoid surfaces exposed to the

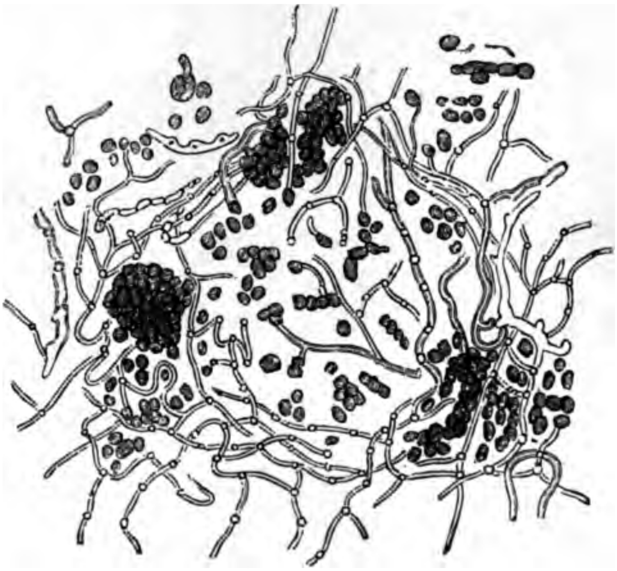
FIG. 365



Chromophytosis guttata. (From Dr. G. H. Fox's Atlas of Skin Diseases.)

light is distinctly manifested. Unna[1] describes an anomalous feature of the disease, in which the maculations occur in annular form with a clearing center. McEwen[2] recorded a case exhibiting follicular papules in addition to the ordinary lesions of the disorder. Rarely, also, a very few irregularly distributed macules may be seen as the sole evidence of the existence of the parasite. Thus, a patient may exhibit a small-coin-sized patch on the surface of the chest, another on the shoulder, and possibly a third over the deltoid region of one arm. These are generally cases partially relieved of a more diffuse eruption. More commonly, the slightest manifestation of the malady is an irregular, vertically arranged, somewhat narrow band of lesions immediately over the sternum, and visible beneath the hairs of that region in the adult male, or upon the intermammary sulcus of women. The face, hands, palms, soles, hairs, hair-follicles, and nails are usually exempt.

FIG. 366



Microsporon furfur. (After Kaposi.)

Etiology.—The disease is produced by a vegetable mould, discovered in 1846 by Eichstedt, to which Robin gave the name *Microsporon furfur*. Its transmissibility is far inferior to that of the vegetable parasites already described, and it illustrates well a point to which attention already has been directed, viz., that these fungi flourish only in soils suitable for their germination and fructification. Members of one family are said to communicate the disease occasionally the one to the other; and Lancereaux[1] reports that in this way he accidentally infected himself from scales collected for examination from a patient in hospital, and afterward unwittingly transmitted the affection to his wife. The disease occurs in both sexes, rarely before puberty or after middle life, and in persons of every social condition, irrespective of personal cleanliness. It is exceedingly common, more so, indeed, than statistics are capable of demonstrating, inasmuch as hundreds who are annually annoyed by it never seek professional advice. In physical examinations of men for military service, as also of government pensioners, the disease is often recognized upon the persons of those who pay no attention to its presence. Being concealed by the clothing and unproductive of much discomfort, many subjects of tinea versicolor endure its presence with unconcern.

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[1] Vierteljahr., 1880, xii, p. 165.

[2] Jour. Cut. Dis., 1911, xxix, p. 19.

By some it has been supposed that the fungus selects the chest of the phthisical as its habitat, a supposition doubtless based upon the fact that tuberculous men and women, more than all others, expose the chest to the view of medical men in order to permit of its auscultation and percussion.

Pathology.—*Microsporon furfur* (Fig. 366) is readily recognized with the aid of the microscope, as it exists in luxuriant profusion upon every affected surface. The scales may be scraped from the skin, placed on a slide moistened with a 1 to 10 per cent. solution of potassium hydroxid, and be examined at once, when innumerable clustered spores and short threads become visible; the former highly refractive and resembling in their circular and oval contours droplets of oil. Their aggregation in clusters is distinctive among the other forms of cryptogamic vegetation. They measure 0.0023 to 0.0084 mm., while the hyphæ vary in diameter from 0.0015 to 0.0038 mm. (Duhring). Among the latter, sporophores are distinguishable, with contained conidia and terminal elements emerging at one extremity or the other of the spore-case. Both elements are stained more readily by eosin and methyl-violet than those of the trichophyton or of favus. By the use of special media, Matzenauer[2] and Gastou and Nicolau[3] have succeeded in cultivating the fungus.

One of the strongest arguments against the claim for the identity of all the vegetable parasites is furnished by the history of this interesting mould. It never by any possibility invades the hairs, and rarely the hair-follicles, though it may be seen flourishing at the orifice of a follicular duct, and even beneath a vigorous hairy growth upon the chest of a male subject. It avoids light and air, and singularly refuses to encroach even upon certain covered portions of the body, preferring, for instance, in its extreme development, to linger unobtrusively at the neck, near the verge of the collar.

Diagnosis.—In this disease, as in all parasitic affections of vegetable origin, the microscope may be required to decide the diagnosis. In its simpler manifestations the recognition of the affection is readily assured. The location of the eruption, its irregular reticulations, its characteristic yellowish or fawn-tinted color, due to the nature of the fungus, and the exfoliation of the epidermis which it excites by its superficial penetration of the outer layer of the stratum corneum, producing thus a mealy, branny, flaky, or roll-like exuvium, are all significant. None of the chloasmata due to pigment-changes in the skin, however much they may resemble tinea versicolor in color, shares with it this peculiarity of desquamation. Chloasma may involve, moreover, the face; tinea versicolor almost never. Vitiligo commonly occurs on the forehead, face, and dorsum of the hands; tinea versicolor very rarely. The macular syphiloderm may be mistaken for the disease under consideration, but, when developed to such an extent as to rival tinea versicolor in its diffuseness, the syphiloderm will creep out over the face, the hands, and the feet, and will be accompanied by adenopathy, alopecia, mucous patches, palatine hyperemia, or will furnish evidence of a polymorphic tendency. Often, indeed, with such an eruption, the survival of the initial sclerosis will at once betray the nature of the disease. These are important considerations, since in the mere matter of subjective sensation, color, shape, and size of lesion there may be marked resemblance between the two disorders. Patients exhibiting the lesions of tinea versicolor may suffer from syphilis; and many having the former disease believe, in consequence of a suspicious exposure, that they are infected with lues, and yet, indeed, are not. These incidents serve to illustrate the importance of making an accurate diagnosis in every case of cutaneous disease.

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[1] Traité d'Anatomie pathol., p. 265; Paris, 1875.

[2] Archiv, 1901, lvi, p. 163.

[3] Bull. de la Soc. franc. de Derm., 1902.

The most common error committed in this connection, however, is based upon the fancied resemblance in color between the patches of tinea versicolor and either the liver itself or the color-changes which disease of that organ is capable of producing in the skin. Hence, the existence of "liver-colored" spots in the skin is erroneously attributed to hepatic disease. Few patients consult physicians for relief of this disorder who have not a belief in the internal origin of the disease.

Treatment.—A successful method of treatment is as follows: A hot bath is taken, if possible, for three nights in succession, and when the surface is well macerated in hot water the affected skin is scrubbed either with the cheap yellow soap of the shops, or with sapo mollis in substance or in tincture. When the disease is extensively developed, this process is aided by friction with a flesh-brush or with a coarse towel. The skin is then washed clean with a surplus of hot water and dried, after which the affected patch is first moistened with vinegar and water, or dilute acetic acid, and afterward well sponged with a solution of sodium hyposulphite, 1 drachm (4.) to the ounce (30.) being usually sufficient. As a rule, the greater part of the eruption is removed with the third application. If there be recrudescence in isolated patches, as is often the case, or outlying areas which have withstood the parasiticide employed, they should subsequently be attacked with a solution of mercuric chlorid, 1 to 2 grains (0.066–0.133) to the ounce (30.). Another method employed with success by the author consists in the local application of an ointment containing 6 per cent. of salicylic acid and 12 per cent. of benzoic acid in petrolatum. This formula was originally recommended by Whitfield in the treatment of ringworm of the hands and feet. Other measures, however, are popular with physicians, and among them may be named the topical use of boric or sulphurous acid, phenol, and tincture of iodin; sulphur in bath, ointment, or lotion; calomel in ointment; the alkalies in bath or lotion; potassium sulphuret in bath; chrysarobin, pyrogallol, tar, Wilkinson's salve, and the other parasiticides employed in the treatment of ringworm of the body. Leven[1] secures an exfoliation of the skin and a removal of the disease by the embrocation of oil of turpentine four or five nights in succession. Whatever parasiticide be employed, after treatment the inner clothing should not be worn until it has been immersed in boiling water.

The following formula is also recommended:

℞—Hydrarg. chlorid. corros.,	℈j;	1	33
Saponis mollis,	℥ij;	60	
Alcoholis,	℥iv;	120	
Ol. lavandul.,	f℥j;	4	M.

(Anderson.)

Prognosis.—The disease can readily be relieved by simple treatment. Relapses often occur, and require to be radically treated. Untreated, the disease may continue for years without the slightest impairment of the general health. It is probable that when untreated the parasite undergoes spontaneous exfoliation in advanced years, a period when, presumably, the fungus fails to find in the epidermis the nutriment upon which it thrives.

ERYTHRASMA.[2]

Burckhardt first described this disorder in 1859, but it received its name in 1862 from von Bärensprung. It has since been studied and described by Balzer, Riehl, Koebner, Pick, and others.

Symptoms.—The disease first appears in punctiform to palm-sized, roundish, definitely circumscribed maculations, presenting a sharp contrast in color to that of the adjacent integument. This hue varies somewhat according to the location of the patches. The younger lesions may exhibit a vivid redness over the entire surface or over their borders only. The older lesions exhibit a yellowish or a brownish tinge. These colors are compounds of ordinary erythematous redness and yellowish or brownish discoloration of the horny layer of the epidermis.

The macules are circular or rosette-shaped, or they may display very irregular outlines. They are not raised to any great extent above the general level of the skin, though the finger passed over the surface can recognize a slight elevation of the border, due to hyperemia, and subsequent moderate, flour-like, furfuraceous desquamation, most conspicuous also at the periphery. Vesiculation and papulation do not occur. The colors recognized in different patches may be light reddish-brown, pale reddish-yellow, and light or dark orange.

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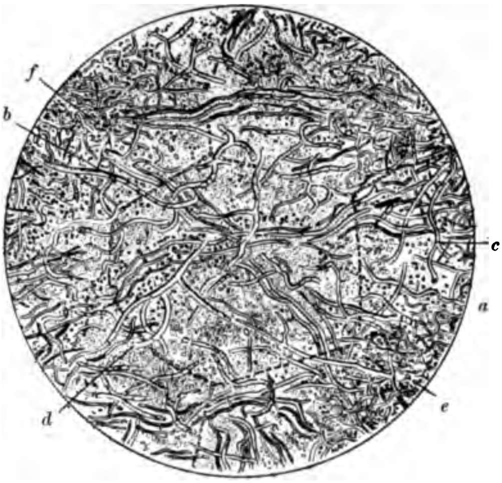
[1] Monatshefte, 1901, xxxii, p. 197.

[2] Payne: Some Rare Diseases of the Skin, London, 1889 (review of literature); Balzer, La Pratique Dermatologique, ii, p. 540 (bibliography).

The eruption is most commonly encountered where apposed surfaces of the skin come in contact, as in the axillæ, the groins, the cleft of the nates, and the regions where the scrotum touches the thigh; it occurs, however, in typical expression on both sides of the chest. The eruption spreads slowly and in serpiginous outline until the affected surfaces are completely invaded. It is much more chronic in its course than the other dermatomycoses, lasting for months and years without apparent change.

FIG. 367



Microsporon minutissimum, from patches of erythrasma.

Etiology.—Erythrasma is produced by the growth, in the superficial layers of the epidermis, of the fungus described below. Men are much more affected than women; children not at all. The youngest patient whose case is recorded was sixteen years old; the oldest fifty-five.

Pathology.—The fungus, termed *Microsporon minutissimum* (Fig. 367), to which the disease is attributed is chiefly remarkable for the extraordinary delicacy and fineness of its threads and its very minute spores. The threads are either simple cylindrical bodies of variable size, or they may exhibit partition-septa; they may divide dichotomously, and may terminate in hooked or knobbed expansions. They are inextricably interwoven when occurring in large masses. The largest transverse diameter is 0.6μ; in length the mycelium presents the greatest variation. Bacteria and heaps of zoöglea are visible among the scales. The granules are piled into irregular heaps, according to Burckhardt, and they give a dusty appearance to the epidermal cells on which they lie. Often the outline of these granules is indistinct. According to the same observer, the breadth of the hyphæ is $\frac{1}{1200}$ mm., and the length from $\frac{1}{15}$ to $\frac{1}{200}$ mm.

Pasquale de Michèle[1] discovered the leptothrix in cases of supposed erythrasma.

Diagnosis.—From all ordinary chloasmata and pigment-macules the spots of erythrasma are distinguishable by the ease with which the superficially embrowned epidermal layers are removed by erasion. Tinea versicolor is distinguished from erythrasma with greater difficulty; but the latter occurs by preference in different situations, its patches are more vividly tinted, and the parasite, under the microscope, presents distinctive features.

Treatment.—Treatment is that of tinea versicolor.

Prognosis is favorable, subject to the disappointments arising from frequent relapses.

PINTA.

Synonyms.—Mal de los pintos, Mal pintado, Pinto, Caraate or Cute, Cativi, Tina, Quirica, Spotted sickness, Spotted disease of Central America, Pannus carateus. Fr., *Caraté.*

Definition.—Pinta (Spanish, *pintar*, to paint) is an endemic contagious dermatomycosis, characterized by the development of pigmented patches of different colors upon the skin, unconnected with the general health of the patient, and affecting subjects of both sexes and all ages.

The disease occurs in tropical America, especially in Mexico, Central America, Venezuela, Colombia, Bolivia, Chili, Peru, and Brazil, but has been found also in North Africa, and, it is believed, also in Guiana.[2]

Symptoms.—Pinta begins at one or several points of the body-surface, whence it is distributed more or less generally by auto-infection. The disease is said by some authors to be preceded by prodromata of chills, fever, anorexia, cephalalgia, diarrhea, and emesis, lasting for one week, the cutaneous symptoms developing about one month later. The occurrence of such a prodromal stage has, however, been denied.

The eruptive symptoms develop gradually. The hands, face, and other exposed parts usually are involved first. Large areas subsequently are formed by increase in the dimensions of the original macules, and also by their coalescence, the spread of the disease being asymmetrical and peripheral. The spots may be characteristically yellowish, reddish, bluish, blackish, whitish, or violet, the hue at first being monochromatic; later, as the disease spreads, the different colors named above may be exhibited side by side. The patches are well defined. On the scalp the hairs whiten, become atrophied, and fall. The follicle is said not to be invaded by the fungus, but is attacked by a peculiar fibrosis, which induces the atrophy (Castellani). The disease does not affect the palms and soles.

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[1] Annales, 1891, s. iii, ii, p. 776.

[2] Bibliography: Manson, Tropical Diseases, p. 776. Gomez: Du Carathés ou Tache endémique des Cordillères, Paris, 1879. Hirsch, Handbuch histo-geogr. Pathologie, 1883, 2d ed., ii, p. 263. Montoya and Florez, Annales, 1897, s. iii, viii, p. 464. Castellani and Chalmers, 1913, p. 1513. Wooley, Jour. Cut. Dis., 1904, xxii, p. 479.

The surface of the body, when extensively involved, presents an odd-looking, piebald appearance, due in part to epiphytic changes and in part to the development of vitiliginous patches in the skin. Itching is produced in various degrees, according to the extent and severity of the disorder. When the affection has lasted for some time, a disgusting odor is exhaled, and the surface exfoliates an early furfuraceous desquamation, which is replaced later by scaling in large flakes.

Two types are described: one superficial, represented by blackish and bluish patches; and a deeper form, said to be more obstinate, with reddish and whitish patches, in which the deeper portions of the epidermis are involved.

Though displayed for the most part asymmetrically, the patches may cover the entire surface of the body, and even invade the mucous membranes of the alimentary tract. When confluence occurs, large areas of the skin may be involved, displaying then the usual features of hyperkeratinization, with pityriasic, occasionally larger and coarser, scales, infiltration, occasional fissuring, and complete or partial color-change and loss of hair. In final evolution, the symptoms are highly suggestive of other dermatoses, such as trichophytosis, favus, some of the forms of lupus, and erythematous eczema. There may be ulcerative complications.

Etiology.—Pinta is a contagious disease affecting persons of both sexes and all ages save infants; but it is much more common among the filthy and the neglected than in others. It is produced by the growth of a cryptogamous fungus in the superficial portions of the skin.

Pathology.—Scales scraped from the skin, moistened with potassium hydroxid and placed under a microscope exhibit round or oval, blackish spores, 8–12μ in diameter; and highly refractile, short, dichotomous filaments of mycelium are distinguishable. The effective parasites recognized by Montoya y Florez (cited by Manson) are fungi, chiefly *Penicillium*, *Aspergillus*, and *Monilia*. Fine filaments furnish hyphæ, which terminate in clubs, surrounding chaplets of spores. Sterigmata encircle the sporulating elements. The fungi are found chiefly in the superficial layers of the epiderm, but may also, in advanced cases, be recognized in the rete. Whether the differences in color be due to variations in the fungi or to pigmentation of the spores and filaments is not determined.

Diagnosis.—The patches of chloasma, vitiligo, and lepra are distinguished readily from those of pinta by considering that, in the two diseases first named, there are no surface-changes in the horny layer of the epidermis, and in the second the existence of a systemic affection is established readily. The absence of anesthesia in the patches of pinta, the discovery of *Microsporon furfur* in tinea versicolor, and of *Microsporon minutissimum* in erythrasma, and the special characters of the psoriasiform dermatoses, are all of significance. Care should be taken to exclude the symptoms of the prefungoid stage of granuloma fungoides.

Treatment.—Chrysarobin, sulphur, iodin, naphtol, and, if needed, corrosive sublimate lotions have been found useful. Cleanliness and strict observance of the requirements of hygiene are demanded, especially in the class of patients who are affected most often by the disease.

Prognosis.—The disease may persist indefinitely if not relieved. It yields to proper parasiticidal treatment. Mild relapses occur. The general health is not involved.

MYCETOMA.[1]

Synonyms.—Podelcoma, Madura foot, Morbus pedis entophyticus, Ulcus grave, Endemic degeneration of the bones of the foot, Morbus tuberculosis pedis, Elephant foot, Madura disease, Fungus foot of India, Fungus disease of India, "Egg-foot." Ger,, *Madurafuss;* Ind., *Perical, Slipada;* Fr., *Mycétome, Pied de Madure.*

Definition.—Mycetoma is a localized affection, limited to the skin and adjacent parts, due to invasion of the tissues by vegetable parasites, and characterized by the production of an unshapely tumefaction of the invaded part, which becomes covered with nodules or tubercles, for the most part penetrated by fistulous sinuses. The disease not only affects the skin, but also the underlying structures to a variable extent. It long was thought to be a malady occurring only in India, but more lately has been recognized in China, Syria, parts of Africa, and in both North and South America.

The disease was first referred to by Kaempfer,[2] in 1712, though Carter was the first to describe it as an entity caused by a specific parasite. American cases have been recognized and recorded by Adami and Fitzpatrick; Hyde, Senn and Bishop; Arwine and Lamb; Pope and Lamb; Wright, and Sutton. In addition, Parkes reported operating on a case of mycetoma in Chicago. The disease, however, in this instance was contracted in India.

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[1] BIBLIOGRAPHY: Adami and Kirkpatrick, Trans. Assoc. Amer. Phys., 1895, x, p. 92. Arwine and Lamb, Amer. Jour. Med. Sci., 1899, cxviii, p. 293. Boyce, Hyg. Rundsch., 1894, iv, No. 12. Surveyor, Brit. Med. Jour., 1892, p. 575. Carter, Treatise on Mycetoma, or the Fungus Diseases of India, London, 1874. Dantec, Le, Arch. de Méd. naval, 1894, p. 447. Gémy and Vincent, Annales, 1896, s. iii, vii, p. 1253. Hatch, Keith, and Childe, Lancet, 1894, p. 1271. Hyde, J. N., Senn, N., and Bishop, D. D., Jour. Cut. Dis., 1896, xiv, p. 1 (bibliography). Kanthack, A. A., Lancet, 1892, i, p. 195, and ii, p. 169; Jour. Path. and Bact., October, 1892. Manson, Tropical Diseases, 4th ed., p. 760. Paltauf, Intern. klin. Rundsch., 1894, No. 26. Pope and Lamb, New York Med. Jour., 1896, lxiv, p. 368. Ruelle, Contribution a l'Étude de Mycétoma, Bordeaux, 1893, p. 13 *et seq.* Scheube, Diseases of Warm Countries, 1903, p. 552 (full bibliography). Shah, T. M., Med. Rep., Calcutta, 1893, p. 225. Vincent, Annales de l'Inst. Past., 1894, p. 129. Wright, Trans. Assoc. Amer. Phys., 1898, xiii, p. 471. Emma Dübendorfer, Ein Beitrag zur Histologie und Bakteriologie des Madurafusses, Archiv, 1907, lxxxviii, Band 1, pp. 3–10 (1 clin. illustration, 4 hist. illustrations). Clemon, Brit. Med. Jour., April 21, 1906, p. 918. Oppenheim, Archiv, 1904, lxxi, p. 209; abstr. Jour. Cut. Dis., 1905, xxiii, p. 411 (pathological anatomy of the black variety). Wright, Osler's System of Modern Medicine, i, p. 344. Musgrave and Clegg, Philippine Jour. of Sci., 1907, ii, No. 6. Sutton, Jour. Amer. Med. Assoc., 1913, lx, p. 1339: Mycetoma in America (report of two cases, with histological study; bibliography. Chalmers and Archibald, New Orleans Med. and Surg. Jour., November, 1917; Mycetoma and Pseudo-mycetomatous Formations. Reviewed in Practical Medicine Series, 1918, vii, p. 92.

[2] Kaempfer, 1712: *Amœnitatum Exoticorum Politico-Physico-Medico*, v, p. 561 (quoted by Castellani and Chalmers, Tropical Medicine, 2d ed., p. 1527).

Symptoms.—Three varieties of mycetoma were once loosely distinguished by the color of the morbid material contained in the discharge, viz., the black, the red, and the white or ochroid; the last-named the most common, the second the rarest, the black rather less frequently encountered than the white. The part principally affected in most of the Indian and in the American cases is the foot, and this chiefly of persons walking barefoot; but the hand, the shoulder, the thigh, the knee, the toe, the abdominal wall, the scrotum, and other regions have been attacked. Simultaneous involvement of different regions of the body has never been noted.

In a typical case the sole of the foot is involved by progressive spread of the disease from the site of a trifling traumatism, which often at first heals, but is followed later by the development, near the site of the wound, of a rounded, firm, painless, small-nut-sized, subcutaneous button or nodule, which increases slowly in volume and later is surrounded by similar lesions. In the course of five to ten weeks or more, the tumor softens and bursts, discharging a characteristic viscid, "oily," semipurulent, blood-streaked fluid, which contains minute, roundish (grayish, reddish, or blackish) particles, which have been compared to fish-roe. These may be agglomerated in pea-sized masses. At the site of each lesion a permanent sinus penetrates deeply beneath, and is said never to undergo spontaneous healing. The repetition of the process by the multiplication of nodules and fistulous tracts produces eventually the deformity characteristic of the disease. The progress of the malady may be exceedingly chronic, as ten and more years have been recorded not rarely as requisite for its complete evolution.

In fully developed cases, when the foot is involved, that organ is seen to be largely increased in volume, producing without elongation an elastic bulging of the parts posterior to the digits, over the dorsum above and below over the plantar region, giving the sole a convex appearance. The toes may be in forced separation or misdirected. Over the tumid parts, the skin is beset with numerous pea- to nut-sized, isolated nodules, elevated to the extent of several millimeters above the general level, each pierced with a fistulous channel extending from without to the deeper structures. At times these fistulous tracts lead only to the soft parts, and especially to muscular tissues; at others the surface of the bone is reached and the osseous tissue is eroded by the growth of the parasite and the coccogenous infection which results from long exposure of the parts to the air. It is through these fistulous orifices that in different cases exit is given to a blackish, fish-roe-like substance, or to a whitish material, or even still more rarely, as indicated above, to a reddish substance.

In place of nodules or papules, the skin may be the seat of pustules, vesicles, bullæ, or even of abscesses. When but relatively small organs of the body are invaded, such as a finger or a toe, it becomes clear that the tumefaction is not due chiefly to a hypertrophy either of the integument or the bones. When the foot is affected seriously, the leg above commonly atrophies from disuse.

The discharge varies in different cases. In some it is almost wholly wanting; in others it is scanty; in yet others exceedingly profuse and fetid. It is generally oily or syrupy in character. When blackish in hue, the contained granules have been likened to truffles or fish-roe; when of paler hues, it resembles fish-spawn.

The course of the disease is exceedingly chronic; and while one or more nodules have been seen to heal, the mass of the disease persists until relief is obtained by artificial methods.

Pain is usually not pronounced; sensibility is maintained; and the general health may be undisturbed for long periods of time. Death, when it ensues, results from long-continued drain upon the vitality of the patient.

Etiology and Pathology.—The disease is caused by invasion of the tissues of the body by vegetable parasites. It is probable that the parasites secure access to the skin through the medium of a traumatism; and the occurrence of a large majority of all cases on or near the foot, most often in men, and among individuals who have been walking barefoot, lends support to this view. Further, the origin of a few cases has been traced to foot-lesions (bruising of the organ with a stone and consequent abscess; injury with a pitchfork, or a fall on the knee); while the relative freedom of persons who protect the feet while residing in the districts where the disease is common is in evidence. The lower classes of poor agriculturists, during the twentieth to the fiftieth year of life, are most liable to the affection, while children and infants escape. The origin of the disease has not been traced to any peculiarities of soil.

Several varieties of the fungus producing the disease in its various manifestations have now been differentiated. Castellani and Chalmers arrange them, according to the color of the granules, into three groups: a melanoid, an ochroid, and a red group. These are further subdivided into fifteen varieties.[1] On microscopic examination the fungi present many resemblances to the actinomyces, but their identity is not accepted by most investigators. Morphologically and tinctorially, the differentiation may be made.

In a well-advanced case, on section the foot is found to be tunnelled in various direction by sinuses, which communicate with oval or roundish cavities. The latter may be superficial or deep, and may occupy the soft tissues or the bones, and have a connective-tissue lining. Here the granules, singly or in masses (mulberry-like), are found, as well as in the neighboring softened tissues.

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[1] For details, see Castellani and Chalmers' Manual of Tropical Medicine, 2d ed., pp. 1533, 1534, and 1535.

In the period of early reactive inflammation, the fungus is surrounded merely by the results of cell-proliferation; typical granulation tissue, epithelioid cells, and new vessel-formation follow. Somewhat later plasma-cells, giant-cells, and amorphous granular masses occur, and finally degenerative changes attack the entire area.

The bones when denuded are found to be honeycombed with finely carved seams, depressions, furrows, and pits, leaving delicate spicules of osseous tissue projecting between the excavations wrought by the growth of the parasite. It is possible to find, as Adami suggested in the study of his case, intrusive organisms, the result of exposure for so long a period of time of the deeper tissues to the atmosphere.

Diagnosis. — In all cases of long standing, the disease is readily recognized by the characteristic deformity produced; in certain varieties of the affection by the escape of fish-roe-like particles; and in all by microscopic recognition of the parasites present. In well-defined instances, the nodes or papules externally visible are often perforated with sinuses leading downward to the deeper structures. The painlessness of the invaded part is also characteristic.

Treatment.—Early in the disease potassium iodid may be employed, with curettage and packing of the superficial lesions. As a rule, amputation is the only method of eradicating the disease.

Prognosis.—As spontaneous recovery does not occur, the prognosis depends upon the treatment employed.

ACTINOMYCOSIS OF THE SKIN.

Synonyms.—"Lumpy-jaw." Ger., *Aktinomykose;* Fr., *Actinomycose.*

In 1877 Ponfick proved that the disease first recognized by Bollinger in the jaws of cattle was the same as that which Israel, in 1877, had observed in man.[1] Hartz, judging largely from its morphological character, described the parasite as the ray-fungus. Majocchi was the first to describe the disease as it involves the skin.

Symptoms.—In actinomycosis, the parasite commonly gains access to the economy by the mouth, especially by the avenue of a carious tooth; and the skin, when implicated, as a rule is involved secondarily. Such skin-lesions as are displayed are more often about the face and neck, more particularly the lateral surfaces of the neck beneath the jaw, where deep subcutaneous nodes, tumors, or swellings may form.

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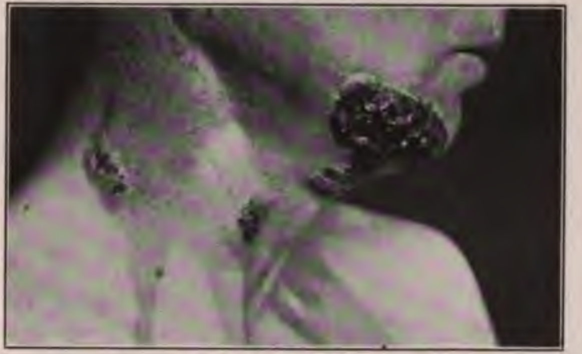
[1] REFERENCES: Neumann's Atlas, Plate XIII; Morris, Lancet, June 6, 1896; Pringle, Med.-Chir. Trans., 1895; Kopp's Atlas, Plate LXXV; Corlett, cut appearing in Stelwagon's Treatise, 1914, p. 1155; Illich, Wien, 1892; Darier et Gautier, Annales, 1891, p. 449; Ponfick, Treat., Berlin, 1882; Israel, Treat., Berlin, 1884; Skerritt, Amer. Jour. Med. Sc., 1887; Poncet et Bérard, Trait., Paris, 1898; Bodamer, Med. News, 1889; Crookshank, Lancet, 1898, p. 11; Legrain, Annales, 1891, s. iii, ii, p. 772; Baracz, Wien. med. Presse, 1889, xxx, p. 6; Ljunggren, Nord. med. Arch., 1895; Kopfstein, Wien. med. Rundschau, 1901, p. 21; MacCullum, Centralb. o. Bakliv., 1902, xxxi; Howard, Jour. Med. Research, 1903, ix, p. 301. Newman, Jour. Cut. Dis., 1916, xxxiv, p. 290; Actinomycosis: Cutaneous and Systemic. Little, Brit. Jour. Derm., 1918, xxx, p. 218; case report. Actinomycosis in child aged twelve years.

These are often firm to the touch, livid in hue, thinning at one or at several points after involvement of the integument, and finally bursting, forming fistulous tracts, which give exit to a sero-sanguineous or bloody and purulent fluid, containing friable, yellowish or grayish masses, in which the fungus may be recognized. The orifices of the sinus or sinuses after such discharge are usually beset with cutaneous and subcutaneous nodules and uneven lumps, some softened, others firm and indurated, usually reddish or purplish in hue, tender, painful, and often accompanied by pains elsewhere, particularly in mastication, in deglutition, and in certain movements of the head. The outlying skin becomes infiltrated, tumid, empurpled, and boggy. Rarely papillomatous growths develop.

Primary actinomycosis of the skin is rare. Wallhauser[1] observed an extensive case, in which the primary infection occurred on the point of the chin, from without. From this site the disorder spread by the formation of papules and nodules in the usual manner. Merian[2] found only 25 recorded cases occurring primarily in the skin. A case of this type involving the dorsal surface of the hand was reported recently by Pels.[3]

FIG. 368



Actinomycosis.

The onset of the disease is insidious, and, though occasionally rapid in its career, its evolution may extend over months and even years. The nearer to complete development of the disease, the more rapid, as a rule, is the oncoming of its symptoms. In exceptional cases the malady attacks the fingers, the hands, and other parts of the body. Rarely, secondary actinomycosis of the lymphatic glands occurs. Pringle reported a case in which large areas on the back, lumbar region, and hip were affected secondarily after involvement of deeper organs.[4] Lymphatic metastasis is, however, rare, due, it is believed, to the large size of the fungus-granules as compared with the lumen of the lymphatic vessels. Subjective symptoms may be insignificant or be related to the pain and stiffness of the neck concurrent with the subcutaneous abscesses.

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[1] Jour. Cut. Dis., 1904, xxii, p. 77.

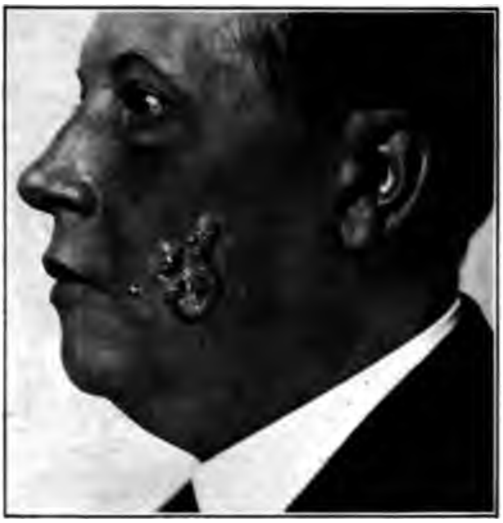
[2] Derm. Wochenschr., January 13, 1912; abstr. Brit. Jour. Derm., 1912, xxiv, p. 320.

[3] Jour. Cut. Dis., 1919, xxxvii, p. 740. Primary Actinomycosis of the Skin: report of a case involving the Hand.

[4] Med.-Chir. Trans., 1895, lxxviii, p. 21.

Etiology and Pathology.—More men than women are attacked, as a result of special exposure; a few of the affected have been occupied with cattle and horses; others, having carious teeth, may have been infected by accidents of contact or in the operations of dentistry. Cases are on record of transmission from man to man, from animals to man, and by traumatism, when inanimate objects were the media by which the fungus was introduced. The affection is communicable by inoculation. In most instances there have been submaxillary lesions and carious teeth. The general dispersion of the fungi in the atmosphere, water, and upon the soil is held to explain in large measure the occurrence of the disease in man. Beards of barley, bits of wood and stone, and vegetable fragments have been found in actinomycotic lesions.[1]

Fig. 369



Actinomycosis.

The pathological anatomy of actinomycosis is practically that described in Mycetoma. In the most commonly recognized type of the disease, the fungus is found in the yellowish or grayish masses discharged in clumps from the fistulous tracts and found also in sections of morbid tissue. Often there are seen fine interlacing threads or filaments radiating from a common center, some considerably projected above their fellows, many with a bulbous expression at the tip ("clubs"). The threads are slender, sinuous, often with dichotomous branches, and have an external sheath and protoplasmic medulla. The filaments grow rapidly and probably produce the disease. Coccus- and bacillus-like cells, regarded by Boström as spores, are also present.[1]

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[1] Hektoen, International Clinics, vol. iv, 9th series: The General Etiology of Actinomycosis (bibliography); Varney, Jour. Cut. Dis., 1909, xxvii, p. 234 (history of chewing kernels of wheat); Alworthy, Brit. Jour. Derm., 1909, xxi, p. 395 (patient chewed and ate uncooked oatmeal).

Diagnosis.—All supraclavicular and submaxillary lesions constituted of dark reddish tumors or swellings, subcutaneous in origin, call for scrutiny. Scrofuloderma is to be recognized by the general condition of the patient (actinomycosis may occur in vigorous young adults); by the absence of pronounced gumma and lymphoma (*gomme scrofuleuse*); and by the absence of the parasite. The occupation of the subject of the disease (as a farrier, stable-boy, or drover) may furnish a clue to the nature of the disorder in some cases. Care should always be taken, in making a diagnosis, to exclude cases of swellings discharging pus, practically limited to the skin immediately over the lower jaw, with sinuses leading to the bone beneath, which disorder is exclusively due to a carious fang of one of the lower central or canine teeth.

Treatment.—The treatment is both internal and local. Internally, potassium iodid has been found of great value in many cases. It is employed in the same manner as in syphilis, beginning with 5 or 10 grains, and increasing gradually until the toleration of the patient is reached. It should be given well diluted and after meals. Copper sulphate, as suggested by Bevan,[2] is used internally in the dosage of from $\frac{1}{4}$ to 1 grain (0.016 to 0.065) three times daily, and locally in a 1 per cent. solution as an irrigation or wet dressing. This method has proven of value in many cases. Surgical procedure, as indicated in the various cases, should be employed, as well as local antiseptic dressings and applications, which include the mercuric chlorid, Lugol's solution, and boric acid. Gautier has employed with success an electro-chemical method of treatment, by the use of platinum needles and injections of a 10 per cent. potassium iodid solution. Two needles are inserted, one connected with each pole of the battery, and a current of 50 milliampères is passed. A few drops of the iodin solution are injected during the flow of electricity, the patient being anesthetized. *X*-rays have been used during the last few years with beneficial results. In three cases treated by the author, treatment with a combination of *x*-rays and potassium iodid was followed by complete recovery. In a fourth and comparatively severe case involving the entire cheek, forming a boardy nodular tumor mass, arsphenamin and *x*-rays were used successfully. Copper sulphate, combined with *x*-rays, was successful in the case recorded by Zeisler.[3] Remer and Steinke[4] recorded successful treatment with *x*-rays alone. Jensen and Schery[5] employed methylene blue combined with x-rays. Radium has been employed with complete eradication of the disorder in six cases recorded by Heyerdahl[1] and in another by Hektoen (personal communication).

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[1] Hektoen, Phila. Monthly Med. Jour., November, 1899: The Classification and Nomenclature of the Ray Fungi; and Chicago Med. Recorder, June, 1900: The Ray Fungi and the Actinomycelial Processes.

[2] Jour. Amer. Med. Assoc., November 11, 1905, p. 1492.

[3] Jour. Cut. Dis., 1906, xxiv, p. 510.

[4] Jour. Cut. Dis., 1918, xxxvi, p. 187.

[5] Jour. Amer. Med. Assoc., 1920, lxxv, p. 1470; Actinomycosis Treated with Methylene Blue and Roentgen-ray.

Prognosis.—The prognosis is necessarily grave, on account of possible internal involvement. It was held until recently that the prognosis was favorable only in case of thorough and prompt removal of all diseased tissue. Schlange, at the Congress of German Surgeons held in 1890, called attention to the fact that of nearly two hundred patients under his observation, forty had remained well for more than two years, and in eighty the disease remained limited to the head and neck. After thirteen years of involvement, one patient at the date of the report was relieved. Untreated, the disease may eventually destroy life after years of exhaustive drain.

BLASTOMYCOSIS.[2]

Synonyms.—Blastomycetic dermatitis, Saccharomycosis hominis, Dermatitis blastomycotica. Ger., *Hefenmykose.*

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[1] Jour. Amer. Med. Assoc., 1919, lxxiii, p. 1928. Actinomycosis Treated with Radium.

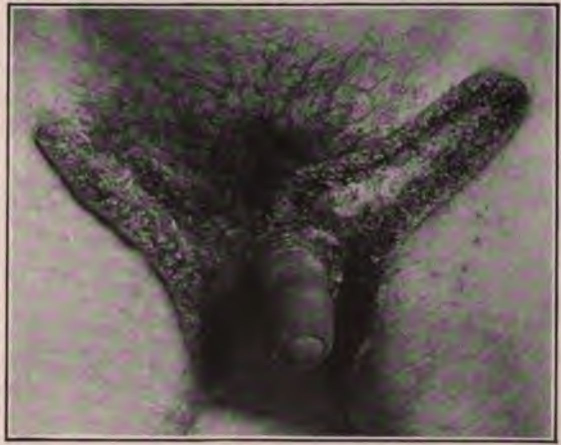
[2] BIBLIOGRAPHY: For a more detailed review of the clinical, histological, and bacteriological features of cutaneous blastomycosis, with 16 clinical and 25 histological and bacteriological illustrations, a brief summary of 13 cases, and bibliography, see report by Hyde and Montgomery, Jour. Amer. Med. Assoc., 1902, i, p. 1436. For a full consideration of experimental work, and animal inoculations with blastomycetes, see Buschke's complete monograph, Bibliotheca Medica, D. II, H. 10, 1902 (illustrations and bibliography); and monograph: "De la Blastomycose humaine," by Harter, Nancy, 1909.

Additional reports: Gilchrist, a case in a negro, with illustrations, review, and bibliography, Brit. Med. Jour., 1902, ii, p. 1321. Sheldon, report of a case, Jour. Amer. Med. Assoc., 1902, ii, p. 1356. F. H. Montgomery, a case of cutaneous blastomycosis followed by systemic tuberculosis, Jour. Cut. Dis., 1903, xxi, p. 19. Ormsby and Miller, a systemic case, with multiple cutaneous and subcutaneous lesions (a full report, with illustrations), ibid., p. 121. Sequeira, report of a case, Brit. Jour. Derm., 1903, xv, p. 121. Evans: "A case of Cutaneous Blastomycosis from Accidental Inoculation," Jour. Amer. Med. Assoc., 1903, p. 1772. Pusey, two cases presented to the Chicago Derm. Soc., Jour. Cut. Dis., 1903, xxi, p. 223. Fischkin, report of a case, Ill. Med. Jour., 1903, v, p. 472. Gilchrist, three cases, with 4 clinical illustrations and abstract of McGarrison's report of a case which occurred in a native of Northern India, Jour. Cut. Dis., 1904, xxii, p. 107. Hessler, Ind. Med. Jour., 1898, xvii, p. 48. Hyde, Hektoen and Bevan, Brit. Jour. Derm., 1899, xi, p. 261. Hektoen, Jour. Exper. Med., 1899, iv, Nos. 3 and 4. Owens, Eisendrath and Ready, Annals of Surg., 1899, xxx. Murphy-Hektoen, Jour. Amer. Med. Assoc., 1899, xxxiii, p. 1383. Anthony-Herzog, Jour. Cut. Dis., 1900, xviii, p. 1. Coates, Medicine, February, 1900. Brayton, Ind. Med. Jour., April, 1900, July, 1901, and Jour. Amer. Med. Assoc., February 1, 1902. Montgomery-Ricketts, Jour. Cut. Dis., 1901, xix, p. 26. Dyer, ibid., p. 14. Stelwagon, Amer. Jour. Med. Sc., 1901, cxxi, p. 178. Harris, ibid., p. 501. Ricketts, Jour. Med. Research, vi, No. 3. Bevan, Jour. Amer. Med. Assoc., November 11, 1905. Montgomery and Ormsby, Arch. Intern. Med., August, 1908: "Systemic Blastomycosis—Its Etiologic, Pathologic, and Clinical Features, as Established by a Critical Survey and Summary of 22 Cases, 7 Previously Unpublished; the Relation of Blastomycosis to Coccidoidal Granuloma." (In this review the authors give an abstract of twenty-two established and five probable cases of systemic blastomycosis. For a review of the entire subject the reader is referred to the original article.) Krost, Moes and Stober, a case of systemic blastomycosis, Jour. Amer. Med. Assoc., January 18, 1908. Shields, two cases of blastomycosis, one systemic and fatal, Jour. Cut. Dis., 1909, xxvii, p. 156. Fontaine, Haase and Mitchell, systemic blastomycosis, Arch. Intern. Med., 1909, iv, p. 101. Shepherd, systemic blastomycosis, a fatal case, Jour. Cut. Dis., 1911, xxix, p. 588. For other systemic cases, see Arch. Intern. Med., April 15, 1914, xiii, No. 4; reports by Stober, Krost, Moes, Lewison, Jackson, Myers, Boughton, Bechtel, LeCount, Eisenstaedt, Shaffner, Churchill, Clark, Riley, and Bartlett.

Definition.—Cutaneous blastomycosis is a chronic, inflammatory, infectious disease, characterized by the appearance upon the skin of a small papule or papulo-pustule, which becomes crusted and extends peripherally to form a sharply outlined, elevated, verrucous patch, situated upon a pus-infiltrated base, and presenting a characteristic abruptly sloping border, in which are seen minute, deeply seated abscesses. Blastomycetes are found in the sero-purulent contents of the abscesses, from which both budding and mycelial forms of the organism have been obtained in pure culture.

The invasion of the bodies of animals by blastomycetes had been studied before the disorder was recognized in the human family. In 1894 Busse published an account of a fatal case of pyemia, with subcutaneous abscesses and cutaneous manifestations, in which the pathogenic agent was a yeast. A few months earlier, Gilchrist had demonstrated before the American Dermatological Association microscopic sections containing budding organisms from a lesion which Duhring considered a scrofuloderm. Later communications from Busse and Buschke and from Gilchrist and Stokes have been followed by reports from a number of observers. Among those whose important work has furthered our knowledge of the disease should be mentioned Hyde, Montgomery (Frank Hugh), Hektoen, Bevan, Ricketts, and Wells. The records of about one hundred cases, published or unpublished, in which the nature of the disease has been demonstrated satisfactorily, are now available. The following description is based chiefly on the clinical, pathological, and bacteriological study of a large number of cases of both cutaneous and systemic blastomycosis.

FIG. 370

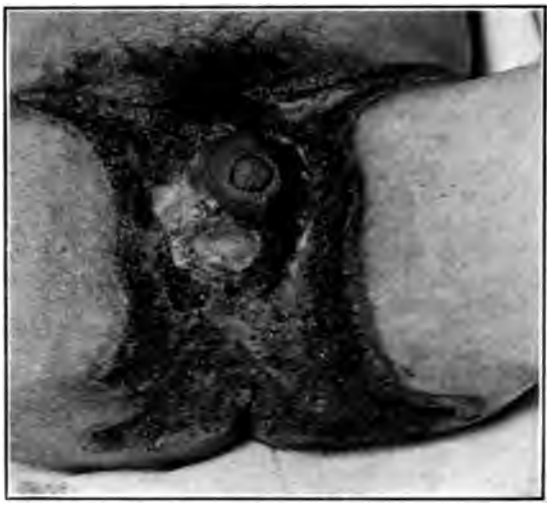


Blastomycosis.

Symptoms.—The disorder in the skin begins as a papule or papulo-pustule, which soon becomes covered with a crust. The lesion slowly enlarges peripherally in the form of an indolent, flat, wart-like or crusted papule. In the majority of all cases the lesions had existed a number of months and had attained a diameter of an inch or more before the patient applied for treatment.

In lesions that have attained the diameter of half an inch or more, the following characteristics are apparent: The patch is elevated from one-eighth to three-eighths of an inch above the surrounding skin; and the surface is covered by irregular, papilliform elevations, separated by clefts or fissures of varying depth, giving it a verrucous or cauliflower-like appearance. In the younger lesions and near the border of the older ones, especially of those which have been kept clean, the papillary projections are fine and the surface is fairly firm, dry, and wart-like. Portions of larger areas, and especially of those which have been untreated, are covered by more or less bulky and adherent crusts, on removal of which the papillary elevations are seen to be larger, lobulated, even subdivided, and bathed with a sero-purulent secretion. Some of these crust-covered projections are very vascular, a slight touch causing them to bleed. In exceptional instances, the area under a crust may present the appearance of an ordinary unhealthy ulcer, with exuberant granulations. In older lesions, the papillomatous surface may be replaced in part by a thick, elevated, scar-like formation, pinkish-white in color, irregular and often corded, but having a smooth, shining surface. The base of the active lesion is always soft and more or less infiltrated with sero-pus, which, on slight pressure, oozes between the papular elevations.

FIG. 371



Blastomycosis with destruction of scrotum.

The border of the area is one of the most characteristic features. It slopes more or less abruptly from the elevated, roughened surface to the normal skin, from which it is sharply defined. It is smooth, of a dark-red or purplish-red color, is from one-eighth to three-eighths of an inch wide, and on close inspection is seen to be beset with a large number of minute abscesses. Many of these abscesses are so small that they are not visible to the naked eye, but can be recognized with a lens magnifying from two to six diameters. Others vary in size up to that of a pinhead. Some are superficial, but many, especially the smaller ones, are deep-seated. When carefully punctured with a fine needle, these abscesses give exit to a small amount of thick, glairy mucus or muco-pus, the purulent character of the secretion increasing with the size of the pustule. From the smallest abscesses the amount of mucus expressed is sometimes so scanty that it can only be seen with the aid of a lens, yet it is from these minute abscesses that the organisms are best obtained in pure culture. Abscesses of the same sort occur also in other parts of the growth, and not infrequently on the thick, scar-like tissue described above; but in characteristic development they are best seen on the sloping border. The number of abscesses varies in different cases and in the same case at different times, depending somewhat upon the activity of the process.

The cutaneous lesions found in the systemic cases only occasionally correspond to the above description. They are found chiefly in the form of irregular, ragged, rather superficial ulcers, having a soft base, a granulating floor, and a purulent or sanguineo-purulent discharge, which often forms bulky crusts. These ulcers are usually preceded by subcutaneous abscesses, which gradually or rapidly extend to the surface and rupture externally. These subcutaneous nodules and abscesses are characteristic of the systemic cases, and often occur in successive crops.[1]

The course of the disease is irregular but essentially chronic. Usually months elapse before the original patch attains the diameter of an inch or more. It may remain indolent for months or even years, with irregular periods of activity and progress, but, as a rule, extension of the area is slow and continuous. In about half the cases the original patch of the disease has been followed in the course of weeks or months by one or more new lesions in adjacent or other regions of the body. In some instances, the clinical evidence of autoinfection has been very strong. The majority of the areas sooner or later attain the size of a silver dollar or of the palm, and some of them become much larger. As the disease extends at the periphery, healing frequently occurs in the central portion of the growth. In this manner large areas (in Anthony and Herzog's patient, the greater portion of the thigh and leg) may be involved in various stages of the process. Healing sometimes occurs spontaneously. Whether spontaneously or as the result of treatment, the first indication of healing is found in the gradual flattening and disappearance of the papillary projections, partly by absorption, partly by desiccation and exfoliation. At the same time the amount of secretion from the underlying base diminishes, and the whole patch assumes more of an ordinary verrucous appearance. In many instances, the papilliform surface is replaced temporarily by the hypertrophic scar-like tissue described above, which in turn gradually disappears and gives place to the characteristic cicatrix, which eventually becomes soft, supple, non-attached, pinkish-white, and, on the whole, very inconspicuous, though always sharply outlined from the surrounding skin. As a rule, the resulting deformity is very slight. In some instances, where destructive agents or scraping operations have been employed, the disappearance of the characteristic lesion is followed by an ordinary indolent ulcer, which heals with a thickened and somewhat deforming scar.

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[1] Arch. Intern. Med., August, 1908.

During the healing process, though the miliary abscesses decrease in number, careful search will reveal them even in scar-tissue that has become quite thin and soft. It is consequently not uncommon to see areas that apparently have healed become more or less covered again with active points or foci of disease. A single patch may thus present nearly all stages of the disorder, showing at the same time several of the following features: the advancing border; new-forming lesions on old scars; verrucous or cauliflower-like lesions in various stages of development or disappearance; a base in places dry and firm and in others soft and infiltrated with muco-pus; a scar-tissue, in part thick and irregular and in part smooth, soft, supple, and non-attached to the deeper tissues.

The regions involved are usually those most accessible to local infection, the disease occurring with greatest frequency on the face, hands, wrists, or forearms; but no portion of the body is exempt. The eyelids are a frequent seat of the disease, but the conjunctiva escapes, though ectropion, resulting from destruction of the lid, causes conjunctivitis and keratitis, due to exposure. Adenopathy has been noted in systemic cases only, though pus-infection of lesions may be followed by a transitory involvement of adjacent glands.

The subjective sensations of the disease vary greatly. As a rule, pain is slight or absent, except in areas which are acutely inflamed as a result of secondary infection.

The majority of patients have been in good general health, though some have suffered from other systemic disorders, which evidently bore no definite relation to the blastomycosis. Of the entire number recorded, one patient only died of generalized tuberculosis. In nineteen cases death occurred from systemic infection with blastomycetes, the organisms being demonstrated at the autopsy in the viscera, and in three cases in the blood. One of the nineteen patients (Montgomery-Walker case) remained in vigorous health for seven years after the appearance of cutaneous lesions, and then rapidly developed grave constitutional symptoms.[1]

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[1] Arch. Intern. Med., August, 1908 (discussion of clinical symptoms exhibited in systemic cases).

Etiology.—A local infection with the fungus peculiar to each case is the sole recognized cause of the disease. In one instance, a slight wound of the finger incurred at the autopsy of a case of systemic blastomycosis was followed in one week by the appearance at the site

Fig. 372

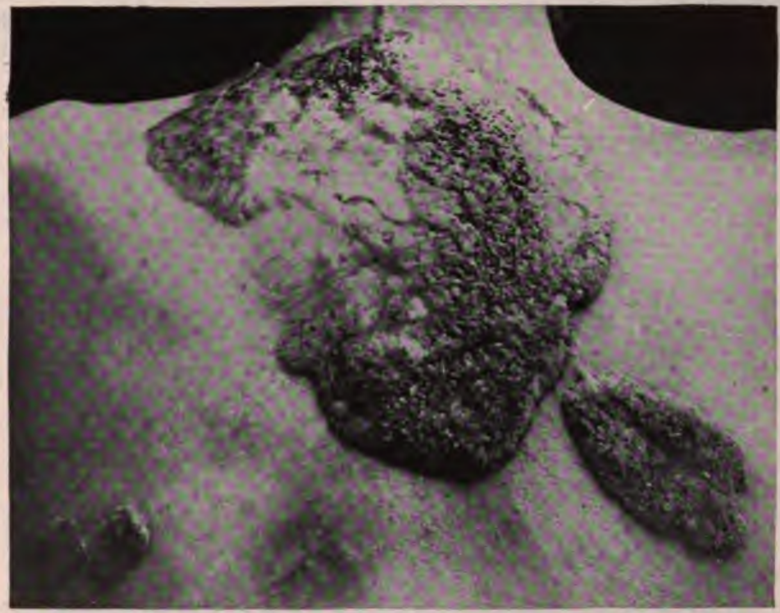


Fig. 373

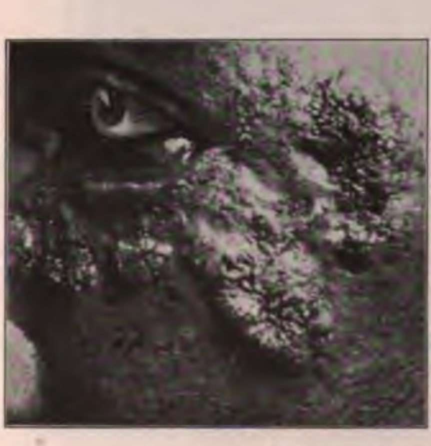
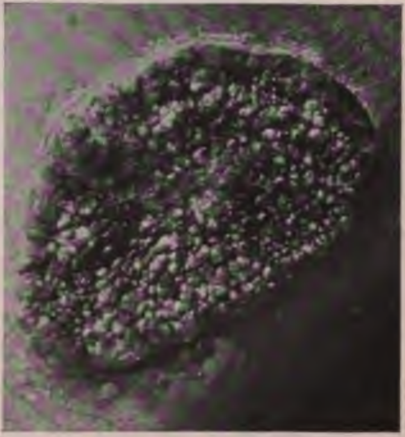


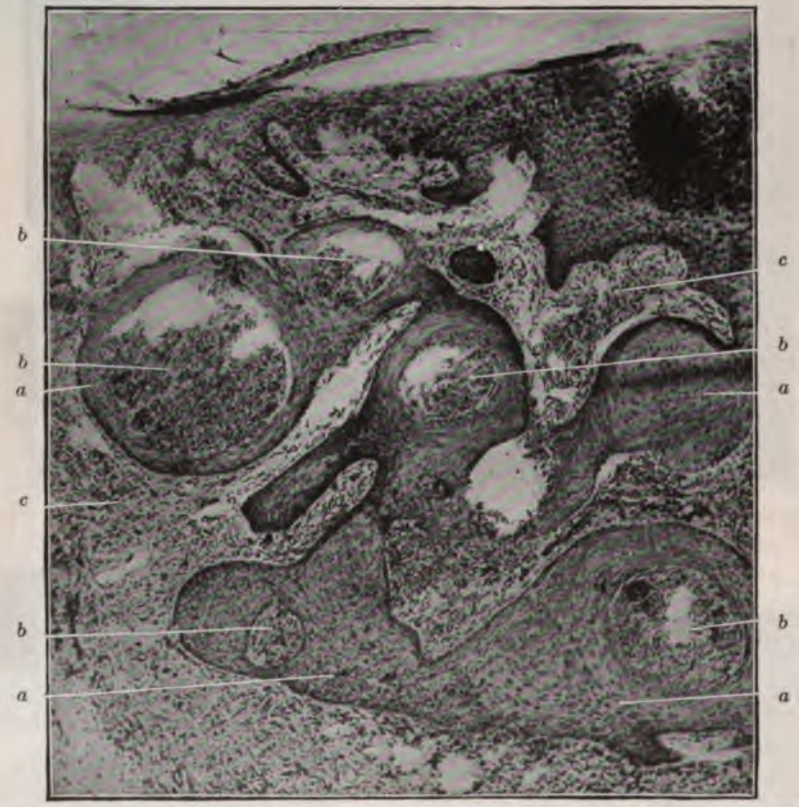
Fig. 374



Clinical types of cutaneous blastomycosis.

of the injury of a pustule, which refused to heal and later developed into a typical cutaneous lesion, in which budding organisms were demonstrated repeatedly. The infectious character of the disorder is demonstrated further by the successful inoculation of animals. In several instances there has been a history of trauma preceding infection. What other conditions favor the origin and development of the process has not been determined. Why certain yeasts and mould-fungi are pathogenic, while others are innocuous; how common in nature the pathogenic varieties are; and how they differ from the ordinary varieties, are unsolved problems.

FIG. 375

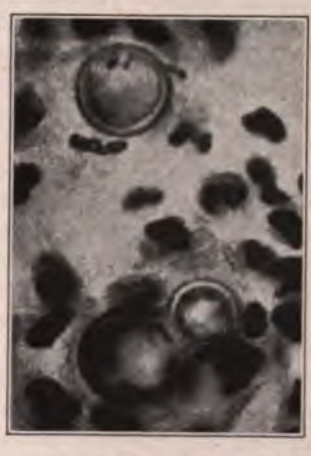


Vertical section from a typical lesion: *a*, hyperplasia of rete; *b*, abscesses in epithelium; *c*, infiltration of cutis. × 55.

No relation has been discovered between the disease and the sex, nativity, occupation, or habits of the individual affected. The fact that the majority of cases occur in men is due probably to their more frequent exposure to infection. About half the cases have occurred after the age of forty, with the majority of the remainder between the ages of twenty and forty. In one case observed by the author[1] the patient was seventy-six years of age. Recently, it has been observed in younger subjects, the youngest[2] being eight months old at the time the nature of the disease was established. No definite relation between blastomycosis and other local or systemic disease has been demonstrated. The possibility of blastomycotic infection being secondary to lesions of other disorders or to trauma is admitted; it is equally possible for the lesions of blastomycosis to be infected secondarily with tuberculosis or other disease.

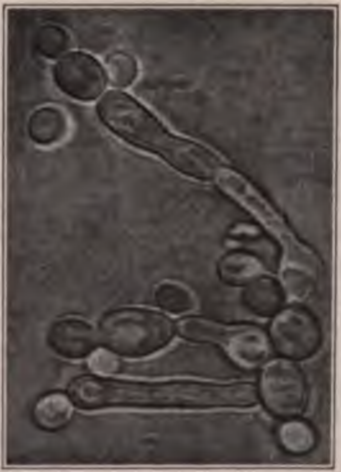
Pathology.—Histologically, the lesions resemble those of verrucous tuberculosis or of superficial epithelioma, yet differ from both. The surface, on which are seen irregular masses of débris, consisting of pus, blood, and epithelial cells and various bacteria, is marked by irregular papilliform projections, between which are corresponding depressions. The horny layer may be destroyed or it may extend in thickened masses between distorted papillæ.

Fig. 376



Blastomycosis of the skin. Budding organism in tissue. × 1200.

Fig. 377



Blastomycosis of the skin. Hanging drop. × 1200. From a photograph.

The rete is everywhere the seat of excessive hyperplasia, producing branching down-growths, varying greatly in size and shape. Polymorphonuclear leukocytes are scattered throughout the epithelium, both between and within the cells, and occur often in small collections, which are the beginning of miliary abscesses. These abscesses are characteristic of the process, and are found in all parts of the hyperplastic epithelium, in places breaking through to the surface. They contain leukocytes, nuclear fragments, detached epithelial cells, epithelial detritus, red blood-corpuscles, the organisms peculiar to the disease, and in many cases giant-cells. The epithelial cells surrounding the abscesses are flattened, but appear to take no active part in the process. The epithelium is separated from the corium in most places by a distinct layer of columnar cells, in which mitoses are seen occasionally. The rete-cells in general are large and appear swollen, the prickles being very conspicuous and the intercellular spaces increased. Premature cornification, more or less complete, occurs in scattered individual cells, in groups of cells, and occasionally in isolated epithelial whorls. Single giant-cells, surrounded by a few leukocytes, are sometimes seen in the epithelium at some distance from the corium.

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[1] Jour. Cut. Dis., 1908, xxvi, p. 235.

[2] Kessler, J. B., Jour. Amer. Med. Assoc., 1907, xlix, pp. 550-552.

The corium is the seat of subacute, chronic, and occasionally of acute inflammatory changes. Miliary abscesses occur, especially in acute lesions. The infiltration consists chiefly of leukocytes, endothelial cells, and plasma-cells, and is sometimes very dense. The number of mast-cells and giant-cells varies in different cases. Tubercle-like nodules are found in some instances. In several cases sections showed numerous hyalin bodies, which varied greatly in size, and occurred chiefly in plasma-, giant-, and new connective-tissue cells.

The appendages of the skin apparently play but a passive part in the process.

The blastomycetes are found in miliary abscesses, between the epithelial cells and in the corium, and are always surrounded by more or less evidence of inflammation. They are rarely found within the cells. The giant-cells, however, usually contain one or more of the parasites. The number present in the tissue varies greatly. In some cases a dozen or more can be seen in a single field of the microscope, while in others they are found with difficulty. They occur usually in pairs of unequal size, but also singly and in groups. They are readily seen in sections stained with hematoxylin and eosin or other common stains, but methylene blue is best for showing the different parts of the organism. The fungus is easily demonstrated by placing fresh or hardened sections, or pus, in a strong solution of potassium hydroxid, or in equal parts of liquor potassæ and glycerin; the organisms then appear as double-contoured, highly refractive bodies.

When well stained, the parasite is seen to be a round, oval, or slightly irregular body, having a well-defined, double-contoured, homogeneous capsule, and a finely or coarsely granular protoplasm, which is separated from the capsule by a clear space of varying width. The capsule resists the prolonged action of strong alkalies and acids. The protoplasm often contains a clear vacuole, which varies greatly in size in different bodies. Mature organisms have a diameter of from 7 to 20μ, though smaller and larger forms are seen occasionally.

Budding forms are seen in all stages of development. The capsules and clear space are pushed out apparently by the protoplasm to form oval buds, which grow to about one-half the size of the mother-cell before separating from the latter. Organisms in pairs of unequal size are more common than budding forms.

Mycelium has not been demonstrated in tissue or in the contents of the abscesses. In two cases organisms in the tissues were filled with small, globular bodies, which reacted to stains like spores, but no further development of these bodies could be seen.

In a systemic case, the author excised a small-bean-sized subcutaneous node, in which the epidermis was normal and the corium but slightly involved, the process being manifested chiefly in the subcutaneous tissue. The zones of infiltration were for the most part fairly well defined about dilated bloodvessels. The infiltration consisted of large numbers of the organism, also leukocytes, erythrocytes, connective-tissue, and plasma-, mast-, and giant-cells in varying numbers. In places there was a suggestion of tubercle-formation, in that the organisms, leukocytes, and red-blood cells were found chiefly in the center of the node and surrounded by giant-, connective-tissue, and plasma-cells. In this and other systemic cases the organisms were very numerous and larger than those found in most of the cutaneous cases.

The organism is obtained easily in pure culture from the minute, deep-seated abscesses in the borders of the cutaneous lesions. Cultures taken from the larger abscesses and from teased tissue are contaminated often with pus-cocci or other bacteria. The blastomycetes have been obtained repeatedly, however, in pure culture from pus-abscesses of considerable size, showing that the organisms are in themselves pus-producing. In cultural features, the organisms from different cases have varied considerably, and it is possible that they will have to be classed in distinct botanic groups. On the other hand, individual organisms have been shown to vary greatly with the media employed and with other circumstances of culture, and the different types seen may be various stages of development of a single variety of fungus.[1] The organisms grow rapidly on most ordinary media, and though by varying the media and other circumstances of growth a given organism may be made to assume a variety of appearances, in most instances the type is that of a mould-fungus, showing on agar or glucose-agar a white, fluffy growth, with aërial hyphæ, and on glycerin-agar a pasty growth, with numerous folds and depressions.

Under the microscope, cultures show budding organisms and mycelium, that may be fine, homogeneous, and branching, or coarser, more or less segmented, and with or without lateral conidia. The mycelium may contain few or many highly refractive bodies, varying in size, which probably are spores. Mingled with the mycelium of older cultures are round, oval or irregular, double-contoured bodies, varying greatly in size, and more or less filled with highly refractive, globular bodies. These globular bodies, like those seen in the coarser mycelium, behave in every way toward reagents as spores do, but in no case have they been observed to develop into mature organisms. Under certain conditions, blastomycetes may develop by sporulation. Young colonies and cultures on glucose-agar are made up of fine mycelium, with or without the presence of budding organisms. Older cultures and those on glycerin-agar show much coarser mycelium and a preponderance of the circular spore-containing bodies. A bit of old culture made up entirely of these round bodies, placed in a hanging drop of bouillon, develops in two or three days an abundant fine mycelium, in which the spore-like bodies are disseminated.

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[1] Hamburger, Jour. Infect. Dis., 1907, iv, p. 201.

Though in tissues and in the abscesses the organism develops by budding only, fresh cultures from the abscesses show fine mycelium more frequently than budding forms. Animals inoculated with cultures composed of mycelium have developed abscesses from which budding forms only were obtained.

Inoculation tests have been largely unsuccessful, but in several instances subcutaneous injection of pure cultures of the blastomycetes has resulted in the production of a local abscess, or of an inflammatory granulation tissue, from which the fungus could be recovered. Intraperitoneal injections with the organism from a systemic case reported by Montgomery[1] were unusually successful. By inoculating the skin of animals with pure cultures of blastomycetes, Buschke succeeded in producing tumors which resembled closely the lesions of cutaneous blastomycosis in man. The organisms in several cases have been inoculated in animals, with the production of tubercular-like nodules, or other inflammatory areas, in the lungs, kidneys, and other organs, from which the fungus has been recovered and cultivated.

Diagnosis.—Though many of the cutaneous lesions of blastomycosis resemble verrucous tuberculosis so closely that a definite diagnosis can be established only after a microscopic examination of the tissue or of the contents of minute abscesses, lesions showing the typical border set with abscesses described above are so characteristic and are present so frequently that a positive clinical diagnosis is possible in most cases.

The readiest means of confirming the diagnosis is to place the contents of one or more of the abscesses, or a bit of teased tissue, between a slide and cover-glass with a drop of a 20 or 30 per cent. solution of potassium hydroxid. If distinct budding organisms are found, which resist the action of the alkali after the tissue and pus-cells have largely disintegrated (a change requiring from ten minutes to one hour), the diagnosis is practically established, but should be verified further by obtaining cultures of the organism and by histological examination of the tissue.

Other disorders to be excluded by a consideration of their characteristic features are lupus vulgaris and other tuberculoses of the skin, the rare vegetating forms of syphilis, and protozoan infection, which, it is now believed, may be a variant of blastomycosis.

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[1] Jour. Cut. Dis., 1907, xxv, p. 393.

Treatment.—Complete excision of the diseased areas has been practised successfully in several cases, no recurrence having been reported. Curetting, employed in a number of instances, has not prevented a return of the disease.

Large doses of potassium iodid, first employed by Bevan, arrest the progress of the disease and produces a marked improvement in the cutaneous lesions. From 200 to 400 grains a day have been required in some instances before any effect on the morbid growth has been produced. In some of our cases and in several reported by others the disease disappeared under this treatment. However, in the majority of patients treated with large doses of potassium iodid, though healing takes place rapidly over the greater portion of the area involved, small patches, usually of the verrucous border, remain for indefinite periods; and on the discontinuance of the potassium iodid the disease reappears with as great activity as before. In several of our patients who improved rapidly under the treatment up to a given point, the few remaining verrucous areas and abscesses disappeared after a few exposures to the *x*-rays. Pusey, Fischkin, and others have had good results from the combined use of potassium iodid and the *x*-rays.

More recently, Bevan[1] recommended copper sulphate, in $\frac{1}{4}$ grain (0.016) doses, internally, with a 1 per cent. solution of the same as a local wet dressing. Arsphenamin was used successfully in three cases by Peterson,[2] and Harris[3] also obtained great improvement in one case. Simpson[4] records complete relief from symptoms with the use of radium. Gilchrist[5] obtained improvement by using an autogenous filtrate from living organisms.

For most lesions, cleansing and antiseptic lotions or dry dressings may be used with advantage.

Prognosis.—Complete excision when practised has terminated the disease. Under the iodin therapy, the condition improves so decidedly that with the aid of the *x*-rays, or other local treatment, the disease should be eradicated completely. Recurrences, however, are common, even after the last clinical evidence except scars has been removed.

The prognosis in systemic cases is grave; the mortality in recorded cases is about 90 per cent.

Phialophora Verrucosa.[6]—Under the above title C. G. Lane described the case of a patient with lesions situated on the buttock which clinically resembled verrucous tuberculosis, but which on histological examination was found to be indistinguishable from blastomycosis. A new fungus is described which presents both a sclerotic cell type and a beaded form in tissue. Mycelial formation occurred in cultures.

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[1] Jour. Amer. Med. Assoc., November 11, 1905.

[2] Trans. XVIIth Internat. Cong. of Med., London, 1913, Sec. xiii, Part ii, p. 79.

[3] Case demonstration, June, 1914, before the Chicago Dermatological Society.

[4] Jour. Amer. Med. Assoc., 1914, lxii, p. 844.

[5] Trans. XVIIth Internat. Cong. of Med. London, 1913, Section xiii, Part ii, p. 405.

[6] Jour. Cut. Dis., 1915, xxxiii, p. 840.

PROTOZOAN AND COCCIDOIDAL INFECTIONS.

Protozoan and coccidoidal[1] infections of the skin have been reported by Wernicke,[2] Rixford and Gilchrist,[3] Posadas, D. W. Montgomery,[4] Morrow,[5] and Ophuls and Moffit,[6] in which the cutaneous manifestations both clinically and histologically resembled very closely those of cutaneous blastomycosis. In the general symptoms, in the formation of subcutaneous abscesses, in the fatal termination, and in the larger size and greater number of organisms in the lesions, these cases correspond closely with those of systemic blastomycosis, with which they undoubtedly are closely allied. In cases of coccidoidal infection, the organism develops by endogenous spore-formation and never by budding; whereas in blastomycosis the only method of development of the organism in tissue is by budding. Though in at least two cases of the latter disease the organisms have contained what undoubtedly were endogenous spores, the development of these spores into mature bodies could not be demonstrated. The cultures obtained from cases of coccidoidal infection differ slightly from those obtained repeatedly in blastomycosis.

SPOROTRICHOSIS.[7]

In 1898, Schenck reported a case of "refractory subcutaneous abscesses caused by a fungus possibly related to sporotricha." The lesions here were multiple subcutaneous abscesses, occurring along the course of the lymphatics up the arms, and starting from a wound on the finger. The abscesses when opened were found to contain a gelatinous or aqueous fluid, and from this a sporothrix was isolated. In 1899, Brayton made a clinical report on an apparently similar case. In 1900, Hektoen and Perkins reported a similar case as to clinical symptoms and bacteriological findings to that recorded by Schenck. In 1903, De Beurmann published a case of the same type, under the title: "Multiple Subcutaneous Abscesses of Mycosic Origin." In this article he referred to the two proven American cases and the third clinical case. From 1906 on the disease was studied and reported on by a large number of observers, chief among whom were De Beurmann and Gougerot in France. In America, in addition to the early reports above noted, Hyde and Davis, Stelwagon, Pusey, Sutton, Hamburger, Wilder and McCullough, and others have reported cases.

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[1] For comparison between coccidoidal granuloma and systemic blastomycosis, see Arch. of Intern. Med., August, 1908.

[2] Quoted by Gilchrist.

[3] Johns Hopkins Hosp. Rep., 1896, vol. i (a full report of two cases and of the organisms, with illustrations).

[4] Brit. Jour. Derm., 1900, xii, p. 343 (bibliography), and Jour. Cut. Dis., 1903, xxi, p. 5 (a new case).

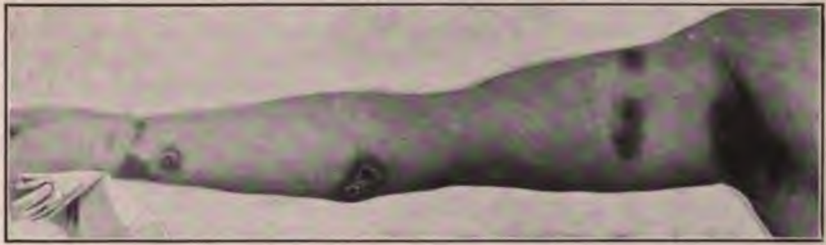
[5] Jour. Cut. Dis., 1903, xxi, p. 5.

[6] Philadelphia Med. Jour., June, 1900.

[7] Schenck, Johns Hopkins Hosp. Bull., 1898, p. 286. Brayton, Indiana Med. Jour., 1899, p. 272. Hektoen and Perkins, Jour. Exper. Med., 1900, p. 77. Adamson, Brit. Jour. Derm., 1908, xx, p. 296 (case report and *résumé* of the literature relating to sporotrichial infections of the skin, with bibliography of cases reported up to that date). Hyde and Davis, Jour. Cut. Dis., July, 1910, xxviii, p. 321 (a report of a case observed by them; notes on another case furnished them by Zurawski; a short account of a group of probable cases of a similar nature occurring in North Dakota in 1904; a complete histological and bacteriological study, and a comparison of sporotrichosis with mycotic lymphangitis occurring in horses; and very full bibliography, covering the extensive reports of the French writers and others). Stelwagon, ibid., p. 352. Pusey, ibid., p. 352. Page, Frothingham and Paige, Jour. Amer. Med. Research, August, 1910, xxiii, No. 1, New Series No. 1, pp. 137–150 (a question of identity between the Sporotrichium Beurmannii and Sporotrichium Schenckii). Sutton, Jour. Amer. Med. Assoc., September 17, 1910, p. 1000; and ibid., December 24, 1910, p. 2213. Hamburger, ibid., 1912, lix, p. 1590 (sporotrichosis in man, with a summary of the cases reported in the United States, and a consideration of the clinical varieties and the important factors in the differential diagnosis). Chipman, Jour. Cut. Dis., 1912, xxx, p. 339 (a *résumé* of the views of De Beurmann and Gougerot on the subject of sporotrichosis). Wilder and McCullough, Jour. Amer. Med. Assoc., 1914, lxii, p. 1156: Sporotrichosis of the Eye (pertinent bibliography). Dermody and McMartin, ibid., 1914, lxiii, p. 1028: A Case of Sporotrichosis. Sutton, ibid., 1914, lxiii, p. 1153: Sporotrichosis in the Mississippi Basin (report on five new cases). Meyer, Jour. Amer. Med. Assoc., 1915, lxv, p. 597; "Sporotrichosis;" chronology and geographical distribution of 82 American cases. Blaisdell, Jour. Cut. Dis., 1917, xxxv, p. 452; report of first New England case. Little, Brit. Jour. Derm., 1918, xxx, p. 174; "Sporotrichosis." Eisenstaedt, Jour. Amer. Med. Assoc., 1918, lxxi, p. 726; "Sporotrichosis resembling tuberculosis cutis." Ormsby, Jour. Cut. Dis., 1919, xxxvii, p. 424; sporotrichosis in child five years of age. Davis, Jour. Infec. Dis., 1919, xxv, p. 124; "Experimental sporotrichosis."

Definition.—The disease is characterized by the formation of indolent cutaneous and subcutaneous nodules, abscesses, and ulcers, not infrequently beginning in an infected wound, usually on the hand.

Fig. 378



Sporotrichosis. (Eisenstaedt.)

Symptoms.—The disorder may be initiated by a wound on the finger or other part of the hand, presenting various characteristics. It may be exhibited as a small nodule, or a superficial, inflammatory, open, discharging lesion. Occasionally, it may resemble to a certain extent the initial lesion of lues. At other times it may be insignificant and clear up before other manifestations occur. At a subsequent date, of varying length up to three months, nodules begin to appear upon the arm, along the course of the lymphatics. The lesions gradually enlarge, are painful, and may be seen varying in size from that of a pea to that of a mandarin orange. The smaller ones may be only recognized by palpation, when they are felt as hard, resistant growths in the subcutaneous tissue. After two or three months they become adherent to the skin, assume a reddish or violaceous tint, soften and break through the skin, discharging a grayish-yellow, homogeneous pus. The lesions then may gradually undergo involution or increase in size by new fistulous openings occurring around the original lesion. Incision into the lesions increases their growth rather than promotes resolution. Occasionally, the ulcer enlarges and takes on a fungus, papillomatous growth, producing a lesion closely resembling that seen in tuberculosis verrucosa cutis. The course of the lesions is indolent but progressive, and they may become disseminated irregularly over various parts of the limbs and trunk.

On account of the clinical resemblance to syphilis and tuberculosis, types have been described in France as syphiloid and tuberculoid. These types have been assumed in different stages in many cases. One case is reported as resembling lupus verrucosus, the lesions in this type occurring primarily on the face; another is reported in which a large abscess occurred, from which the sporothrix was isolated. The lymphatic glands are not attacked, and the general health of the patient is not interfered with by the disease, although tuberculosis has been recorded in association with the disorder and the sporothrix has been discovered in the sputum in a case where the lungs were found to be normal. In addition to the skin, the mucous membranes and bones have been invaded. In the former case, granulating ulcers have been described, which, however, have not produced the loss of tissue seen in ulcers similarly situated in cases of tuberculosis or syphilis. Sporotrichial gummata have been discovered beneath the periosteum, and a tibial periostitis has also been described. The muscular tissue has also been attacked with abscesses of a similar character.

Etiology and Pathology.—The disease is due to infection with the sporothrix fungus. As a rule, the organisms cannot be seen in sections nor in fresh pus from lesions in man. They are usually isolated either by culture or by animal inoculation. In sections of tissue removed from infected animals they are readily demonstrated.

The organism is strictly aërobic and grows on ordinary media, but with greater profusion on media containing sugar. Cultures exhibit in the course of several days a delicate fringe, consisting of fine radiating lines, projecting at right angles from the margin of the growth. Later the surface of the growth becomes corrugated and the radiations at the margin more prominent. Their color early is dull gray or nearly white. Different appearances are noted, varying with the culture media employed. In hanging-drop preparations, branched mycelium, with oval spores attached singly by a pedicle or arranged in clusters or bouquet form, are seen. The mycelium is divided at irregular intervals by delicate septa, between which small, refractile granules occur. The spores vary in size and shape, measuring from 3 to 5μ in length and from 2 to 3μ in width. In sections from infected animals oval and oblong spores of varying size are noted.

That the organism is saprophytic is demonstrated by its growth upon various animal structures (caterpillars, fleas, and larvæ) and upon vegetables (lettuce leaves), and the suggestion is made that infection may take place through contact with unclean vegetables, or, from the same source, through the alimentary canal.

Sporotrichosis has been produced in a number of animals, including the horse, the cat, the mouse, the monkey, the rat, and the guinea-pig. In the latter animal the result of inoculation experiments is uncertain, this animal being least susceptible. The rat, on the other hand, is highly susceptible. The disorder has been thoroughly studied in the rat by De Beurmann, and in this animal implication of the viscera has occurred, a condition which has not been seen in man, but the pathological changes noted were similar in other respects. Sporotrichosis has been observed as a spontaneous disease in the horse, the mule, and the dog.

Several species of the sporothrix have been described in various parts of the world. Castellani and Chalmers[1] name eight, as follows: *Sp. Schencki, Sp. Beurmanni, Sp. Dori, Sp. Gougeroti, Sp. Jeanselmei, Sp. indicum, Sp. asteroïdes,* and *Sp. lesnei.* It appears that the organisms described by Schenck, Hektoen, and Perkins in America, De Beurmann in France, and Castellani in Ceylon, present sufficient differences to class them as separate species, though Castellani states that Sp. indicum, described by himself, may be only a variety of Sp. Beurmanni.

The microscopical findings in tissue have been described by De Beurmann and Gougerot as occurring in three types: a lymphoconnective-tissue, or syphiloid type; an epithelioid with giant-cells, or tuberculoid type; and a polynuclear, or ecthymaform type. In Dr. Hyde's case the histology corresponded with the tuberculoid type. In this case the lesions were indolent, subcutaneous nodules and abscesses. There occurred a marked cellular infiltration, consisting of round-, plasma-, epithelioid-, and giant-cells. In addition there were polynuclear leukocytes, but this section was taken from the edge of an ulcer. While the cellular composition was that of tuberculosis, the arrangement of the cells was not typical of the tubercle of that disorder.

Diagnosis.—The characteristic features outlined above, which include multiple cutaneous and subcutaneous nodules and ulcers of indolent character, the nodules being firm early, later softening and discharging a characteristic viscid, grayish-yellow pus; the arrangement of the lesions in a line up the arm; a past history of a primary infection wound; and the lack of interference with the patient's health, are all important points in diagnosis. While the lesions simulate fairly closely those seen in syphilis, tuberculosis, and occasionally blastomycosis, a differentiation can be made. In case of doubt, cultures should be made or animal-inoculation experiments performed. In the latter case pus or tissue inoculated into the perineal cavity of a rat produces a characteristic orchitis, in which the organism may be found abundantly. This corresponds with our previous experience with blastomycosis, which produces similar lesions in guinea-pigs. The cultural diagnostic method is preferable, owing to its more rapid development.

Treatment.—Potassium iodid is specific. With the administration of this drug the lesions rapidly clear up and the disease is entirely eradicated. The ulcers should be dressed in accordance with the principles laid down in surgery.

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[1] Tropical Medicine, 2d ed., 1913, p. 831.

Prognosis.—The prognosis is good with the employment of proper treatment. It is not unlikely that cases of this type have been considered to be syphilis and been treated with potassium iodid, and on the patient making prompt recovery the diagnosis has been apparently verified.

NOCARDIOSIS CUTIS.

Under the above title Guy[1] reported the case of a patient suffering with a disease that presented the clinical symptoms of sporotrichosis, in which a microörganism was found that differed from recognized forms of sporothrix. In tissue this microörganism occurred in parallel and tangled threads in and about dilated lymphatic vessels and spaces. In culture the growth consisted of masses of tangled, branching threads, bearing small granules. Holman classed the microorganism under the Actinomycetaceæ species nocardia. Potassium iodid was successfully employed in the treatment of the case.

DISEASES DUE TO ANIMAL PARASITES.

The human skin may be attacked by animal parasites, which (*a*) habitually exist upon or within the integument, securing their nutrition in these situations; (*b*) exist upon the clothing, furniture, or other articles of environment of the body, attacking the latter only when in search of food; (*c*) are brought accidentally into contact with the human body and attack it when irritated or alarmed, without seeking nutriment; or (*d*) infest the vascular channels or viscera of the body and involve the skin only when approaching the surface as an accident of the human invasion.

PEDICULOSIS.

Synonyms.—Phthriasis, Morbus pediculosis, Lousiness; Fr., *Phtiriase; Maladie pédiculaire;* Ger., *Läusesucht.*

Lice belong to the order *Rhynchota,* subdivision *Parasitæ,* family *Pediculidæ.* They are apterous, provided with two eyes, and have an oral appendage capable of inflicting wounds and producing suction. There are three varieties that infest the human body and these are found in different situations: the head, the pubic region, and the trunk in general. Although each of these parasites is usually found in a particular region, exceptions occur.

The head-louse is usually of a grayish color, but differs slightly with the hue of the hair on the part which it frequents. Its head presents indistinctly the outline of a trefoil, and is provided with two hairy antennæ (each of five articulations) and two eyes. Its thorax is

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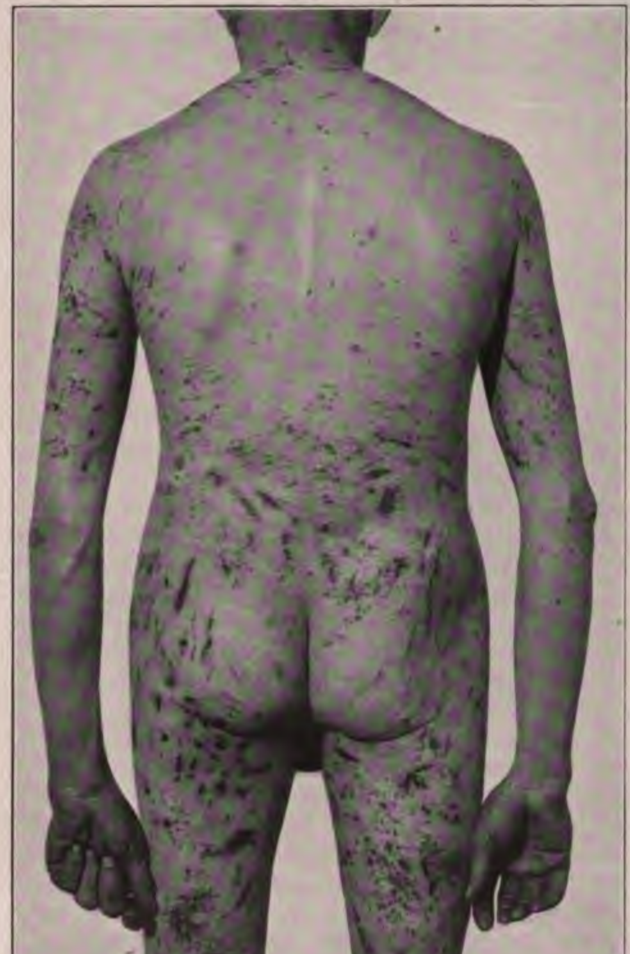
[1] Archiv. of Derm. and Syph., 1920, ii, p. 137: Nocardiosis Cutis Resembling Sporotrichosis.

relatively narrow, with six tracheal stigmata and three hairy legs on either side, each leg being provided with tarsal hooklets. The abdomen is divided into seven segments defined by blackish indentations on either side. The males are fewer and smaller than the females, and they present upon the dorsum an ano-genital orifice and a large conoidal

Fig. 379



Pediculosis.

penis and testes. The females are provided with ovaries and with an anal aperture in the terminal abdominal segment. They measure from 1.2 to 3.2 mm. in length and are about one-half as broad as they are long. The body-louse closely resembles the head-louse, being, however, larger in size (1.2 to 4.2 mm. in length), the female also larger than the males. The thorax is separated from the abdomen, which is hairy, yellowish at the margins and provided with eight segments. The eyes are black and very prominent in both sexes. In color the insects vary from a dirty-white to a light-grayish hue, except when distended with blood, when they are dull-reddish or purplish, and more indolent in their movements.

The pubic louse is smaller than either of the above, measuring from 1 to 2.0 mm. in length and being nearly as broad as it is long. Its head is also attached more closely to its thorax, having a shape which is compared to that of a violin. The thorax is not distinctly separated from the abdomen, and of the six stout legs with which the louse is provided, the second and third pairs are conspicuously powerful and armed with relatively large hooks at the tarsal extremity. The resemblance of the latter to the claws of a crab has given to this parasite the common name of "crab-louse." The lateral abdominal indentations are much less distinct than in the other varieties; and the blackish marginal marks of body-lice and head-lice are here scarcely apparent. The abdomen is less elongated, having a more rounded contour. The pubic louse is provided on its lateral borders with eight short, conical feet, terminating in bristles. It is also distinguished from the others of its family by the length of its anal bristles and by the peculiar shield-shaped carapace which covers nearly one-half of the dorsum.

The ova or nits are whitish or grayish bodies of oval contour, which are glued to the hairs by a cylindriform sheath of chitin, which completely encases the hair, the free, bulbous end projecting toward the free end of the hair. In the scalp they are deposited in series along the hair, being gradually removed from the scalp with the growth of the hair. The youngest are those nearest the scalp. The ova of *Pediculus corporis* are deposited in the clothing as well as on the lanugo and other hairs.[1] In soldiers in the late war ova of this parasite were commonly found on the hairs in the axillæ, pubic and perianal regions, as well as on the trunk. The productivity of these parasites is enormous. Kaposi stated that a single female could produce fifty eggs in six days, and thus in eight weeks have a progeny of five thousand lice. Nuttall[2] computed the total number of eggs deposited by a single parasite of the body type to be two hundred and seventy-five to three hundred, of which seventy were deposited daily.

Pediculosis Capitis.—(*Parasite, the head-louse*).—Head-lice, while commonly found in the scalp, are sometimes seen in the hairs of the beard in old men and, rarely, in the pubic[1] or other regions. While occurring on all portions of the scalp, the parasites elect the region about the occiput. They are seen more frequently in children, particularly girls, but are occasionally seen in adults. The ova, or nits, are usually abundant upon the hairs of the infested head and can be discovered with comparative ease. In early cases they are attached to the hair near the scalp. In older cases they may extend some distance along the shaft of the hair. The lesions seen are those induced by scratching. The disorder is accompanied by itching of varying grades, in some cases severe. Excoriated and crust-covered areas are most common and along with these pustules are seen, together with denuded areas which are oozing with serum, pus or blood. In rare instances bullæ are formed. The crusts may be few or abundant and consist chiefly of blood and epithelial débris, or there may be an admixture of pus and sebaceous matter. In the dispensary class of patients when the disorder is neglected, the hairs may become matted together with purulent material, among which crusts and other débris are found, the whole of which undergoes decomposition, producing a nauseating odor. In aggravated cases the posterior cervical glands are enlarged in varying degrees by secondary infection. Multiple abscesses may occur in neglected cases.[2] While the disorder is seen, as a rule in public charities and in individuals who come from the lowest social grades of the population, where the surroundings are unhygienic, the disorder is not infrequently seen in children and adults of high social grade.

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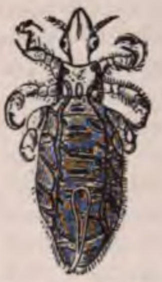
[1] For further information concerning the occurrence of ova on hairs, see Bulliard, Annales, 1916-17, Tome vi, p. 501; abstr., Brit. Jour. Derm., 1918, xxx, p. 188; many references in the abstract.

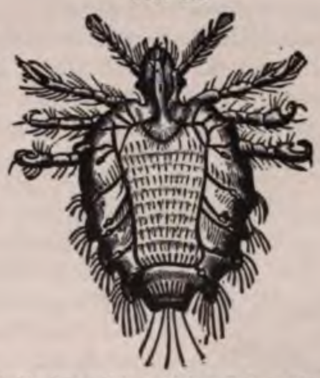
[2] Parasitology, x, p. 69.

FIG. 380



Pediculus capitis—male. (After Küchenmeister.)

FIG. 381



Pediculus pubis. (After Schmarda.)

Diagnosis.—The diagnosis of pediculosis capitis is important, as this disorder, when occurring in individuals whose personal hygiene is all that it should be, is rarely considered. A marked itching disorder occurring in the back of the scalp should always suggest a careful examination for the parasites or their ova. Occasionally impetiginous lesions about the nares and lips, especially of young girls, are significant of pediculosis of the occipital region, the lesions being due to picking and scratching the face under an impulse to relieve the itching sensations of the scalp.

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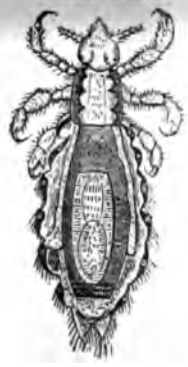
[1] Nicolas and Massia, Annales, January, 1916; Pediculus capitis found in pubic region; no parasites present in scalp.

[2] Schamberg, Jour. Cut. Dis., 1910, xxviii, p. 686.

Treatment.—For the destruction of the parasites petroleum is ordinarily employed. This is applied thoroughly over the scalp, where it should be well rubbed in and the hairs thoroughly saturated. The whole may then be covered with a dressing or a towel and allowed to remain for twelve hours or over night. At the end of this time the scalp is shampooed with tincture of sapo mollis, or other good soap, and hot water. This treatment ordinarily destroys the parasites and most, if not all, of the ova. The latter may be subsequently removed by the application of dilute acetic acid or a 50 per cent. solution of alcohol applied daily for several days. Combing with a fine comb is also of service in removing both the living ova and dead shells. Other remedies which may replace petroleum are staphysagria, one dram (4.) of the powdered seeds to the ounce (30.) of vaseline. This may also be used in the form of a decoction. Tincture of cocculus indicus, phenol in oil or water, and mercurials in ointment or solution. The management of the local lesions depends upon their character and severity. Ordinarily sulphur, salicylic acid, or ammoniated mercury in a strength of 3 per cent. in petrolatum is all that will be required.

Pediculosis Corporis. — (*Pediculosis vestimenti, Phthiriasis, Parasite,* the *body-louse*).— The parasites in this variety are usually found in the seams of the underclothing, or they remain in other situations when coarse woollen or flannel shirts or similar material are worn. Unless the individual is unusually infested they are rarely seen on the skin, though they do at times remain in hairy regions. The rule is that the skin is visited merely for obtaining food. It is occasionally difficult, particularly when the organisms are few, positively to demonstrate their presence when a recent change of underclothing has been made. In all cases a careful search should be made on the hairs for ova, which may solve the problem. The lesions on the skin, aside from a puncture wound produced by the parasite, are all secondary. At the point of puncture a circumscribed hemorrhage occurs. This hemorrhagic spot is level with the skin and is characteristic of the disorder. Nuttall[1] says the lesion produced by the louse bite is a papule. The next most

FIG. 382



Pediculus corporis—female. (After Küchenmeister.)

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[1] Parasitology, x, p. 69.

characteristic lesions in these cases are excoriations. These are usually linear, occasionally circumscribed, vary in depth and length, and radiate irregularly from the primary lesion. Crusts, usually composed of dried blood, rarely of serum or pus, occurring over injured follicles are characteristic. Occasionally transitory wheals, small papules and localized infections complicate the process. In cases of long duration pigmentation follows. In neglected cases, particularly of old men in low-grade lodging houses or similar situations, pediculosis may assume a rather more serious aspect. The skin, through long-continued neglect of the disorder, becomes harsh, dry, scale-covered and deeply pigmented, and in various places presents areas of dermatitis, crusts, even rupioid in character, and other signs of local infection. This type has sometimes been termed "vagabond's disease." As sequels in some cases, furuncles, abscesses, carbuncles and ulcerations occur. Pyodermias,[1] some serious, were common among soldiers during the late war.

In rare instances, particularly when large numbers of the parasites feed on the individual, systemic reaction occurs, indicated by fever, headache and other signs of such disturbance. Jamieson[2] ascribed this to cutaneous irritation alone. Payne[3] accounts for the systemic reaction through a poisoning produced, rather than irritation, by the parasites. Moore[4] reported similar reactions which he stated appeared to be an intoxication of the system produced by the introduction of a toxin at the time the parasites are obtaining food.

Diagnosis.—The lesions in this disorder are usually limited to the trunk. Excoriations over the neck, about the shoulders, the buttocks and external surface of the thighs are suggestive. The presence of itching at irregular times, the rather characteristic small, dark-red, crusted points which occur in the lines of excoriation are important. Scabies is readily ruled out by the absence of the gallery, and of vesicles, and the difference in location of the lesions. Pruritus hiemalis and essential pruritus are both to be differentiated.

Treatment.—The clothing should be boiled when possible or placed in an oven and subjected to a temperature sufficient to destroy the parasites and their ova, 160° to 175° F. At the same time measures should be employed to destroy any ova that may be attached to the hairs of the body. The irritation on the skin is to be treated according to its severity. As a rule, the itching and secondary dermatitis disappear quickly upon the withdrawal of the exciting cause. Warm alkaline baths, followed by a soothing ointment or lotion, such as are recommended in the treatment of acute eczema, may be employed. Dusting the clothing and body surface with sulphur is of immediate service and may also be employed as a prophylactic measure.

Pediculosis Pubis.—(*Crab-louse, Parasite, the Pubic Louse;* Fr., *Morpion*).—In this variety the genital region is chiefly involved, though in exceptional cases all the hairy portions of the body may be attacked, as well as the eyebrows and lashes. The pubic louse is much less active than others and is ordinarily found firmly attached to the hair with its head buried in the hair follicle. A moderate degree of force is required for its removal from this situation, and it not infrequently will slide along the entire length of the hair, which is held by the claw of the parasite. In rare instances it is seen moving over the surface. Interspersed throughout the area the ova are found, the detection of which is a valuable diagnostic point. Pubic lice are usually acquired during the contacts incidental to the sexual act and hence are more frequently encountered among adults. The lesions are those induced by the wounds inflicted by the parasites and by scratching, though these are rarely severe. In the major portion of cases the chief symptom is itching, which may be severe. Not infrequently a secondary dermatitis is seen which has been induced by local treatment employed by the patient. In certain cases of pediculosis pubis a peculiar pigmentation occurs, described as *maculæ cerulæ* (Fr., *Taches ombrées, Taches bleuâtres*) which are pea- to small coin-size, grayish stains found on the chest, abdomen, thighs and upper arms, especially in individuals having a thin, light colored skin. These spots have a steel-gray tint, do not disappear under pressure, and are believed to be due to the action of the parasite. Duguet[1] after inoculations with the juice of crushed pediculi, believed he demonstrated that the lesions arise from pigment occurring in the body of the insect. Montgomery[2] believes that this lesion is the direct consequence of the action of the saliva of the insect on the blood of the host, the saliva acting on the hemoglobin, turning it green. As this occurs in the small hemorrhages induced by the bite of the insect, the lesion is situated within the skin and presents a bluish color through the horny layer. Jamieson[3] considers this pathognomonic of the presence of pediculosis pubis. Occasionally the parasites attack the eyelashes (*Phthiriasis palpebrarum*), especially in children, where they produce a condition resembling blepharitis ciliaris or eczema.[4] The borders of the lids present what appear to be small dark crusts, which on careful examination prove to be pediculi closely attached to the ciliary margin, with their heads deeply imbedded in the hair follicles.

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[1] *Cf.* article by Knowles, Jour. Amer. Med. Assoc., 1918, lxxi, p. 1312 and others.

[2] Brit. Jour. Derm., 1889, i, p. 321.

[3] Ibid., 1890, ii, p. 209.

[4] Jour. Amer. Med. Assoc., 1918, lxxi, p. 1481.

Diagnosis.—Pediculosis pubis is to be differentiated from genital pruritus and eczema. In most instances the location of the disorder is different. Eczema is not apt to be so limited to the pubic region and is characterized by more marked and definite changes in the skin. In any case of local pruritus a careful search should be made for the parasites. They are most commonly found firmly attached to the hair with their heads buried in the follicle, and are frequently barely perceptible. At other times the major portion of the parasite is rather plainly visible immediately beside a hair. The presence of ova is important and these are readily found in most cases. The reddish excrement of the parasites, together with scratch marks and excoriated papules are usually present.

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[1] Annales, 1880, p. 544.

[2] Canadian Prac. and Review, 1916, xli, p. 231; Pediculosis pubis.

[3] Loc. cit.

[4] Winfield, Jour. Cut. Dis., 1889, vii, p. 332; recording 4 cases.

Treatment.—The commonly used mercurial ointment is efficient but disagreeable and sometimes produces dermatitis. A 1 to 500 solution of mercuric chlorid is cleaner and efficient. An ointment constituted as follows is valuable and clean: Salicylic acid, 2; benzoic acid, 4; petrolatum, 30. This is to be applied every day for several days. Montgomery[1] suggests creolin 10, vaseline 90, and states that this ointment destroys the insect, is antipruritic, and assists in clearing up a secondary dermatitis. The ova may be removed by the application of a dilute solution of acetic acid, or a solution of biborate of sodium. Either one of the latter dissolves the chitinous substance which holds the ova onto the hair. Should there be a dermatitis, this is to be treated according to its degree, as suggested in the section devoted to eczema.

CIMEX LECTULARIUS.

Synonyms.—Bugs, Bedbugs, Acanthia lectularia. Fr., *Punaise des lits;* Ger., *Bettwanze.*

Strictly speaking, the bedbug is not a parasite of man, but finds its congenial habitat in the bed, bedding, and bed-covering, and the walls and floors of apartments occupied by persons of both sexes and all ages. It may find a host in certain of the lower animals, such as the guinea-pig. It infests also furniture, including chairs, sofas, and the cushions of seats occupied in public vehicles and hotels. From the cracks, crevices, seams, folds, or other protected points where it has found lodgment, it emerges, usually at night, for the purpose of securing its nutriment in the blood of its victims.

This insect is an apterous member of the order *Cimicidæ*, and has a rusty or reddish color, this differing slightly according as it is or is not distended with blood. It is provided with a blunt-pointed head, broadly attached to the thorax; two long, slender antennæ; and a three-jointed haustellum, capable of projection and retraction, beneath the head. There are three pairs of long, slender legs, by which it is enabled to accomplish rapid movements. The abdomen is broad, flattened, and oval in shape, with nine segments. The parasite emits a disgusting odor, which is much more distinct when it is crushed.

The wound inflicted by this bug is accomplished with or without the consciousness of its victim, who in the former case is made aware of a transitory prick or sting. Soon after, decidedly itching, burning, or stinging sensations are experienced, and the wound becomes the seat of an urticarial wheal. The lesion, when examined soon after the infliction of the wound, is seen to be a small- pea- to bean-sized, elevated and circumscribed "button" or papulo-tubercle, either uniformly red or presenting a whitish centre surrounded by a zone of hyperemia. After the lesion has begun to subside and lose its acute features, which may not occur for several hours if it be irritated by rubbing or scratching, a minute, reddish punctum may be seen marking the original site of the wound.

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[1] Loc. cit.

The lesions are usually multiple, even when but a single parasite has been present, the insect obtaining its nutriment from several distinct points upon the surface. In this way, at times, its course upon the integument may be traced for a short distance. In cases in which the pests are numerous, as in filthy dwellings, prisons, ships, and barracks, and when infants have been attacked, the resulting eruption is often greatly masked by the scratching and resulting excoriations of the skin-surface. Associated lesions are vesicles, pustules, crusts, purpuric blotches, and even infiltrations.

Diagnosis.—The diagnosis is a matter of importance, and upon it may hang a professional reputation. Physicians are often consulted respecting these lesions by patients who believe themselves to be suffering from "hives," "humors," exanthemata, and even from syphilis. The insect attacks the parts of the body to which access is easy as the patient sits or reclines; namely, the legs about the ankles, the buttocks, the shoulders and the neck. The eruption is not to be confounded with urticaria *ab ingestis*, which is more apt to be symmetrical in disposition.

Treatment.—The eruption is best relieved by the topical application of spirit of camphor, alcohol, weak carbolated lotions, or solutions of boric acid, 1 drachm to the pint (4. to 500.). Untreated, it disappears spontaneously when the source of the disorder is removed. The most effective treatment is by prophylaxis, with soap, corrosive-sublimate solutions in alcohol, and hot water employed over all accessories of the dwelling-house inhabited by the insects. Once discovered to be present, infested furniture should be scrubbed and all its crevices treated with a strong solution of corrosive sublimate in water and the bed-clothing be immersed in boiling water.

GRAIN ITCH.[1]

Synonyms.—Acarodermatitis urticarioides (Schamberg), Straw itch, Barley itch.

This disorder was first described in this country by Schamberg,[2] in 1901. It has since been recognized, in addition to Philadelphia and the region about it, in Indiana, Ohio, and other States.

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[1] Schamberg, Jour. Cut. Dis., 1910, xxviii, p. 67 (In this article, from which the description used in this text was largely taken, the author reviews his early work, describes the disease in detail, and gives a critical and thorough review of the literature). Rawles, Indiana State Med. Jour., August, 1909: Straw Itch. Wills, Brit. Jour. Derm., 1909, xxi, p. 249: Barley Itch (The causative organism was not discovered by this author in his cases). Goldberger and Schamberg, Public Health Reports, U. S. Public Health and Marine Hospital Service, vol. xxiv, No. 28, p. 1909: Epidemic of an Urticarioid Dermatitis Due to a Small Mite (*Pediculoides Ventricosus*) in the Straw of Mattresses.

[2] Phila. Med. Jour., July 6, 1901.

Symptoms.—The disorder is characterized by an eruption consisting of wheals, exhibiting a central pinpoint-sized vesicle. Schamberg states that this is the peculiar lesion of the disease, and is so characteristic as to suggest immediately the affection. The contents of the vesicle are clear for a brief period, and then become lactescent or distinctly puriform, constituting a well-marked pustule. Instead of wheals, the efflorescence may consist of slightly elevated, erythemato-urticarial spots, or papulo-urticarial lesions.

The lesions vary in size from that of a lentil to that of the finger-nail, and are rounded, oval, or irregular in shape. They are edematous, like the wheals of ordinary urticaria, and not infrequently elevated from 1 to 2 mm. above the level of the surrounding skin. The color is usually a rose tint: only rarely is seen the pinkish-white, anemic area which occurs in ordinary urticaria. The central vesicle or pustule, which is usually minute, may in certain cases reach the diameter of 3 mm. In such cases the large vesicles situated upon an erythemato-urticarial base present a strong resemblance to the lesions of smallpox. In many patients, through excoriation, the lesions are greatly altered, and those of traumatic dermatitis supplement the ones above described.

The eruption is usually profuse, and involves the neck, chest, abdomen, and back, and in lesser degree the arms and legs, the greater number of lesions being on the trunk. The face is often free, although at times scattered lesions may be present. The hands and feet are nearly always exempt. The extent of the eruption and the size of the individual lesions are apt to bear an inverse relation to each other. In rare instances, the eruption may resemble that of multiform erythema. It is accompanied by intense itching, which is worse at night and seriously interferes with sleep. As in most other itching disorders, the trauma inflicted by the nails induces secondary pyogenic infection and other lesions of this type. The disease may be accompanied by mild constitutional symptoms, the temperature being elevated from one-half to three or four degrees. A certain degree of malaise and anorexia may also be present, but in many patients no systemic symptoms are exhibited. A moderate leukocytosis and eosinophilia were found by Schamberg in a large number of cases examined.

Etiology.—The disorder is produced by a mite termed *Pediculoides ventricosus*, which is found most commonly in straw. The disease occurs in epidemic form, usually between the months of May and October. Several members of a household or groups of laborers or sailors may be simultaneously attacked. It may occur at any age, the necessary factor being contact with grain or straw infected with the mite. Schamberg and Goldberger, in their original investigations in Philadelphia, traced the infection to the use of new straw mattresses. It was eventually discovered that all of these had been made by one manufacturer, who used straw from a certain county. The organisms were found in the dust and siftings from the straw.

Pathology. The histopathology described by Schamberg, taken from a vesico-pustule situated on an urticarial base, is as follows:

Over the region of the vesico-pustule there is circumscribed elevation of the epidermis. The stratum corneum is reduced to one or two layers, and the stratum granulosum is absent. Two layers of fusiform rete-cells extend from the lateral portion of the roof of the whole vesicle toward the middle, but are lost over the central area. No vertical cleavage was noted in the center suggestive of a puncture by a parasite, nor was any parasitic appendage noted anywhere in the skin. Beneath the altered rete, large numbers of leukocytes were noted. The bloodvessels and lymph spaces showed much dilatation. In the corium a dense, circumscribed, cellular infiltration was present. This infiltration consisted of round cells, a considerable number of polymorphonuclear leukocytes, and in the middle and deeper portion of the corium large numbers of mast-cells. There were marked dilatation of the vessels and edema. Fragmentation of the leukocytic nuclei was noted to a limited degree. The pathological changes were essentially those of an urticarial lesion.

Pediculoides ventricosus belongs to the class of *Arachnida*, order *Acarina*, family *Tarsonemidæ*, and genus *Pediculoides*. The parasite is described as follows: " Normal and free rostrum; legs of the fourth pair in the female of the same dimensions as the others, terminated by two nails and a sucker; legs of the first pair in the female normal, terminated by a claw; legs of the fourth pair in the male but little different in dimensions from the others, terminated by a claw. Dorsal plaque segmented. Abdomen of the gravid female swells up into a sort of sphere. The last anatomical characteristic is the most striking of those possessed by the genus. The female is ovoviviparous and gives birth to small octopods, which can be fecundated immediately. It is possible that the pediculoides parasites upon man do not belong to a single species." These parasites are said to be of economical importance, for the reason that they are inimical to the grain insects, and therefore favor the preservation of that cereal. They are parasitic upon the larvæ of soft-bodied insects, which are usually grain-destroying parasites.

Unlike *Acarus scabiei*, the mite *Pediculoides* does not burrow into the skin. Microscopic examination of vesicles and pustules has demonstrated the absence of the mite or any part of it beneath the surface of the skin. Schamberg states that it is probable that in the process of extracting liquid nourishment from the skin the mite synchronously injects an irritating substance, which gives rise to the lesion.

Diagnosis.—The affection is to be differentiated from urticaria, chicken-pox, and scabies. The longer duration of the individual lesions, the accompanying vesiculation, the constitutional disturbance, the greater persistence of the attack, and the occurrence of the disorder in groups of persons, distinguish it from urticaria. From chicken-pox it is distinguished by its occurrence largely in adults, the presence of intense itching, and the difference in the character of the vesicles. From scabies it is differentiated by the location of the lesions and by their individual characteristics. The absence of lesions on the hands and the presence of urticarial and erythemato-urticarial lesions also distinguish it from scabies. A history of contact with straw is of great diagnostic value.

Treatment.—As *Pediculoides* does not burrow beneath the skin, it is a comparatively simple matter to rid the patient of the parasite. Frequent warm baths are recommended, and Schamberg suggests the use of the following ointment: beta-naphtol, grains 30 (2.); sulphur precipitate, grains 40 (2.66); adipis benzoate, 1 ounce (30.). In order to prevent reinfection of the patient, his clothing should be disinfected either by boiling or careful fumigation with sulphur or formaldehyd. When the source of the parasite is a straw mattress, the latter may be rendered free of the mites by exposure in a closed chamber to steam, sulphur fumes, or formaldehyd. Ordinarily, the itching will subside in from twelve to thirty-six hours, and the eruption will disappear within a week or ten days. When the cause of the eruption has not been recognized and the use of the mattress has continued, patients have been known to suffer for periods varying from three to seven weeks.

Prognosis.—The disease tends to spontaneous disappearance in time. It may be prolonged by reinfection, but when recognized and appropriate treatment instituted recovery takes place in from one to two weeks.

COPRA ITCH.[1]

This disorder is described as occurring in Ceylon, in people working in copra mills, and presents an eruption resembling scabies, but without burrows or cunicula. The lesions are found on the hands, arms, and legs, sometimes on the whole body except the face. They are fairly numerous, itch extremely, and may consist of papules, papulo-pustules, pustules, crusts, and excoriations. The eruption begins on the hands and spreads to the arms, legs, and trunk. There is no tendency to clear spontaneously as long as the patient continues at his work.

Etiology.—The disorder is produced by *Tyroglyphus longior* Gerv., var. *Castellanii* (Hirst). This mite produces a dermatitis in the same manner as *Pediculoides ventricosus*. A large number of the organisms may be found in the dust of the copra mills. Castellani suggests for treatment beta-naphtol ointment, 5 to 10 per cent. The organism described by Hirst[2] resembles *Acarus scabiei* in a general way.

BROWN-TAIL MOTH.[3]

A dermatitis induced by the caterpillar of the brown-tail moth (*Euproctus cysorrhœa*) was first described and recorded in this country by J. C. White[1] in 1901. Subsequent reports were made by Meek,[2] Towle,[3] Spear,[4] Tyzzer,[5] Potter,[6] and others.

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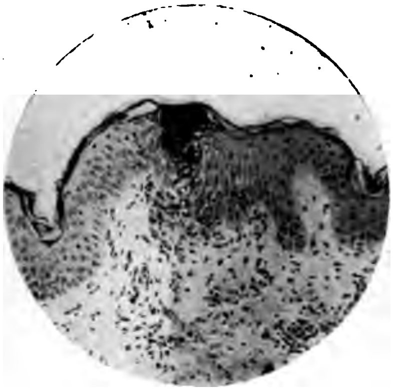
[1] Castellani, Brit. Jour. Derm., 1913, xxv, p. 19.

[2] Ibid., p. 21.

[3] This moth is said to have been imported from Holland and has proved very destructive to vegetation in the New England States and Canada. *Cf.* Fernald and Kirkland, "The Brown-tail Moth," Bulletin, Massachusetts State Board of Agriculture, 1903. 52d Annual Re. Massachusetts State Board of Agriculture, 1904, Nature Leaflet No. 26. Ibid., 1908, April 2.

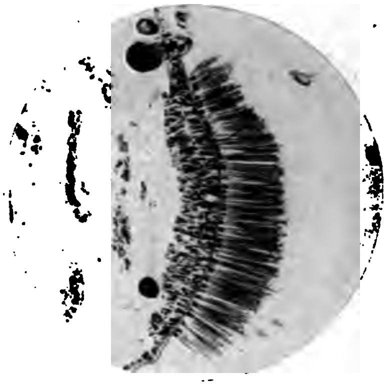
Symptoms.—The disorder begins with itching, shortly after the irritating hairs have been deposited upon the skin. Erythematous macules follow which are succeeded by firm, pea-sized, moderately elevated, urticarial lesions. The color of the lesions disappears under diascopic pressure. The lesions may be few and discrete or numerous. The individual lesion lasts from a few hours to a few days and the eruption from several days to several weeks. The areas commonly affected are the face, neck, arms, chest and back, though any area may become involved. In severe cases confluence of lesions occurs forming variously-sized patches. Intense itching may be present. In rare instances eczema follows.

Fig. 383



Section showing inflammatory changes in the corium twenty-four hours after the skin had been rubbed lightly with a small brown-tail caterpillar. (Tyzzer.)

Fig. 384



Section of a caterpillar showing the nettling hairs as they are developed upon its skin. (Tyzzer.)

Etiology and Pathology.—The disorder is seen most commonly in the months of May and June when the caterpillars are maturing. The hairs are found on the moth, its caterpillar and cocoons. They are commonly deposited on the skin directly by the caterpillar, but may be indirectly transferred through clothing upon which they have lodged or through handling the cocoons. According to Tyzzer the "nettling" hairs are of the form of straight, tapering, needle-pointed shafts, possessing three rows of recurrent barbs and are from 0.07 to 0.02 mm. in length and slender. They possess a chemical substance which acts as an irritant in the skin. When deposited upon the skin they penetrate the epidermis and at times the corium and through the chemical and mechanical irritative effects the inflammatory reaction is induced. Necrosis occurs about the hairs and a fluid exudate is found in the epidermis.

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[1] Boston Med. and Surg. Jour., 1901, cxliv, p. 599

[2] Ibid., p. 657.

[3] Ibid., 1905, clii, p. 74.

[4] Ibid., p. 12.

[5] Jour. Med. Research, 1907, n. s. xi, p. 43 and Trans. Internat. Derm. Cong., 1908, i, p. 169.

[6] Jour. Amer. Med. Assoc., 1909, liii, p. 1463.

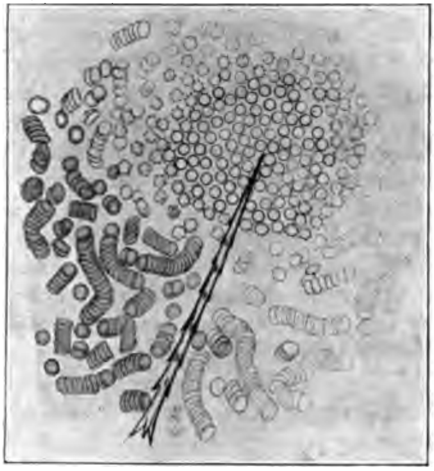
Treatment.—Soothing lotions such as calamine or zinc oxid and aqua calcis are advised. At times the affection is resistant to treatment.

PULEX IRRITANS.

Synonyms.—Flea. Fr., *Puce commune;* Ger., *Gemeiner floh.*

Fleas exist in all parts of the world, the pest which especially attacks man living in dwellings as well as out of doors, infesting crevices in floors, walls, and even the clothing. Fleas belong to the order of *Diptera,* family *Aphaniptera.* The males are 2 to 2.5 mm. in length, and the females about 4 mm. The insects are reddish-brown in hue, and are provided with a head having no bristles; these, however, well projected backward, mark its thoracic and abdominal rings. The eggs are white and barrel-shaped, and are deposited in the crevices inhabited by the mature insects as well as on the clothing of men. The legless larvæ, having fourteen segments, are changed to pupæ in eleven days.

FIG. 385



A sketch showing the effect of the nettling hairs of the brown-tail moth upon mammalian red-blood corpuscles. (Tyzzer.)

The flea which specially attacks man has a laterally compressed body; an oral haustellum; serrated, soft mandibles; a tongue sheathed in an inferior labium; and a pair of labial, four-jointed palpi. Each of the triple segments of the thorax bears a pair of five-jointed, double-clawed legs. The nympha is enfolded in a cocoon, but only the mature insects prey upon man.

According to Geber, the insect injects an irritating fluid into the skin at the moment of attack. It attacks the human skin for the purpose of securing its blood-food. The lesion it produces is a hemorrhagic punctum, followed by a transitory hyperemia and a hemorrhagic exudation, which may persist for a few hours. When the pests are numerous, the resulting distress is considerable. The wounds are recognized by a central punctum, with an areola of reddish or purplish hue. This central punctum or point distinguishes the wounds produced by the insect from macules of simple erythema; but care should be taken, when fever is present, to exclude the symptomatic erythemata.

The site of the wound may become an urticarial wheal, and other urticarial lesions are produced in sensitive skins by the effort to relieve the itching produced by the bites of the insects. In severe cases the nervous system is harassed to a grave point by the resulting discomfort. The petechial character of the cutaneous lesions is often well marked.

It is important to identify flea-bites on the skins of patients suspected of being the victims of typhus or other fevers; and of filthy subjects who are affected with other skin-disorders.

Mixed cases of flea-bites with wounds produced by bugs and lice are often seen in the lowest classes applying to public charities for relief; and the deeply pigmented skins they exhibit, often with purpuric lesions distributed over the lower extremities, and commingled with syphilitic eruptions, are in the highest degree confusing. The practitioner should always be on his guard in pronouncing on these cases, especially if the purpuric blotches occur in the cachectic or in those suffering from other diseases than those of the skin.

The fleas of the lower animals occasionally are transferred to the human body, but rarely thrive on such a host.

Treatment.—The treatment of flea-bites is by alkaline and tarry lotions, and other local applications suggested in the treatment of urticaria. Stelwagon[1] recommends the wearing of bags filled with gum-camphor or pyrethrum beneath the clothing. Sulphur has been employed similarly.

IXODES.

Synonyms.—Wood-tick. Fr., *Pou de bois*, *Tique*; Ger., *Holzbock*.

Several species of tick are recognized, such as the *Rhipicephalus annulatus* (cattle-tick), *Amblyomma americanus*, *Ixodes unipunctatus*, and *Ixodes ricinus* (wood-beetle), the last named being more common in Europe. In America wood-ticks are found in wooded districts, especially where pine- and fir-trees are growing. The female occasionally attacks the human skin by thrusting into it her beak, armed on either side with a maxillo-labial projection having recurved hooklets. The mandibles also present similar obstacles to the forcible extraction of the head. After suction of the blood from the wound, the body of the tick swells to the size of that of a pea or small bean, and may remain for several days in the same position. At such times the parasite may be mistaken for a small, pedunculated tumor. Forcible attempts at extraction of the intruder are liable to detach the mandibles from the body, and thus leave them in the skin, to become the source of future irritation and even disagreeable inflammatory symptoms in the site of the punctured wound. On applying over the tick a drop of spirit of turpentine or benzin, the head is spontaneously retracted and the body falls from its position. Soldiers on the plains of the United States accomplish the same end with the juice of tobacco. The sensation produced at the moment of the insertion of the beak of the insect is said to be so trifling as often to pass unnoticed.

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[1] Dis. of the Skin, 7th ed., p. 1187.

Other insects may persistently or occasionally attack the human skin: gnats, mosquitoes (*Culicidæ*), midges (*chironomidæ*), bees (*Apes mellificæ*), and wasps (*Vespidæ*). They produce by their bites or stings various cutaneous lesions, including urticarial wheals, papules, ecchymoses, and in rare cases even ecchymomata. The lesions produced by the midge, like those of the mosquito, are seen on the face, the hands, and exposed parts; though, when numerous and voracious, these insects will penetrate the clothing for the purpose of obtaining blood. In America severe eruptive lesions are often seen on the faces and extremities of infants and children exposed during the night to the incursions of these marauders. It has recently been shown by Stokes[1] that black flies induce a characteristic and annoying eruption when they attack the skin. The early lesion is a hemorrhagic spot at the site of puncture which is followed by a papulo-vesicle situated upon an urticarial base and which requires several days for its complete development. A weeping stage follows after which the lesion undergoes resolution leaving a minute scar. Marked itching and burning accompany the eruption. The lesions are usually discrete, but when numerous, confluence occurs forming edematous, weeping and crusted areas. The parts attacked are the cheeks, the region about the eyes, the neck along the hair line and the areas back of the ears. Regional adenopathy is usually present.

Treatment.—For the bite or sting, aqua ammoniæ or spirit of camphor may be employed. For subsequent lesions, soothing lotions, such as the zinc oxid and lime-water or calamine lotion, are useful.

SCABIES.

Synonyms.—"The Itch." Fr., *Gale;* Ger., *Krätze.*

Definition.—Scabies is a contagious disorder of the skin produced by *Acarus scabiei*, characterized by intense itching, which occurs chiefly at night, and by multiform lesions.

Symptoms.—The characteristic lesion of the disorder is the cuniculus burrow or gallery produced by the itch mite, as it invades the skin. With this lesion vesicles are commonly developed. After this various lesions occur, such as papules, pustules and crusts, excoriations, and pigmentations. The number and extent of the lesions varies within wide limits. The areas involved are the hands, especially between the fingers, about the wrists, the anterior folds of the axillæ, the breasts in women, the buttocks in children, and the genital region, particularly the penis and scrotum, in men. In severe cases the distribution may become more or less generalized, but even in these the major portion of the lesions are between the shoulders and knees. The face is rarely attacked, and then chiefly in nursing infants. Itching is a prominent feature of all cases and occurs chiefly at night after retiring. Many patients have no discomfort during the day, but are tormented at night.

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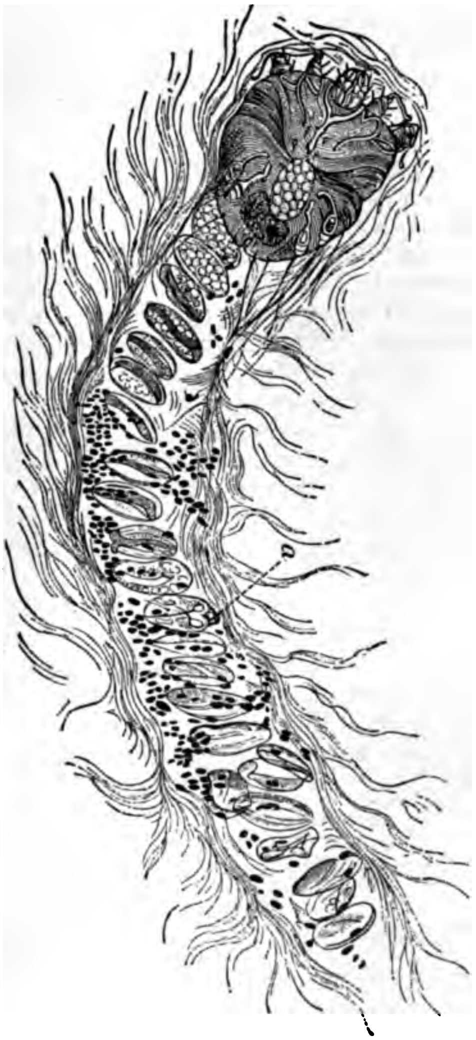
[1] Jour. Cut. Dis., 1914, xxxiii, pp. 751 and 830; a clinical, pathological and experimental study of the lesions produced by the bite of the black fly Simulium venustum.

The disease usually develops gradually, beginning in the areas of predilection and by multiplication of lesions progresses until checked by treatment. When fully developed the disease presents the following: Galleries or burrows of varying length and coloration, vesicles of different sizes situated near the galleries, inflammatory papules, some of which may be pea-size, but are commonly smaller, pustules and crusts in different stages of evolution, excoriations, areas of secondary dermatitis, and possible pigmentations and relics of lesions which have undergone complete

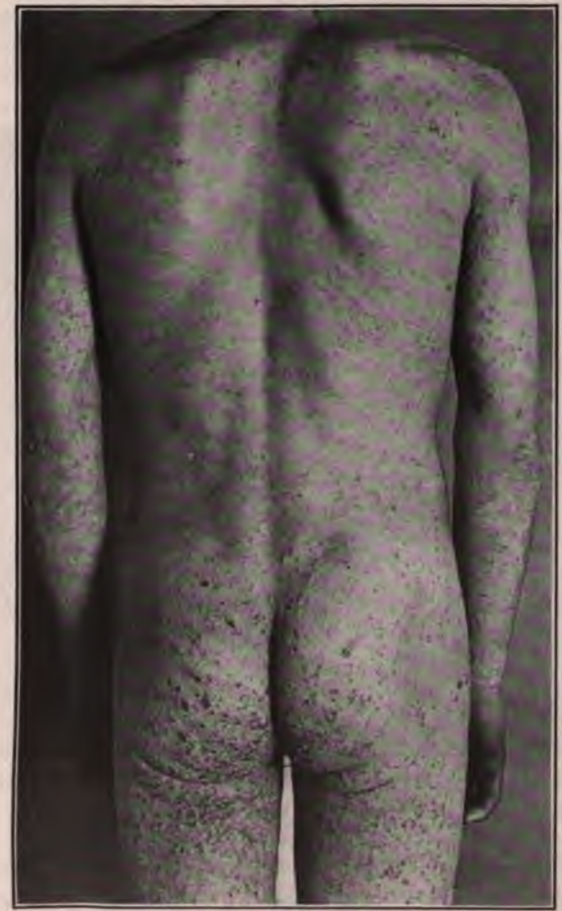
FIG. 386



Acarian furrow, from the lumbar region. The female acarus is visible at the terminal extremity of the furrow, with ventral surface exposed, and containing a mature ovum; two ova, next her, have been laid during the day; the third exhibits traces of the embryo; the twelfth exhibits a mature larva (*a*); twelve empty shells are also seen; between these feces are represented by black points. (After Kaposi.)

involution. The average case seen in private practice presents, as a rule, only a moderate number of lesions consisting of the galleries, a few vesicles and small crusted areas. The cuniculus or burrow of the itch mite is the tunnel produced in the stratum corneum by the parasite. It is a whitish or yellowish, slightly arciform lesion with regular parallel borders, covered with dark points which, according to Darier and others, represent points of exit of newly hatched organisms in which dirt has collected. It may be curved, angular, or tortuous, and resembles a beaded, dotted, yellowish or blackish thread. The acarus is found at the deeper extremity, which is indicated by a whitish or yellowish, shining, point. The head of the gallery where the parasite has entered the skin is usually whitish and more elevated than the tail. At times the entire cuniculus forms

Fig. 387

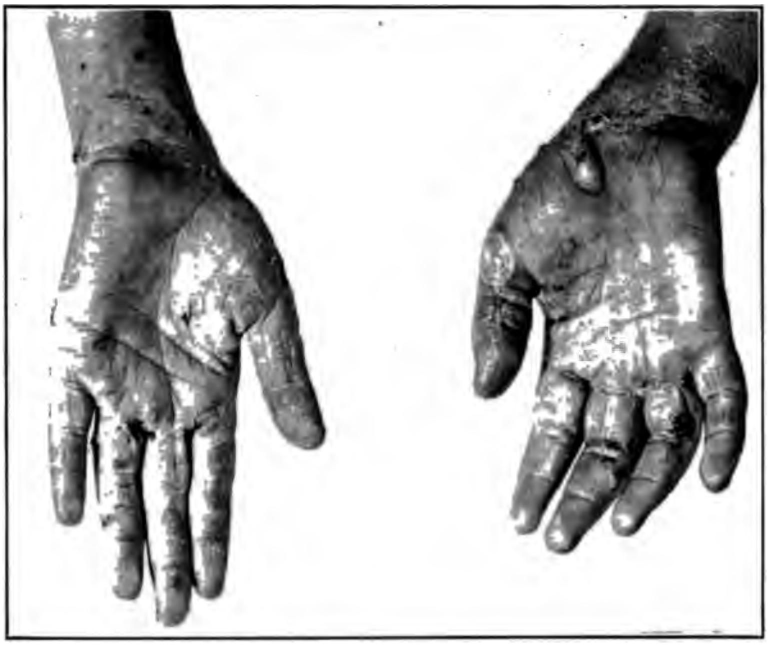


Scabies showing the distribution of the lesion. (Fordyce and MacKee.)

an elevated ridge rather than a thread-like depression, with white dots running along the summit. As a rule, a vesicle is situated at one end of the cuniculus, but occasionally the latter may be covered by a vesicle, pustule, or bulla.

Variations in the galleries are numerous. In infants and young children they may be long and well-developed and markedly vesicular. On the hands of laborers they are small, poorly developed, and difficult to find. The hands were comparatively free in soldiers during the late war, even when the disease was well developed. The gallery may be dark colored and plainly visible, or lighter and difficult to discover.

FIG. 388



Scabies showing the vesicles and galleries on the hand. (Fordyce and MacKee.)

In many cases comparatively large inflammatory papules occur which may remain for a long period, even with treatment. These are seen usually on the scrotum, penis, and about the elbows, but occur elsewhere. In these the acarus may be demonstrated comparatively deeply in the lesion. Itching occurs with as much intensity in cases having a few lesions as in those with an extensive eruption. The vesicles are rather characteristic of the disease, may be difficult to demonstrate, but usually one or several may be found even in mild cases. In children or in persons with delicate skin large vesicles occur and occasionally bullæ are noted. In exceptional instances vesicles may be so well developed on the hands as to simulate dysidrosis.

The areas of involvement are those most accessible to the hands, thus demonstrating one reason of the special localization. The diamond-shaped area on the back between the shoulders is thus rarely involved, though lesions do occur here.

The disease is unaccompanied by constitutional symptoms; albuminuria occurs occasionally as a result of the disease. It may also occur following certain methods of treatment.[1] Kolmer[2] found mild leukocytosis and an increase in the number of eosinophiles in 18 cases of scabies. Schamberg and Strickler[3] found eosinophilia in 80 per cent. of 47 cases examined. Eosinophilia has been demonstrated by most observers.

Animal Scabies.—(*Sarcoptes scabiei equi, ovis, capræ, cameli, aucheniæ, suis, caneis,* and *vulpis*).—In rare instances a mild type of scabies is induced by parasites commonly found in animals. In these cases galleries are rarely seen and the disorder runs a mild course and soon subsides. Exceptions occur, however, as Besnier[4] reported the case of 1 patient whose entire body was invested, including the face and scalp, after transmission of the parasites from a horse. Bosselini[5] describes 2 cases of scabies in an old man and a boy, contracted from an ass. The eruption principally affected the extensor surfaces of the arms and the trunk; there were no intermediate lesions. In the case of a man who contracted the disease from scraping a hog with a pruriginous affection of the skin, a generalized eruption followed similar to that seen in the other cases. The animal, when examined, was found to be infested with acari.

Scabies Norvegica.—(*Norwegian itch*).—This type of scabies was first described by Boeck, as it was seen in the case of a leper. Other cases have been recorded in various places, including one by Hessler[6] and Ravogli[7] in this country.

The disease is characterized by extensive crusting, the crusts being composed of dried pus and scales and riddled with galleries containing innumerable parasites and their ova. In some instances a generalized scaling occurs and the face and scalp are involved. Distortion and destruction of the nails is not uncommon. Beatty[8] recorded 2 cases in which the finger ends and nails were excessively involved. In 1 instance the nails were distorted and disintegrated. In the other they were separated from the bed by crusting beneath the nail plate.

In Hessler's case the entire surface of the body was covered with large thick scales, which were shed freely and were riddled with acarian burrows. By counting the number of parasites in a scale of given size Hessler calculated that the man had upon his person at one time not less than two million mites and seven million eggs.

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[1] Nicolas and Jambon, Annales, 1908, p. 65.

[2] Jour. Cut. Dis., 1911, xxix, p. 339.

[3] Ibid., 1912, xxx, p. 53.

[4] Annales, 1892, iii, p. 624.

[5] Giorn, Ital., 1905, fasc. 1, p. 64.

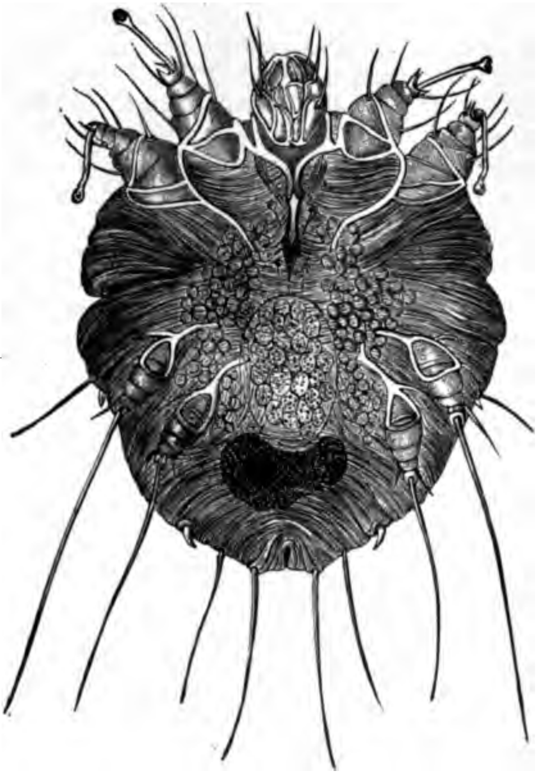
[6] Science, March 3, 1893.

[7] Cincinnati Lancet-Clinic, July 16, 1898.

[8] Brit. Jour. Derm., 1913, xxv, p. 56; "A case of Norwegian or crusted scabies." Idem, ibid., 1915, xxvii, p. 404; "A second case of Norwegian scabies."

Etiology.—The disease is produced by *Acarus scabiei* (*Sarcoptes scabiei*), is contagious and may be transmitted directly or through mediate objects. It is usually acquired by occupying a bed with an infected individual. It may be transferred through the bed clothing or other articles infested with the parasites. It occurs about equally in the sexes in children, but in adults, men are more frequently affected. The proportion of 3 to 1 is given by Wilke.[1] The disorder occurs at all ages and immunity is said not to exist, although examples of apparent immunity are recorded.[2] While the disease is common in public practice, it occurs not infrequently in individuals whose surroundings are of the best. Its frequency in private practice among several observers varies from 5 to 12 per cent. Its prevalence increased following the Civil War and another wave of increase occurred following

Fig. 389



Female acarus fecundated (ventral surface). An ovum arrived at maturity is visible within the body. (After Kaposi.)

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[1] Derm. Wchnschr., 1915, lx, p. 281; abstr., Jour. Cut. Dis., 1915, xxxiii, p. 642; "A contribution to the statistics of scabies based on 2470 cases treated in the Leipzig Skin Clinic from 1903 to 1910."

[2] Montgomery, Jour. Cut. Dis., 1917, xxxiv, p. 90.

the unusual immigration into this country in 1900 and the following several years. A similar increase was noted in Canada at this period,[1] and a similar increase is being noted at the present time following the World War. In Europe many more cases of scabies were noted in civil life shortly after the soldiers began having leaves of absence from military duty.[2]

Pathology.—The female parasite alone penetrates the skin. During her passage in the stratum corneum she deposits ova, about 20 to 50 being deposited in the course of two weeks. In addition to the ova the gallery presents many dark spots which represent the feces of the mite. According to Török,[3] Unna and others the galleries and the acari and ova are situated entirely in the stratum corneum. The parasite (*Sarcoptes scabiei, Sarcoptes hominis, Acareus scabiei, Sarcoptes communis*) has an oval or nearly circular body, whitish in hue, traversed by interrupted rugæ or folds, running for the most part at right angles to its long axis. It is provided with transverse rows of minute bristles on the dorsum, and with groups of trichomæ on the front, sides, and back. There are chitinous hairs at the base of the legs. The two first pairs are provided with pedunculated ambulacra in both sexes; the two posterior pairs in the female terminate each in a long bristle; in the male, the third pair of legs terminate in a bristle, the fourth pair in a pedunculated ambulacrum. The anus lies at the posterior border of the dorsum. The male is 0.2 to 0.3 mm. in length, and 0.145 to 0.19 mm. in breadth; the female is 0.33 to 0.45 mm. in length, and 0.25 to 0.35 in breadth. The tunnels contain the excrement and ova, the latter measuring 0.14 mm. in length. The males commonly perish after copulation; females only after the ova are all excreted. Six-legged larvæ hatch out in four to eight days, slough their skin three times and then are prepared to burrow (Braun).

The ova are oval and their longitudinal axes are placed transversely to the cuniculus. They are deposited at the rate of one or two daily during the passage of the parasite. In the two or three ova found nearest the female only a yellowish color can be distinguished. In the third to fifth traces of an embryo are recognizable, the sixth to ninth contain larvæ, and in the oldest the head and front legs are discerned. When mature, the shell of the ovum is ruptured, usually between the third and sixth days, and the young acarus reaches the surface of the skin by way of the gallery, or through rupture of its roof wall. The acarus survives but a few days when removed from the skin and immersed in liquids which deprive it of air, such as water or oil.

The histology shows a mildly inflammatory process beneath the gallery. Edema occurs in the rete which may be of sufficient degree to induce vesicles. The vesicles are commonly situated beneath one end of the gallery. In the corium edema is present in the papillary layer, the bloodvessels are dilated, and there is a small amount of perivascular cellular infiltration. In the case of so-called Norwegian scabies Nagel[1] found horizontal burrows confined to the stratum corneum. Ijiri[2] found innumerable eggs and parasites in the horny layer. In the epidermis, as a whole, there was marked hyperkeratosis and parakeratosis and moderate acanthosis, and in the cutis were signs of a chronic inflammatory process.

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[1] Hyde, Amer. Jour. Med. Sc., March, 1905, Scabies in the United States and Canada.

[2] Thibierge, Bull. Acad. de Med., 1917, lxxviii, p. 601.

[3] Monatshefte, 1889, viii, p. 360.

Diagnosis.—The diagnosis of scabies is made by recognizing the multiform character of the lesions and their particular location. The characteristic lesion of the disease is the cuniculus or burrow, and when found settles the diagnosis. This burrow may be shaved off with a sharp scalpel and placed on a slide in a solution of sodium hydroxid and immediately examined, when the parasites and ova are easily detected. Some difficulty may be experienced in finding the gallery owing to its concealment by excoriations or other evidences of a traumatic dermatitis, but a careful search in areas of predilection is usually rewarded with the discovery of this lesion. The important lesion in addition to the gallery is the vesicle. After that all of the lesions of a traumatic dermatitis may be found. The important sites are the hands, between the fingers, about the wrists, axillæ, breasts, buttocks, and genital region. The important factor relative to itching is the fact that this occurs after the patient has retired and is well warmed in bed.

From pruritus hiemalis, for which it is frequently mistaken, it is differentiated by the occurrence in the latter of lesions limited, as a rule, to the thighs, legs, forearms, and occasionally the trunk; the genital region and the areas between the fingers being free. The lesions of pruritus hiemalis are simply those of a traumatic dermatitis on an abnormally dry skin, galleries and vesicles both being absent. The itching occurs while the patient disrobes, rather than after he has retired.

From eczema the disease is readily differentiated by the discrete character of the lesions, their special locations, and the absence, except in severe cases, of the formation of the ordinary patches which occur in eczema. Eczema is a much more common disorder than scabies in this country.

Treatment.—The indications for treatment are the destruction of the parasite, the relief of itching, and the management of the cutaneous lesions induced. The remedial agent commonly employed is sulphur and it is best used in the form of an ointment, one to two drams, (4 to 8) to the ounce (30), and it is well to have a part of the ointment base consist of an animal fat, either lanolin or adeps. The technic of its application is important. This ointment should be applied each night for five nights. Before the first application a thorough bath with hot water is taken and the skin well scrubbed with soap. The underclothing put on after the application of the ointment should be worn until treatment is completed, and no baths are taken in the interim. The ointment is reapplied each night. On the morning of the sixth day a bath is taken, all the clothes sterilized by boiling, and no further treatment employed unless a dermatitis is present. If so, this is to be treated by mild and soothing ointments or lotions suggested in the chapter devoted to eczema. Should irritation arise before the fifth application the treatment may be suspended. Overtreatment is as detrimental as undertreatment. If, after two weeks any areas show activity, they may be again treated. The dermatitis produced by sulphur is not infrequently mistaken for an exacerbation of the disease and is made worse by still more energetic applications. Various modifications of the above ointment are employed, the most popular of which is Wilkinson's salve, the formula of which is:

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[1] Archiv, 1913, xcviii, p. 651; abstr., Jour. Cut. Dis., 1915, xxxiii, p. 229: "A case of scabies Norvegica, with a contribution to the histopathology of the disease."

[2] Jap. Zeit. für Derm. und Urologie, 1915, xv, p. 20; Abstr., Jour. Cut. Dis., 1915, xxxiii, p. 656: "Contributions to the clinical and pathological study of scabies Norvegica."

℞—Cretæ preparatæ,	℈iiss;	3	33
Sulphur sublimat.,	℥ss;	15	
Ol. rusci,	℥ss;	15	
Saponis mollis,	℥j;	30	
Adipis,	℥j;	30	

Naphthol as suggested by Kaposi is also employed, and styracis in liquid or ointment form has been much used. Sherwell[1] recommends sulphur in powdered form and states that it is as efficient as when incorporated in an ointment base and is less disagreeable. He directs the patient, after a soap and water-bath, to rub gently over the body one-half a teaspoonful of sulphur lotum, and to dust the same amount between the sheets of the bed to be occupied at night. The bath, the powdering of the body and bed, and a change of clothing are repeated every two or three days. In the average case one week of such treatment is sufficient.

Colloidal sulphur is recommended by Heden.[2] He states that a single application is ordinarily sufficient to eradicate the disease.

In the late war when large numbers of patients with scabies had to be treated in a short time, other methods were suggested, such as fumigation with sulphur vapor and chlorin gas.[3]

Prognosis.—Scabies is a curable disease even after being present for long periods of time. It must be remembered that in sensitive skin, particularly in neurotic individuals, itching may continue for some time after the disease has been eradicated. Another feature is the possibility of an eczema which may have been induced by scabies, and which may require some time for its eradication.

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[1] Jour. Cut. Dis., 1899, xvii, p. 494.

[2] Derm. Wchnschr., April 4, 1914, lviii, p. 404; "The treatment of scabies with colloidal sulphur."

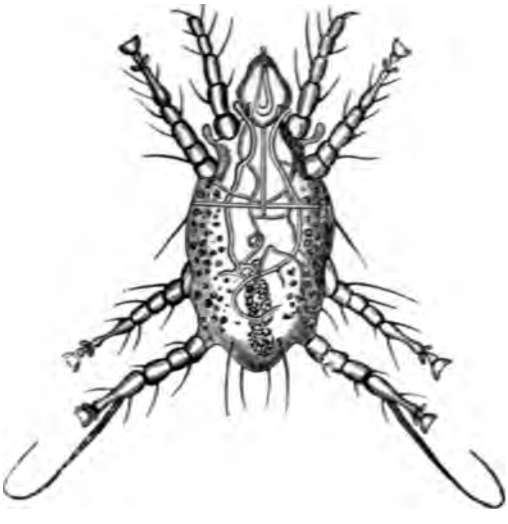
[3] For information on this subject see papers by Bruce and Hodgson, Brit. Med. Jour. August 5, 1916, p. 177; "Treatment of scabies by sulphur vapor." Clark and Raper, Brit. Med. Jour., July 28, 1917, p. 113; "The treatment of scabies by chlorin gas." Bruce, Brit. Jour. Derm., 1917, xxix, p. 100; "Treatment of scabies by sulphur fumigation."

LEPTUS.[1]

Synonyms.—Leptus autumnalis, Harvest-bug, Mower's mite. Fr., *Rouget;* Ger., *Erntemilbe.*

Definition.—The *Leptus* (Figs. 390 and 391) is a minute, reddish or yellowish-red insect of the family *Trombidæ,* visible to the naked eye, and found in summer and autumn clinging to bushes and grasses. It is found in both America and in Europe. Several varieties (*Leptus autumnalis, Leptus irritans, and Leptus americanus*) are described. It attacks man only after its accidental location upon the skin, where it perishes in the course of a few hours. In such situations, however, it induces considerable irritation, betrayed in erythematous, urticarial, papular, and even eczematous symptoms, accompanied by itching of various grades. The parts chiefly affected are the ankles, legs, arms, and feet. According to Duhring, children are especially liable to its encroachments. In these it is found in the axillæ, upon the scalp, and over other parts of the body. The mite may be seen in the skin as an orange-red or brick-red point, which represents often the body of the insect, its head being buried in the aperture of a follicle beneath. Examined after extraction, it is seen to have a relatively large cephalic extremity. It has a short, cylindrical, and conical haustellum, composed of fused double maxillæ; and two strong, hooked, five-jointed palpi, which can be rolled up. There are

FIG. 390



Leptus americanus.

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[1] William MacLennan: The Leptus Autumnalis and it Skin Lesion. Lancet, 1905, p. 1765.

also two hatchet-like mandibles. It has a well-rounded or oval body, 0.3558 mm. long and 0.32 mm. broad, provided with three pairs of legs.

Treatment.—The disorder is relieved by the application of balsam of Peru in olive oil, carbolated oil, spirit of camphor, or other mild stimulant or parasiticide.

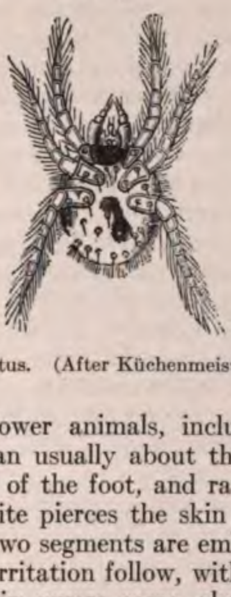
PULEX PENETRANS.

Synonyms.—Dermatophilus penetrans, Sarcopsylla penetrans, Rhinochoprion penetrans, Nigua, Chigoe, Jigger, Sand-flea. Fr., *Puce de sable;* Ger., *Sandfloh.*

The sand-flea, found originally in tropical America, including the West Indies, within the last thirty years has appeared in many portions of tropical Africa and the adjacent islands, as well as in India.

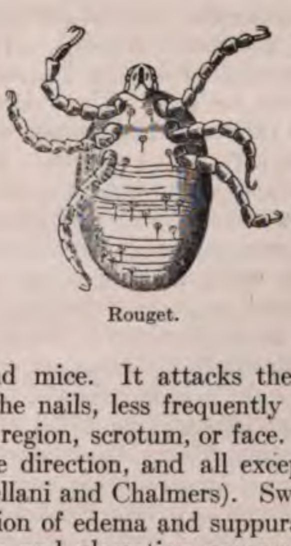
This parasite is a minute, brownish-red, egg-shaped insect, the female of which, after impregnation, penetrates the skin of man and of the lower animals, including rats and mice. It attacks the skin in man usually about the toes, near the nails, less frequently other parts of the foot, and rarely the anal region, scrotum, or face. The parasite pierces the skin in an oblique direction, and all except its last two segments are embedded (Castellani and Chalmers). Swelling and irritation follow, with the production of edema and suppuration, and, in severe cases, abscess-formation and ulceration, accompanied by lymphangitis.

FIG. 391



Leptus. (After Küchenmeister.)

FIG. 392



Rouget.

The treatment of the disease is by extraction of the flea by means of a heated needle, whereby it is destroyed simultaneously. The resulting wound should be dressed antiseptically.

Prophylaxis in "chigger-regions" is of chief importance and is secured by sweeping accumulations of dust from walls and floors, which should be regularly dusted with an insecticide powder, such as pyrethrum.

DRACONTIASIS.[1]

Synonyms.—Filaria medinensis, Filaria dracunculus. Guinea-worm disease, Dracunculus medinensis, Medina worm, Guinea worm. Fr., *Dragonneau, Ver de Kutégal;* Ger., *Peitschenwurm, Medinawurm;* Dutch, *Guineesche draake.*

Definition.—The records of the guinea-worm disease extend to a remote antiquity. It is a disorder due to invasion of the body by a nematode parasite, *Medina worm,* and occurs almost exclusively in tropical countries, more particularly along the West African coast, in the Soudan, Egypt, and Abyssinia; in Asia, especially in the countries bordering on the Persian Gulf, and British India; in the Fiji Islands; and in America, particularly in Guiana, Brazil, and the Antilles.

Symptoms.—The lesions due to invasion of the skin by *Dracunculus medinensis* are observed first at the point where the worm is about to make exit, which point may be at a considerable distance from that where it entered, and the exit may be made after an interval of several weeks or months. This approach to the surface for the purpose of securing exit is accomplished only when the worm is quite mature, at which time it can be felt beneath the surface, suggesting the presence of a soft cord. A local inflammatory process is followed by the formation of a vesico-papule, a pea- to nut-sized inflammatory nodule, or a large furuncular swelling, accompanied by sensations of tension and more or less pain. The lesion, after accidental or intentional rupture, gives exit to a clear, serous fluid, in which the uncolored head of the worm may be recognized. If the fluid be turbid, it is believed that the young embryos have escaped from the uterus. The head appears either at once or in the course of a brief time, producing slow and sinuous movements by alternate contractions and elongations. The entire worm and its young may be wholly extruded in the course of a week or more; or the head may be withdrawn and another swelling form at another part of the surface, the first orifice meantime closing; or, in unskillfully managed cases, the worm may be torn so that the head only is removed, and then a severe lymphangitis, with inflammatory, suppurative, and even gangrenous symptoms may supervene, producing, in fact, the train of symptoms now well recognized in connection with septicemia. In some cases, however, the body may be discharged later than the head, after the mechanical separation of the latter, without serious consequences. The escape of embryos into the adjacent tissue is regarded also as a grave complication.

The chief sites of exit are the ankle and the foot, particularly the heel; but in rarer cases the leg, thigh, buttock, penis, scrotum, hand, trunk, neck, or face may be selected. There is usually but one worm in a single subject of the disease, but the number may be indefinitely large in persons exposed.

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[1] BIBLIOGRAPHY: Bloch, Allg. med. Ctr.-Ztg., 1899, lxviii, p. 729; Foulkes, Brit. Med. Jour., 1898, ii, p. 236; Fox, T., Lancet, 1879, i, p. 330; Harrington, Brit. Med. Jour., 1899, i, p. 146; Mackenzie, Ind. Med. Record, 1898, p. 326; Manson, Brit. Med. Jour., 1895, ii, p. 1350, and Tropical Diseases, 4th ed., 1910, p. 667; Perrin, Annales, 1896, s. iii, vii, p. 131; Roux, Traité prat. des Mal. des Pays Chauds, 1888, iii, p. 553; Scheube, Diseases of Warm Countries, p. 379 (bibliography); Castellani and Chalmers, Manual of Tropical Med., 2d ed., 1913, p. 1441.

Persons of both sexes and all ages are liable to be invaded; but the disease is of more frequent occurrence in the rainy season, and in male negroes and day-laborers. The disorder is produced by *Dcraunculus medinensis* gaining access to the body through drinking water containing cyclops infested with the embryos of the worm. After entering the stomach the cyclops are destroyed by the gastric juice, and *Dracunculus* pierces the gastric wall and develops to maturity, after which it migrates to the various parts where it is commonly found.

Pathology and Natural History of the Worm.—The female worm alone produces the disease. It is, when mature, a yellowish-white, cylindrical filament, gradually tapering toward the caudal extremity. It averages 60 to 80 mm. in length and 0.5 to 1.7 mm. in diameter, the body being extremely extensible by reason of the elasticity of its cutaneous envelope. The cephalic extremity is rounded and terminates in an oval, shield-like disk, in which is a centrally placed triangular oral orifice. There is a small papilla on each dorsal and ventral edge and six smaller ones on the borders of the shield. While there is a straight intestinal tract extending through the body of the parasite, its bulk is composed of an enormous uterus, which is capable of containing, according to estimates made, between eight and ten millions of embryos.

The embryos, without decidua, are flattish, possessing a long, awl-shaped tail, a three-lipped mouth, and a digestive canal. They are capable of surviving for a week in water; and longer in moist earth or water polluted with material which provides them sustenance. Manson states that the embryos, when they have obtained access to the water, transfer themselves to the body-cavity of *Cyclops quadricornis*, fifteen or twenty at a time inhabiting the host, without apparent inconvenience to the latter. Soon the exuvium is shed two or three times, the tails drop off, the worms acquire a cylindrical shape, and develop a tripartite arrangement of the caudal extremity.

In from ten to a maximum of fifteen months the maturity of the female which has been impregnated is attained, and the parasite finds her way from the muscles or other tissues in which she has been lodged or to which she has travelled to the surface of the body.

Diagnosis.—The diagnosis (to be made in countries where the disease is endemic) is based upon the discovery of the worm.

Treatment.—The usual method of treatment by the natives of the countries named is to secure carefully the head when it appears, and to tease out the worm very gently day after day until the entire body is extracted, securing the accessible portion by winding it about a bit of stick or of paper. Continuous irrigation of the wound is both recommended and practised where the disease is common.

Manson has protested against winding out the guinea-worm, stating that at best this process merely shortens by a few days the duration of the treatment in case the parasite is situated properly in the tissues without twists or turns, or if it has arrived at a stage of life when, having discharged its young, it is ready to come out spontaneously. If, as is often the case, the worm is twined and twisted among the tissues, and if she is still emitting her young, she will resist traction, a process which will often result in rupture. In consequence of rupture at this time myriads of young escape into the tissues, producing violent inflammation, which is accompanied frequently by secondary infection and possibly by sepsis. To determine if the worm is ready to come out spontaneously, the opening of the tumor may be douched for a number of minutes at a time, several times a day, by dripping cold water over it. When, under the influence of this douching, the worm no longer emits young, careful winding out is not objectionable.

A successful method devised by Émily[1] consists in the injection of a solution of mercuric chlorid (1 to 1000) into several places in the swelling produced by the worm before the skin is pierced. This kills the worm, which may be absorbed subsequently, or, if cut down upon a day or two later, her body can easily be withdrawn. In case the head of the worm be already protruding, the solution may be injected directly into her body, which is removed easily the following day. This method also reduces the time of treatment from not less than four weeks to the much shorter period of four or five days. The method has been successful in the hands of several observers.[2]

Prophylaxis is secured by protectionof drinking water from pollution, or by having it boiled or filtered before use.

Prognosis.—The prognosis is favorable, save in cases in which septicemic symptoms develop as a consequence of coccogenous infection.

CYSTICERCUS CELLULOSÆ CUTIS.

Cysticerci have been recognized in the skin and subcutaneous tissues by Rokitansky, Guttmann, Schiff, Férréol, Duguet, Lewin,[3] and other observers. The subjects are usually consumers of uncooked meats, especially of pork. In these cases one or many oval or roundish, firm, elastic, cutaneous or subcutaneous, pea- to walnut-sized tumors, isolated or disseminated, and unproductive of pain, project from the general level, and are enveloped by an unaltered integument. They occur upon the trunk and the extremities. In this condition they remain without change for years, and may accompany *cysticerci* of the brain and other portions of the body, productive of the serious disturbance of the economy which such invasions induce. If the skin tumors be opened and their contents examined, the parasite (which is the scolex or hydatid of *Tænia solium*) will be recognized as an ampulliform sac, with a cephalic appendage, reëntrant or projecting, and provided with four suckers and a coronal of hooklets. By no external characteristics can such tumors be distinguished from others of similar size and external appearance. Only in the rare cases of nervous complication could a suspicion of the real character of the disorder arise. Respecting this matter, however, the diagnostician is in no worse position than when called upon to recognize *cysticerci* of the viscera. *Cysticerci* of the liver are recognized during life and subsequently removed by operative procedure.

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[1] Arch. de Méd. navale, 1894, No. 6, lxi, p. 460.

[2] Manson and Boyd, Brit. Jour. Derm., 1896, viii, p. 37.

[3] *Cf.* Vierteljahr., 1894, xxvi, pp. 70 and 271, for review of literature.

Diagnosis.—The diagnosis is from gumma, lipoma, epithelioma, and sarcoma. The first occurs only in the syphilitic; the second has a peculiarly uneven surface and firm feeling; the third is largely facial in situation; and the last is of a malignant character and relatively rapid career.

ECHINOCOCCUS.

Geber[1] states that the parasite *Echinococcus* (larva or hydatid of *Tænia echinococcus* of the dog) is found in the human skin. Of 336 cases reported by Davaine, the parasite occurred thirty times in muscular and subcutaneous tissues, more often in women than in men. The soft, fluctuating tumors or vesicles produce a disagreeable sensation of tension, and they undergo fatty or other metamorphosis after the death of the encapsulated parasite, which usually occurs in from one to two years. Exploration of the superficially seated, fluctuating tumor, covered with unaltered integument, usually demonstrates its nature.

DISTOMA HEPATICUM.

Küchenmeister and Zurn[2] report three instances in which the embryos of the large liver-fluke were encapsulated in subcutaneous tissue. The tumors were painful or painless, and occurred on the head, trunk, and extremities.

Other animal parasites or their larvæ are occasionally concerned in the production of cutaneous or subcutaneous lesions. Stiles,[3] under the title, "Proliferating Cestode Larva," describes a case, reported to him by Gates, in which large numbers of cutaneous and subcutaneous nodules were found, in addition to large and small tumors, in the abdominal cavity, as well as enlargement of the lymph-glands on the side most affected. The worms procured from these lesions were identified by Stiles as being cestode larvæ. The author describes a similar case occurring in Japan.

Cutaneous lesions, consisting of vesico-pustules, produced by *Oxyuris vermicularis* about the perianal region, are described by Vignolo-Lutati.[4]

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[1] Ziemssen's Handbook on Skin Dis., p. 550.

[2] Quoted from Geber, Ziemssen's Handbook on Skin Dis., p. 550.

[3] Jour. Cut. Dis., 1908, xxvi, p. 345.

[4] Archives, 1907, lxxxvii, p. 81.

UNCINARIAL DERMATITIS.[1]

Synonyms.—"Ground-itch," Mazamorra.

Definition.—Uncinarial dermatitis is a dermatitis produced by the larvæ of *Ankylostoma duodenale* and *Necator americanus*. The disorder usually occurs on the feet, particularly on the soles and about the toes. The cutaneous symptoms begin with itching, which is followed by redness of varying shades, tumefaction, and the formation of papules, papulo-vesicles, and bullæ. More serious grades may occur, and at times pustulation and ulceration follow from secondary infection. The lesions may be limited to the region of the toes or involve a large part of the foot. Recurrences may happen from reinfection.

The larvæ of the hook-worm are found in the sand and dirt, being originally deposited there in discharges from the intestinal canal of infected individuals. They attack only those who go barefooted. The disorder is more prevalent in warm, rainy weather, heat and moisture favoring the growth of the parasite. The dermatitis above described may be the initial stage of the general disease, **Ankylostomiasis** (*Agchylostomiasis; Uncinariasis; Hook-worm Disease; Dochmiasis; Tropical Chlorosis; Dirt-eater's Anemia; Mountain Anemia*), which is a progressive anemia occurring in man after the invasion of the intestinal canal by *Ankylostoma duodenale* (*Dubini*) and *Necator americanus* (*Stiles*). As determined by Looss, in 1904, it gains access to the body through the skin, as above noted, or is more rarely ingested. Schaudinn confirmed the observations of Looss by the production of ankylostomiasis in monkeys after spreading a watery suspension of the parasites over the shoulder-blades. After penetration of the body, a long train of systemic symptoms may be induced, including not only the characteristic anemia, but also grave disorders referable to the digestive, circulatory, and nervous systems, in both acute and chronic manifestation, death resulting in severe attacks of the malady.

The disease has been recognized in Egypt, Southern Europe, Japan, Australia, the East Indies, Ceylon, and South America. It has attracted special attention here in consequence of its prevalence in the Southern States and in Porto Rico.

Treatment.—The dermatitis of the feet is treated with the same remedial agents that might be used for an acute eczema. In the early stage, salicylic acid in collodion, 1 part to 6, is recommended (Castellani). Antiseptic lotions containing boric acid, phenol, and bichlorid of mercury are employed, as well as salicylated ointments. The internal treatment should be instituted the moment the dermatitis is recognized (Castellani), consisting in the employment of thymol, eucalyptus oil, eucalyptol, beta naphtol, or male fern.

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[1] Stiles, Trans. State Med. Assoc. of Texas, 1903, p. 353. Leonard, Jour. Amer. Med. Assoc., 1905, xlv, p. 588. Tenholt-Bochum, Zeitschr. f. Medizinalbeamte, 1905, No. 4; abstr. Monatshefte, 1905, xli, p. 409 (Ueber die Loossche Lehre, betreffend die Einwanderung der Ankylostoma-Larven durch die Haut). Dubreuilh-Bordeaux, La Presse méd., 1905, No. 30; abstr. Monatshefte, 1905, xli, p. 409 (Ueber das Vorkommen des Ankylostoma in der Haut). Smith, C. A., Jour. Amer. Med. Assoc., November 24, 1906, p. 1693 (3 illustrations). Ashford and King, ibid., August 10, 1907, p. 471. Schüffner-Deli-Sumatra, Centralbl. f. Bakteriol. u.s.w., Bd. 40, Heft 5; abstr. Monatshefte, 1907, xliv, p. 393 (Ueber den neuen Infektionsweg der Ankylostomalarve durch die Haut).

TRYPANOSOMIASIS.[1]

Human trypanosomiasis is an affection produced as a consequence of the invasion of the body by *Trypanosoma gambiense*, which gains access either through the medium of the tsetse fly (*Glossina palpalis*) or a similar insect which may attack the skin. This *Trypanosome* is a flagellate blood parasite, from 15 to 30 μ in length and from 1½ to 2 μ in width. The mode of invasion, the life-history of the parasite, and other questions of importance in this connection have not been fully determined.

Symptoms.—Cutaneous symptoms occur in the region of the original wound made by the fly, and later as a more or less generalized eruption, due to toxic processes accompanying the general disorder. The bite of the insect produces local irritation, which is followed in the course of a few hours by redness, swelling, tension, and local elevation of temperature. This develops into a slightly elevated, inflammatory lesion, red or violaceous in color, varying in size up to 2 cm. in diameter. It is slightly tender and painful on pressure and is frequently accompanied by enlarged glands. Occasionally, the process is more intense, being accompanied by marked redness and swelling, associated with lymphangitis and elevation of temperature, but unaccompanied by suppuration. These lesions are found chiefly about the legs, knees, groins, axillæ, and neck.

Later in the disease, vesico-papular and papular lesions occur, accompanied by subjective sensations, also a fugitive erythema of toxic type, occurring in circinate patches, some of which are several inches in diameter, their margins fading gradually into the surrounding skin. In addition, a polymorphic urticarial erythema may be present.

Darré describes congestion and edema in the corium, with a perivascular, lymphocytic infiltration, also some inflammatory reaction of the perivascular connective tissue, and the presence of the parasite in the superficial plexus of the bloodvessels of the corium.

The terminal stages of the disease are those now well recognized under the title "sleeping sickness," for details of which the reader is referred to the standard treatises on the subject. This stage of the disorder has thus far been followed by fatal results in a majority of cases.

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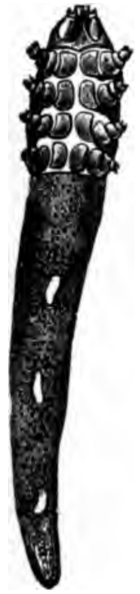
[1] Rogers: Fever in the Tropics, London, 1908, p. 96. Castellani and Chalmers, Manual of Tropical Med., 2d ed., pp. 307–347 and 966–991, inclusive. Manson, Tropical Diseases, 4th ed., p. 150. Darré: The Cutaneous Symptoms of Human Trypanosomiasis, Annales, December, 1908, p. 673; abstr. Brit. Jour. Derm., 1909, xxi, p. 163.

Treatment.—Prophylaxis is to be sought by the avoidance of districts where *Glossina* is found; also by protection from all insects. Arsenic and mercury have been thought to be effectual in some cases. Locally, soothing and antipruritic applications may be employed if necessary.

FIG. 393



Demodex folliculorum.

DEMODEX FOLLICULORUM.

Synonyms.—Steatozoön, or Acarus folliculorum. Fr., *Acare des follicules;* Ger., *Haarsackmilbe.*

Definition.—This parasite was discovered in 1841, by Henle. It is a microscopic creature in the form of an elongated and jointed worm, with clearly defined cephalic, thoracic, and abdominal portions; with eight legs, four on each side of the thorax, each leg with three articulations, and terminating in three small hooklets. The posterior extremity of the body is a vermiform appendage, terminating in a conical point (Fig. 393).

Demodex folliculorum is found upon the free surface of the integument, those parts of the skin particularly where the sebaceous glands are large, and on patients affected with acne or seborrhea oleosa. It is found also upon those free from all evidences of disease. Du Bois[1] found the demodex or its larvæ in all of 200 persons between the ages of 20 and 85 years examined by him. In another series his findings were as follows: In children under 5 years none was found; between 5 and 10 years it occurred in 50 per cent.; while between the ages of 10 and 20 the percentage was the same as in adults. It is encountered also in the substance of the comedo-plug, where at times from five to twenty may be discovered in a single follicle. A demodex which is considered to be a variety of that discovered upon the skin of man infests dogs, mice, and other lower animals; and may in the latter be the source of a disease characterized by furuncular lesions, abscesses, and even fatal results. None of these parasites is, however, known to be transmissible to man.

De Amicis,[2] Majocchi,[3] and Dubreuilh[4] report cases of pigmentation of the skin due apparently to this parasite. The case reported by Dubreuilh was that of a woman forty years of age. The lips, cheeks, other portions of the face, the mammary surfaces, and chest were the seat of light-yellowish spots, where the demodex was discovered in great numbers. The surface was somewhat rugous, due apparently to a peculiar whitish substance, resembling rice-grains, accumulated about the follicular orifices, twelve or more parasites being visible in each field examined. In the case of De Amicis's patient, who was twenty-seven years of age, the face presented a *café au lait* hue over the lips and chin. Majocchi's was a male patient, whose discoloration occurred in the form of a brownish zone surrounding a patch of atrophic lupus.

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[1] Annales, April, 1910, p. 188; abstr. Brit. Jour. Derm., 1911, xxiii, p. 89: The Demodex Folliculorum of Unna in the healthy skin.

[2] Giorn. ital., 1898, iii, p. 205.

[3] Ibid.

[4] Jour. de Méd. de Bordeaux, January 27, 1907, No. 4.

The most effective treatment is by the use of shampooings with green soap.

MYIASIS CUTANEA.[1]

Joseph divides all cases of infection by larvæ of flies into (1) *Myiasis externa* or *dermatosa*, and (2) *Myiasis interna* or *intestinalis*. The former he further subdivides into *Myiasis dermatosa mucosa* and *Myiasis dermatosa œstrosa*.

The larvæ of flies of the families of *Muscidæ* and *Œstridæ* are occasionally deposited on or in the skin, inducing various changes. The different species of *Muscidæ* deposit their ova on open wounds, foul ulcers, or in the nasal cavity or auditory canal in persons suffering with catarrhal or suppurative processes in these situations. A marked example was seen by the author in a fungating carcinomatous ulcer of the face. Markedly destructive effects may be produced in these situations by extension into the deeper tissues and various connecting sinuses about the head and face.[2] The family of *Œstridæ* (*Œstrus*—gad-breeze, gad-fly, bot-fly) deposit their ova on the normal skin by puncture, and the larvæ burrow into the skin and subcutaneous tissue. The areas attacked are usually the exposed parts, and the developing larvæ produce inflammatory nodules and tumor-like new formations. There is usually a central opening in the lesion, from which a sanious, seropurulent or sanguinopurulent fluid escapes, and from which the larvæ may be pressed. The disorder is rarely seen in this country, but is not uncommon in Central and South America.

The larvæ occasionally burrow under the skin, producing irregular lines,[3] which are compared to inflamed lymphatics, but are of a purplish or reddish-purple color; at the end of these suppuration occurs before the larvæ are discharged. Strauch[4] recorded a case with two lesions only, in one of which a tumor the size of a walnut developed, composed of a dense new-formation of connective tissue, in the center of which the larva was found. Smith[1] recorded a case where the parasite travelled from the ankle up the leg to the axilla, then down to the elbow, finally locating on the back of the neck. Tenderness, pain, and itching are described in the lesions.

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[1] BIBLIOGRAPHY: Joseph, Monatshefte, 1877, pp. 49, 106, and 158. Walker, Brit. Med. Jour., 1870, i, p. 151. Abraham, Trans. Derm. Soc. of Great Britain and Ireland, 1896–1897, iii, p. 62 (Remarks on cutaneous myiasis due to œstridean larvæ. A good exposition of the subject, with extensive bibliography). Gilbert, Archiv. Inter. Med., 1908, ii, p. 226 (Infection of man by dipterous larvæ, with report of four cases. Excellent bibliography, covering the entire subject). Costa, Jour. Cut. Dis., 1910, xxxviii, p. 24 (two important parasites of the skin). Mook, Archives of Derm. and Syph., 1920, i, p. 515; Myiasis Dermatosa. Case report.

[2] Yount and Sudler, Jour. Amer. Med. Assoc., 1907, xlix, p. 1914 (Human myiasis from the screw-worm fly. Description of a large number of nasal cases).

[3] Gilbert, loc. cit., p. 233 (Case report. Parasite *Hypoderma lineata*, second stage, constantly changing its location). Miller, Jour. Amer. Med. Assoc., December 3, 1910 (similar case produced by same parasite, as identified by Stiles).

[4] Jour. Cut. Dis., 1906, xxiv, p. 522.

Treatment.—The removal of the parasite is accomplished by free incision of the inflammatory lesion and compression, the subsequent wound being dressed in accordance with the rules of surgery.

CREEPING ERUPTION (LEE).[2]

Synonyms.—Larva migrans (Crocker), Hyponomoderma (Kaposi), Dermamyiasis linearis migrans œstrosa (Kumberg).

Definition.—A disorder characterized by narrow, linear lesions produced by the larva of an insect.

This disorder was first described in 1874 by Lee,[3] later by Kaposi,[4] Van Harlingen[5] and others.

Symptoms.—The characteristic symptom is a progressively extending, thread-like line induced by the progress of the larva through the upper layers of the epidermis. The line is narrow, varying from one to four mm. in width, slightly elevated (about 0.5 mm.), light red in the beginning, dark red later, and of varying lengths. In different portions the line may be straight, curved, wavy or looped. It may begin as a papule, vesicle, or erythematous area, and it may present a beaded appearance or be occupied with vesicles. As the lesion disappears slight pigmentation may follow. Usually only one line is present. In the cases of Stelwagon and others two occurred, and Knowles and others described three. Multiple foci were noted by Whitehouse.[6] Moderate itching and burning may be present over the site of the larva. The line extends over the surface at the rate of an inch or more a day, usually in curves, but it may follow a straight course. In Haase's case the extension occurred only during the night. Occasionally the parasite remains quiet for some time and then resumes its progress. The areas covered with the lines may be limited, or practically the entire cutaneous surface may be traversed during a period of years. The sites upon which the lesions begin are the uncovered portions, hands, face and feet. Not infrequently the early lesion is on the buttocks or about the genitals, and from these situations the lines may extend and involve various areas. Rarely the mucous membrane of the nose, mouth and conjunctiva are involved. Sokoloff described black nits on the hairs near the burrow.

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[1] Trans. Internat. Med. Cong.; abstr. Archiv. of Derm., 1882, xiii, p. 45.

[2] For literature to date of publication see Knowles, Jour. Amer. Med. Assoc., 1916, lxvi, p. 172; "Creeping eruption (Larva migrans) of the skin; particularly in regard to its histological features, including the demonstration of the burrow and the larva in the epidermis." Darier, Annals VI^{e} series, 2 I, No. 3, 1920, p. 113. "Cas de creeping disease (Larva migrans) contracté á Paris."

[3] London Clin. Trans., 1874, viii, p. 44; and ibid., 1884, xvii, p. 75.

[4] Wien. klin. Wchnschr., 1898, ix, p. 399.

[5] Amer. Jour. Med. Sci., 1902, cxxiv, p. 345.

[6] Jour. Cut. Dis., 1917, xxxv, p. 187.

Etiology.—The disorder is produced by the larva of the botfly *Oestridæ*, of the genus *Gastrophilus*, and probable species *Hemorrhoidalis*. The organism was isolated by Sokoloff, who described it as follows: it is 1 to 1.5 mm. in length by 0.5 mm. in width. It has ten segments and is provided with hooklets. The head has two suckers, and the tail presents two club-like processes.

The disorder occurs more frequently in children and begins usually in the summer season. It is much more common in Russia than elsewhere, although several examples have been recorded in America, particularly in the southern portion. In Russia it is seen among peasants working in the fields. Stelwagon's cases had previously been residents of a watering resort. Machado's patients apparently contracted the disorder on sand beaches, whereas in Whitehouse's cases it was contracted from sleeping on the ground.

The histology as described by Knowles follows: The burrows, or passageways, were situated in the lower portion of the stratum corneum, or upper portion of the rete. They were from .25 to .75 mm. in diameter. The contents consisted of degenerated epithelial cells, eosinophiles, lymphocytes and polynuclear cells, fibrin, granular and cellular débris and the parasite. The epidermis, aside from the burrow, showed little change. The rete pegs were either narrow and elongated or broadened. In the corium inflammatory changes were present, indicated by dilated bloodvessels with surrounding cellular infiltration. This was more pronounced in the papillary layer and about the sebaceous glands and hair follicles.

Diagnosis.—The length of the burrow usually readily distinguishes this disorder from scabies, though Hamburger states that in certain cases the resemblance of this burrow to the cuniculus of *Acarus scabiei* is marked.

Treatment.—The treatment usually employed consists in cauterization or excision of the area about the extending point. Stelwagon used cataphoretically a solution of mercuric chlorid, 2 grains to 1 ounce (0.13–30.0) on a one-and-a-half inch area around the advancing end of the burrow, and applied a minute quantity of nitric acid to the suspected site of the parasite, just beyond the end of the line. Hutchins was successful by injecting a few drops of chloroform. In view of the superficial situation of the parasite it would appear that freezing the area with carbon dioxid snow would be effective. Kime[1] advises painting the active area with a 2 to 4 per cent. suspension of salicylic acid in collodion twice daily.

CRAW-CRAW.[2]

Craw-craw is a term used by the natives of the West African coast to designate several diseases of the skin, including scabies, ringworm, eczema, and dermatites of various types occurring in negroes. Most authors agree that great confusion prevails respecting the affection to which the name should be strictly limited. The group of symptoms most commonly described under this title resemble scabies.

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[1] Jour. Amer. Med. Assoc., 1920, lxxiv, p. 527. Treatment of Larva migrans or creeping eruption.

[2] Scheube, Diseases of Warm Countries, 1903, 2d ed., p. 522; Manson, Tropical Diseases, 1910, 4th ed., p. 794.

The lesions begin as pinhead-sized papules, which may be discrete or grouped. These shortly develop into vesicles, which in turn are succeeded by pustules. They occur chiefly on the limbs and trunk, being found particularly between the fingers and on the wrists and elbows. The face is usually exempt. The eruption is associated with violent itching, which is lessened in the cooler part of the day, and disappears entirely, together with the eruptive symptoms, when the patient goes to a cooler climate; but upon a return to tropical temperatures the disease recurs.

O'Neil isolated a small, filarial-like parasite from the lesions, which Manson suggested might be *Filaria perstans*. Its causal connection with the disorder is not proven. Castellani and Chalmers[1] limit the title craw-craw to a disorder described by Plehn as *dermatitis nodosa*. This is purely a papular disorder. The lesions are abundant, and occur on the legs and arms usually, but may spread and occupy the entire surface except the face and scalp. The papules are hard, irregular in shape, and may be roundish, flattened, or acuminate, from millet-seed- to small-pea-sized, and sometimes scale capped. Consecutive lesions occur only from scratching.

Etiology.—The cause of the disorder depends upon the symptom complex to which the term is limited. In all cases the disorder appears to be contagious and infectious. In one group of cases the parasite above noted was described. Plehn was successful in inoculating the disease into healthy negroes.

Treatment.—Locally, cleanliness and the employment of appropriate parasiticides, such as boric acid and phenol solutions, and mercuric chlorid, 1 to 1000, or salicylic-acid preparations, are recommended. Plehn found a 3 per cent. solution of lysol locally applied of value. Removal to a cooler climate was effective in one group of cases.

Prognosis.—In general, the outlook is favorable, though in some varieties the condition of the patient becomes moderately serious.

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[1] Manual of Tropical Medicine, 2d ed., 1913, p. 1593.

CLASS IX.

DISEASES OF THE APPENDAGES.

DISEASES OF THE SWEAT GLANDS.[1]

HYPERIDROSIS.

Synonyms.—Idrosis, Hydrosis, Ephidrosis, Sudatoria, Polyidrosis, Hyperhidrosis. Fr., *Hyperidrose.*

Definition.—Hyperidrosis is an exaggerated quantitative effusion of sweat, localized or generalized, moderate or severe, acute or chronic, persistent or relapsing, the secretion accumulating in visible drops upon the surface of the skin.

Symptoms.—This condition may be physiological, as the result of active exertion in a medium of high temperature; or it may be pathological in character, and in the latter case be either general or partial.

The expression, "general sweating," is self-explanatory. The entire skin of the body participates in the process, and the surface conditions which result favor the development of intertrigo, sudamina, miliaria, and occasionally of folliculitis and furunculosis. Local hyperidrosis is the exaggerated quantitative effusion of sweat limited to certain definite portions of the skin, as the palms, the soles, the dorsa of the hands and feet, the interdigital spaces, the genitals, the axillæ, and the temples. In such cases the secretion occurs moderately or greatly in excess of normal, varying in this respect somewhat in different degrees of temperature and in the rapidity of the circulation. It may involve one or both sides of the body, being generally symmetrical upon the extremities and asymmetrical upon portions of the face.

The typical expression of this disorder may be studied in the hands, which are continually moistened, clammy, or dripping with fluid within a brief time after the most careful drying of the parts. The sweat is commonly cold to the touch of another. In such cases, particularly in women, almost continuous drying of the hands is necessary to protect the clothing, and the rapid soiling of gloves practically interdicts their use. The disadvantages thus arising in individuals of both sexes who are engaged as tradespeople, artists, or hand-workers are obvious. The skin on the palmar surface of the hand, and often on the dorsal aspects of the fingers, has a sodden, thickened appearance, and some degree of hyperkeratosis is always present in severe cases. Occasionally, vesicles are formed, which later become centers of slight exfoliation.

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[1] Excellent bibliographies relating to the various disorders of the sweat-glands may be found in Török's contribution to the subject in Mraček's Handbuch, Bd. 1, pp. 386–485.

With and without this local excess of perspiration involving the hands, there occurs the hyperidrosis of the feet. The outpour of sweat varies in amount from a mere dampening of the feet to a complete saturation of the stockings and the leather of the boots or shoes with the secretion. There is usually a very offensive odor of the region, originating partly in the primary fetor of the secretions themselves, and partly in the subsequent chemical decomposition of the latter. The integument, constantly macerated, may become painful, tender, and slightly reddened; occasionally, there is vesiculation or exfoliation of patches of sodden epidermis, especially between and beneath the toes, in which situation fissures are prone to occur. As upon the hands, though to an even greater degree, a hyperkeratosis is found. The nails are usually thickened and distorted, the result of participation in the keratotic process.

Hyperidrosis of the axillary and genital regions is very often attended with more or less bromidrosis, and almost invariably leads to some degree of intertriginous irritation. The wearing of impervious dress-shields favors the retention and decomposition of the secretion, and thereby adds to the macerating and irritating effects upon the skin of the part. Itching is a frequent complication, and folliculitis, furunculosis, or a dermatitis seborrhoica may supervene. Excess of sweat in the inguinal and genito-crural regions, because of the peculiarities of the location, is especially apt to decompose. More or less fetor results, and if the individual is inclined to obesity a troublesome intertrigo or follicular dermatitis may develop. Occasionally, the sweating may be limited to a small area, such as on one hand or forearm, or in circumscribed areas on the face or back.[2] These are independent of the localized types induced through ingestion of certain foods.

Etiology.—General sweating to a pathological extent occurs in the obese and in those who are subjects of systemic disease, notably tuberculosis, acute rheumatism, malaria, rickets, exophthalmic goiter, and various febrile disturbances. It may result from adynamia due to any cause. Both local and general hyperidrosis has been observed in organic diseases of the nervous system, such as general paralysis[3] and myelitis. Traumatisms, gliomata, gummata, scleroses, and other lesions affecting the cerebrum, medulla, cord, ganglia, and nerve-trunks have all been followed by hyperidrosis of the entire body or of a part only. It is extremely common in persons with an habitually

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[1] Jour. Amer. Med. Assoc., 1912, lix, p. 1193.

[2] Sutton, Jour. Amer. Med. Assoc., 1912, lix, p. 1193, and Jour. Cut. Dis., 1917, xxxv, p. 154. Sweitzer, ibid., p. 281. Ormsby and Mitchell, Pract. Med. Series, 1917, ix, p. 46 and others.

[3] De Montzel, La Presse Méd., January 31, 1903.

rapid heart, and in those who use alcohol, tobacco, coffee, and tea to excess.

Heredity is a factor in some instances, and congenital cases are on record. Localized sweating may be induced reflexly through stimulation of the olfactory and gustatory nerves by ingestion of certain articles of food—tomatoes, cheese, pickles, onions, apples, and other articles.[1]

Local hyperidrosis of the feet is frequently associated with flat-foot.[2]

The frequent obscurity of etiologic relations in hyperidrosis is entirely explicable when certain facts are considered. The predominant influence of the nervous system must be admitted. Though but indefinitely located, the existence of special centers and fibers in the central nervous system for the control and operation of the sweat-glands is evidenced by a mass of clinical observation. The relation of the sympathetic nervous system to all glandular activity is well known. The positive results of experimentation and the deductions warranted by clinical experience indicate that irritation of those centers and fibers, either central or sympathetic, which are secreto-motor, or paralysis of those which are secreto-inhibitory, accounts for general or local hyperidrosis, according to the extent of the distribution of the nervous elements concerned. The etiologic complexity of the situation arises from the fact that this stimulation or paralysis may follow numberless causes: emotion, the action of circulating toxins, direct injury, pressure effects, or reflex action from the periphery or from within. The efforts of the clinician in accumulating data must be supplemented further by those of the anatomist and physiologist before the etiology of hyperidrosis is completely worked out.

Pathology.—Robinson,[3] who examined a number of sections from the palm of the hand, failed to detect any abnormal feature either in the glands or in the epithelium. In a localized case Sutton[4] found hypertrophy of the coil glands with slight cellular infiltration. The disorder is to be regarded as purely functional; and any anatomical changes in the coil glands or the sweat pores are probably accidents of such derangement of function.

Treatment.—When universal, hyperidrosis is to be treated internally by the aid of such remedies as are indicated by the general condition of the patient, and especially by the condition of the heart. The various ferruginous tonics, mineral acids, arsenic, strychnin, strophanthus, quinin (the latter particularly when, as is often the case, a malarial affection is responsible for the disorder), and ergot, with both belladonna and atropin, are all of unquestioned value. Crocker administered sulphur internally. Even though but temporarily serviceable, belladonna and atropin are well used at the outset of most cases. Aconite, jaborandi, and pilocarpin, white agaric (agaricin is recommended in doses of $\frac{1}{6}$ grain (0.011), repeated as required), phenol, and salicylic acid may be named as in the second rank. The narcostimulants as a rule should be excluded.

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[1] Wende and Busch, Jour. Amer. Med. Assoc., 1909, liii, p. 207; Parounagian, Jour. Cut. Dis., 1913, xxxi, p. 515; Sequeira, Dis. of the Skin, 1911, p. 488.

[2] Lesser, Deutsch. med. Wochenschr., 1893, No. 2: Flat-foot and Hyperidrosis. Cutler, Jour. Cut. Dis., 1888, vi, p. 43. Hardaway and Allison, ibid., 1906, xxiv, p. 127: Association of hyperidrosis with flat-foot and Morton's foot.

[3] Manual of Derm., 1884, p. 77.

[4] Loc. cit.

External treatment, which is often promptly efficacious, should not be neglected in any case. The simplest method is by wiping, not washing, the skin-surface until it is dry, and applying a dusting-powder, such as lycopodium, talc, salicylic acid, boric acid, bismuth, magnesia, chloral hydrate (1 part to 5 or 6 of starch), or starch. Alternately with either of these, or in lieu of them, baths or lotions may be employed, aqueous or alcoholic, and medicated with corrosive sublimate, formaldehyd (1 to 5 per cent. solution), tannic acid, ferrous sulphate, naphtol (Kaposi), turpentine, zinc sulphate, alum, potassium permanganate, or common salt. Daily sponging of the affected surface with weak solutions of formaldehyd (1 to 6 per cent.) will remove the odor, and will in most cases greatly diminish the amount of perspiration, but on suspension of the treatment the condition usually returns. G. H. Fox[1] advises a lotion containing 1 part of quinin to 100 of alcohol. Van Harlingen[2] recommends the use of juniper-tar, phenol, and sulphur soaps with the bath as alone sufficient to relieve some cases. Grosse[3] praises highly tannoform, either in powder (1 part to 2 of talcum) or as a 25 per cent. plaster.

For hyperidrosis of the feet, the treatment by the method of Hebra[4] has deservedly high repute. It consists in neatly and completely enveloping the entire foot, the toes separately (after thorough washing and drying), in strips of cotton-cloth, over which has been spread to the thickness of a common knife-blade the unguentum diachyli albi. The parts are well bandaged, and the patient subsequently either remains at rest or pursues his vocation. In twenty-four hours the feet are redressed, without washing, after dry rubbing with charpie and a dusting-powder. This treatment is repeated daily for from ten to twenty days, after which a dusting-powder (boric acid) may be substituted for the local dressing. There occurs a parchment-like desquamation of the epidermis in thick, yellowish-brown lamellæ, beneath which is formed a new, and at first tender but apparently normal, epidermis. When the latter has lost its tenderness the feet are for the first time washed with water. In case of failure, the routine of treatment is repeated. It is scarcely necessary to add that no ill effects are known to have resulted from these therapeutic measures adopted in checking a local hyperidrosis. For the diachylon salve there may be substituted tar, ichthyol, or naphtol ointment.

Gerdeck[5] makes three applications to the soles, at intervals of about eight hours, of the strongest solutions of formaldehyd the skin of the individual will bear. In some instances full strength is well tolerated. A few drops are put in the shoes, the influence on the leather being preservative and not destructive. Relief follows for several weeks, when the treatment may be repeated.

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[1] Jour. Cut. Dis., 1885, iii, p. 24.

[2] Handbook of Skin Dis., 3rd ed., 1895, p. 344.

[3] Klin. therap. Wochenschr., 1889, Nos. 16 and 17.

[4] Dis. of the Skin, New Sydenham Soc., 1866, i, p. 89.

[5] La Riforma Medica, 1898, No. 38.

Morrow[1] recommends foot-baths in the extract of *Pinus canadensis*, followed by the application of boric acid, or of salicylic acid mixed with lycopodium. Frédéricq[2] employs finely pulverized tartaric acid, applied at first with some caution, and always in small quantities. Stewart[3] first bathes the feet in hot water and then soaks them for a few moments (once only) in a solution of potassium permanganate, 4 to 6 grains to the ounce (0.266–0.4 to 30.), after which white-lead plaster is applied as directed above. Weiss[4] highly recommends baths in strong solutions of potassium permanganate. Legoux[5] orders pediluvia of tar-water twice daily for three days, followed by painting of the feet with a solution of iron perchlorid. A solution of aluminum chlorid in the strength of 25 per cent. is of much value as suggested by Stillians.[6] This is applied daily for two or three days and may be repeated later as necessary. *X*-rays have been a valuable means of treatment in many cases treated by the author. In axillary hyperidrosis we have found it useful when pushed to the point of producing a slight reaction. MacKee[7] records the successful treatment of hyperidrosis with *x*-rays.

Prognosis.—The future of any case of hyperidrosis is uncertain. The disease, whether local or general, may spontaneously disappear, may recur, may promptly be amenable to treatment, or may prove obstinate to all therapy. Myrtle[8] reports the case of a male patient, seventy-seven years old, who sweated to death after repeated recurrences of severe hyperidrosis, and after temporary relief from the use of Fowler's solution. As regards these reported fatal cases, it must be said that it is extremely doubtful whether hyperidrosis *per se* has ever caused death.

ANIDROSIS.

Synonyms.—Anhidrosis. Fr., *Anidrose*.

Definition.—This name is applied to those morbid conditions in which no sweat is secreted from the surface of the body. *Hypohidrosis* and *oligidrosis* are terms more exactly used to designate a relative, general, or partial decrease in the quantity of the sudoral fluid. Anidrosis, however, often is used to indicate the latter.

Symptoms. Diminution in the quantity of sweat excreted, or its *complete suppression*, whether general or local, may be a congenital *or acquired peculiarity of the* individual, or may be a symptom of *several disorders*, but as an idiopathic cutaneous affection it is rare.

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[1] See his *résumé* of this subject in Jour. Cut. Dis., 1887, v, p. 68.

[2] *Quoted by Morrow, loc. cit.*

[3] Ibid.

[4] Jour. Amer. Med. Assoc., 1904, xliii, p. 369.

[5] Quoted by Morrow, loc. cit.

[6] Jour. Amer. Med. Assoc., 1916, lxvii, p. 2015.

[7] Jour. Cut. Dis., 1913, xxxi, pp. 101, 361, and 581.

[8] Medical Press, *February* 25, 1886.

It occurs in ichthyosis, atrophy, and in those conditions in which destructive changes have taken place in the skin. It is common to many dermatoses, as, for example, psoriasis, erysipelas, and some forms of eczema; but in these the symptomatic character of the anomaly is shown by the fact that when the skin is relieved of these cutaneous troubles the function of sweat-secretion is restored. Similarly, in neuralgias and certain forms of paralysis a circumscribed and temporary anidrosis may be the local expression of the nervous disturbance, precisely as in the case of the symmetrical hyperidroses. Anidrosis has been observed in association with chronic nephritis; in such relationship it is probably causal to some degree. Not infrequently, individuals who do not sweat are prone to display upon the skin manifestations of an erythematous or urticarial type.

Treatment.—The measures capable of stimulating the sweat-secretion are: the ingestion of water in quantity; the external application of heat; and the use of jaborandi or pilocarpin by the mouth or by hypodermic injection. In the anidrosis accompanying cutaneous disease, the indication is always primarily for the relief of the latter.

BROMIDROSIS.

Synonyms.—Bromhidrosis, Osmidrosis, Fetid or stinking sweat. Ger.. *Stinkschweiss;* Fr., *Bromidrose.*

Symptoms.—In bromidrosis[1] the perspiration when excreted has an unusual odor, or, through subsequent decomposition, develops the odor. It is often associated with hyperidrosis, but may occur independently of the latter, and like the latter also be either general or localized. The odor may be either agreeable or disagreeable, having been in various cases compared to that of certain flowers and fruits as well as to that of several stench-emitting animals. In this respect the sweat presents a striking analogy to the urine, with which it sustains a close and well-recognized physiological relation.

General bromidrosis may be physiological, as in the case of individuals of the African race, or in those with dark skins who profusely sweat during labor or in high temperatures. General pathological bromidrosis is rare. The odòrs emanating from the person in ulcerating syphilodermata, small-pox, malignant pemphigus, granuloma fungoides, and other disorders may in certain cases be associated with the sweat-secretion, but in other cases they doubtless are connected with the decomposition of pathological products of the inflammatory process.

The local varieties of bromidrosis affect the regions in which the sweat is oftenest secreted in excess and its immediate evaporation prevented, as in the axillæ, the groins, the feet, the ano-genital, and the intermammary and inframammary regions. In a qualitative sense, every degree of odor is noted, from that which is slightly disagreeable or offensive to the most intolerable stench. When complicated by a seborrhea, in situations where the parts are not only warm, moist, and covered by clothing, but are also subject to friction and lack of cleanliness, the most intolerable and nauseous fetor is perceived. As in hyperidrosis, there may be coincident or resulting redness, swelling, and even vesiculation or superficial inflammation of the region where the symptoms are chiefly declared.

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[1] Monin, "Sur les Odeurs du Corps humain," Ann. de la Soc. de Méd. d'Anvers, Paris, 1885; abstr. Jour. Cut. Dis., 1885, iii, p. 211.

Etiology and Pathology.—The use of strong-smelling drugs, such as valerian, asafetida, or musk, has been known to produce odorous perspiration. A similar observation can be made regarding certain foods (onions, garlic) and drinks. It is occasionally due to emotional causes,[1] to chronic alcoholism, or to the gouty state. Systemic diseases, such as diabetes, Asiatic cholera, typhoid fever, typhus, dysentery, scurvy, septicemia, and pyemia, may impart peculiar odors to the perspiration. Neurasthenia and dietetic errors (meat-eating in excess, alcoholism) may be responsible for the affection at almost every age and in individuals of either sex. In a mild form it is common in vigorous brunettes soon after the puberal epoch and during menstruation. In bromidrosis of the feet, Thin[2] has recognized microörganisms (*Bacterium fetidum*) in the sweat obtained. Parkes concludes that the only cause of the disease is the covering of the feet, as soldiers with uncovered feet do not suffer from this affection. The fact is patent to every observer that sweat may be effused in a normal condition upon and within the articles of clothing worn, and subsequently generate a stench by chemical changes.

Treatment.—The treatment of bromidrosis is in general that of hyperidrosis, already described. Internally, sodium salicylate has been employed with success in 5 grain (0.33) doses. The regulation of the diet, and especially the disuse of alcohol and tobacco, is essential to the management of some cases, and the general health of the patient should always receive attention.

Locally, the indication is to remove and cleanse frequently the clothing of the part, and to make antiseptic and astringent applications. Formaldehyd solutions in the strength of from 1 to 10 per cent. in alcohol are of the greatest value. They should be followed by the external use of boric acid in powder. Thin[3] successfully employed stockings and cork soles thoroughly dried after being saturated for hours in a jar containing a solution of boric acid. The efficacy of this antiseptic measure he ascribes to the fact that the odor is the result of the development of *Bacterium fetidum* in the secretions. An ointment is also employed by him for similar purposes. This is a solution of boric acid in glycerin, incorporated with a fatty basis of white wax and almond oil, making thus a "glycerated cream of boric acid."

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[1] Hammond, New York Med. Rec., 1877, xii, p. 460.

[2] Brit. Med. Jour., 1880, xviii, p. 463.

[3] Practitioner, December, 1881, xxvii, p. 401.

Clement Hawkins finely triturates 15 grains (1.) of red-lead oxid, and to this adds gradually 1 ounce (30.) of liquor plumbi subacetatis. This preparation is used as a lotion, following a nightly foot-bath containing 1 ounce (30.) of alum. Radiotherapy is also efficient.

Fox advises a 1 per cent. solution of chloral or of potassium permanganate as a topical application. Chromic-acid solutions in 5 to 10 per cent. strength, and potassium-permanganate solutions in the strength of 1 to 1000, may be employed. An efficacious procedure is the nightly bathing of the feet in a saturated solution of boric acid, followed by a thorough rubbing with alcohol; the feet are then dusted with a powder containing equal parts of boric acid and tannoform, with an addition of 2 per cent. of salicylic acid. If desired, tannoform may be used alone, or talcum, or magnesium carbonate may be added to the combination. It must never be forgotten in the management of any case that the covering of the parts affected must receive careful attention; treatment can be only partially successful if this point is neglected.

CHROMIDROSIS.

Synonyms.—Ephidrosis tincta, Colored sweating, Stearrhea nigricans, Pityriasis nigricans. Fr., *Chromidrose.*

Definition.—By this term is indicated the condition in which effused sweat exhibits an abnormal color—yellowish, reddish, greenish, bluish, or blackish. *Cyanidrosis* and *melanidrosis* are terms which have been employed to indicate blue and black sweating. The perspiration may be effused upon the surface already colored, or it may develop color by oxidation in the air, or it may be commingled with substances upon the surface of the skin which produce the abnormal color (bacteria, dyes, chemical agents in themselves without color). The term *pseudo-chromidrosis* is used by some to designate those forms in which, after secretion, the color is produced by the action of microorganisms.

Symptoms.—The usual location for chromidrosis is the region of the eyes, especially the lower lids. Adjacent portions of the face may be involved by extension. The condition occurs less frequently in the axillary, crural, and genito-urinary regions, and has been observed upon the breast, back, and hands. Melanidrosis is the common form, the predominating color presenting a variable admixture of brown or blue hues. Cyanidrosis stands next in order of frequency, often modified by brown or yellow tints. The pigment material is removable with considerable difficulty, imparting to the cloth used a distinct smudgy stain. Oil rather than water facilitates the cleansing process. The color may be diffuse, or the fine, amorphous particles of pigment may be grouped so as to give a punctate appearance to the part involved. In certain forms the hairs participate in the dyschromia. Cases of red sweating are not uncommon. Hartzell[1] has seen it limited to the perineal region. Brownish-red,[2] purplish-red, and other shades occur. Hyperidrosis is not necessarily present.

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[1] Jour. Cut. Dis., 1908, xxvi, p. 380.

[2] Davis, ibid., p. 379.

Whether the sweat is effused rapidly or slowly, the intensity of the color of the area obviously becomes greater with the accumulation of pigment. Subjective symptoms are practically absent.

Etiology and Pathology.—Any age[1] and both sexes may be affected, but the subjects of the disorder are usually women of a neurasthenic type, and in view of the admitted rarity of chromidrosis the suspicion of dissimulation not infrequently arises. While it has been observed repeatedly in vigorous individuals, it is commonly found that the patient's general health is below par. Pelvic disorders in women have been noted in many cases.[2] Excitement through hearing good news produced chromidrosis in a patient observed by Dr. Hyde. Residence near the sea is credited with some predisposing tendency. An instance of red sweating is reported by Temple[3] in a patient who was taking potassium iodid for syphilis. Greenish sweating, due to the presence of copper in the system, has been reported.[4] Dr. Hyde observed one case of this disorder in which the effect was produced by the copper plate of an electrode in contact with an abraded surface of the skin. Authors have attributed the color of the sweat to the presence of compounds of phosphorus, iron, cyanogen, hematin, chromogen, and indican in the secretion. In the case of the last-named substance, there is reason to believe that in many cases of cyanidrosis the indican is excreted in the colorless form (white) and changes to blue on the surface of the skin as the result of exudation.

At the meeting, in 1881, of the American Dermatological Association Dr. Hyde exhibited the hairs of a middle-aged man that had changed in a night from grayish-white to a greenish and yellowish-brown hue. White, of Boston, has observed similar cases of hair-coloration as the result of profuse sweating. It is possible in these instances that the chromidrosis is produced by the mechanical washing of pigment to the surface by the outpouring sweat. In the case reported by Prentiss,[5] that of a young woman affected with an acute, purulent cystitis, whose hair, under the influence of profuse sweating induced by the action of pilocarpin, changed speedily from a light blond to a nearly jet-black hue, a similar explanation might hold, were the possibility excluded of a color-change due to the excretion in the sweat of indican-forming substances absorbed from the focus of suppuration in the bladder.

The red and yellow forms of chromidrosis are believed to be due for the most part to the presence of bacteria. In ten patients examined by Drs. Hyde and Montgomery, five of them women, the sweat was pale-red to blood-red in color, and the axillæ were the regions involved. In all the cases the microscopical examination revealed similar changes. The hairs of the axillæ were thin, pale red, brittle, and surrounded with a colloid-looking, rusty- or bright-red sheath, in places of considerable thickness and having a rough surface. This sheath consisted of red masses presenting a radiating striation, more or less confluent, apparently proceeding from fibers of the cortex of the hair, or from some broken part of its surface. The radiating striations were found to be due to an aggregation of round or ovoid bacteria, which were united in zoöglea masses by a reddish intermediate substance. Nodular swellings on the hair were produced by infiltration of the organism between the separated fibrils. The roots of the hair were free from bacteria. The red tint of the sweat was found to depend upon the numerous roundish masses of zoöglea.

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[1] Le Roy de Méricourt, first to name the disorder, describes a case of rosy sweating in an infant. Arch. gén. de Méd., November, 1857; and Bull. Acad. de Méd., 1884, s. ii, xiii, p. 425.

[2] New York Med. Jour., 1903, xxvii, p. 26.

[3] Brit. Med. Jour., August 29, 1891, p. 477.

[4] Clapton, Med. Times and Gazette, 1868, p. 658.

[5] Phila. Med. Times, 1881, xii, p. 385.

Trommsdorff[1] found a yellow and a red bacterium in a case of red sweating of the armpits. He believes that microörganisms are always present in these types of chromidrosis, and that they should be regarded as special forms of *Lepothrix* (*Trichomycosis palmellina,* Pick). He is of the opinion that microörganisms will ultimately be found to be causally related in some instances to cyanidrosis.

Under the title *seborrhea nigricans,* Mitchell[2] describes an unusual case of chromidrosis in which there was a dark, greasy-looking discoloration of the eyelids and adjacent skin.

The relation of the sebaceous glands to the disorder is as yet not determined. In a case reported by Barié[3] the palms of the hands (free of sebaceous glands) were affected. In Heidingsfeld's case[4] the sweat-glands were normal and the sebaceous glands were absent in the part involved (left forearm).

In all cases, before accepting statements of patients as to the existence of symptoms of this character, it is needful to eliminate the possibilities of deceit and accident. Coloring matters received upon the hands may be transferred, either willfully or ignorantly, to the surface of the body. Hall[5] reports several cases in which supposed cyanidrosis was found to be due to cheap black stockings, the dye of which, when brought in contact with acid sweat, produced a peacock-blue discoloration of the toes.

Treatment.—The treatment of chromidrosis is that of the general state of the patients exhibiting the symptoms. Attention should be directed to the gastro-intestinal tract with a view of preventing excessive indol-formation. Locally, where a parasitic source is suspected, antiseptic measures may be employed.

URIDROSIS.

Synonyms.—Ger., Harnschweiss; Fr., Uridrose.

Definition.—Uridrosis is that condition in which some of the constituents of the urine, chiefly urea, are excreted in excess with the sweat.

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[1] Münch. med. Wochenschr., July 19, 1904, p. 1285 (with bibliography.)

[2] Phila. Med. Jour., 1898, i, p. 117.

[3] Annales, 1889, s. ii, x, p. 937 (with bibliography).

[4] Jour. Amer. Med. Assoc., 1902, xxxix, p. 1519 (with bibliography).

[5] Brit. Jour. Derm., 1902, xiv, p. 418.

While a small amount of urea is to be recognized in normal sweat, this ingredient under peculiar conditions may be increased, and, together with urinary salts, be deposited upon the skin-surface after evaporation of the exuded fluid. Such symptoms have usually occurred either as the result of grave constitutional affections (such as cholera), of organic renal diseases accompanied by anemia, or of the ingestion of jaborandi. In a few cases the symptoms have been presented in individuals who were apparently in good health. The salts of the urine appeared upon the skins of these patients in the form of minute lamellæ, or of a fine powder of whitish color and crystalline aspect. In some cases reported the symptoms have been noted to precede by a few days a fatal issue.

The constantly adjusted equilibrium between the sweat-secretion and the urinary excretion would explain, for cases of mild type, temporary augmentation in the urea found in the sweat of unusually free diaphoresis. Geber supposes that decomposition products, such as ammonium carbonate, possibly in association with volatile fatty acids, may in part account for these conditions.

In the effort to eliminate certain substances accidentally or purposely introduced into the system, the sweat may possibly become charged with iodin, turpentine, tar, arsenic, and other substances. Several of the eruptions described in the chapter on Dermatitis medicamentosa are due to a similar eliminative effort, especially those accompanied by excessive sweating and the production of vesiculation. In the same manner, it may be inferred that the sweat is at times charged with excrementitious and other products of the body; as, for example, the elements of the bile. In patients affected with yellow fever, the skin and even the sweat which exudes from it often exhibit the characteristic hue of that disease. The so-called *galactidrosis*, from supposed metastasis of milk, does not occur; cases thus described have been instances of pathological sweat in the puerperal state.

Phosphoridrosis,[1] in which a phosphorescent quality has been imparted to the sweat-secretion, is reported in rare cases to have resulted from ingestion of phosphorescent fish, and in such wasting diseases as pulmonary tuberculosis, tabes, and scurvy.

Hematidrosis[2] (*Hemidrosis, Sudor sanguineosa, Bleeding stigmata, Neurotic excoriations, Bloody sweat*), reported as observed by several authors (Foot, Ebers, Parrot), is the name applied to conditions in which blood has been seen to exude from an unbroken skin. The phenomena described under this title belong properly to the *ensemble* of symptoms called "hemophilia," and may in some cases be due to direct transudation of red and white blood-corpuscles and fibrin into the interepithelial spaces traversed by the sweat-pores. In a case macroscopically examined by Török red corpuscules were found in the lumen of the coil portion of the sweat-gland.[3] Geber points to the neuralgic, hyperesthetic, pruritic, or emotional symptoms that are the usual precursors to the flow of pale- or bright-red blood. The fact that patients thus affected are mostly women, hysterical, dysmenorrheic, or near the puberal epoch, also throws light upon these cases. In many of them petechiæ, or signs of hemorrhage into other tissues of the body, are observed. The condition may obviously occur in any acute affection presenting purpuric symptoms.

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[1] See Merck's case, Wien. klin. Wochnschrft., 1903, xvi, p. 1091.

[2] Hyde, Jour. Cut. Dis., 1897, xv, p. 557: A Contribution to the Study of Bleeding Stigmata (bibliography).

[3] Mraček's Handbuch, Bd. i, pp. 416-418 (with bibliography).

The bleeding may occur from a single point, or from several in succession, or simultaneously from multiple stigmata. There may be a precedent elevation of the surface forming vesicles, blebs, macules, or papules; or the skin at the site of the hemorrhage may be unaltered. Gangrene has resulted in a few instances. Often pain or other sensations announce the occurrence of the bleeding.

Special caution is to be taken lest patients complaining of these symptoms solicit the hemorrhage by self-injury. In a few cases the persistence of the sanguineous flow has induced a dangerous anemia. The treatment is that indicated by the conditions present.

SUDAMEN.

Synonyms.—Miliaria crystallina. Ger., *Friesel;* Fr., *Miliaire cristalline.*

Definition.—Sudamen is a short-lived eruption, unaccompanied by inflammation, and characterized by vesicles containing sweat.

Three forms of sudamina have been described: (*a*) *sudamina alba;* (*b*) *sudamina rubra;* and (*c*) *sudamina crystallina.* The last-named is the only form to which the term sudamen is properly applied, since it alone of the three designates a purely functional derangement of the sweat-secreting apparatus.

Symptoms.—In this disorder the lesions are thickly agglomerated, but discrete, transitory, and translucent, pinpoint-sized vesicles, resembling dew-drops or seed-pearls, upon the surface of the skin, often requiring the touch to define their real character. These lesions are usually limited to certain regions of the body, as the neck, chest or other parts of the trunk, but rather more generally develop upon the front and sides of the abdomen and in the iliac regions, though they may occur upon any part. They contain each a droplet of sweat, which is removed by evaporation. Their course is rapid, both in evolution and involution, and their sequelæ are exceedingly delicate desquamative flakes, the thin roof-wall which originally covered the sweat-drops having been lifted from the superficial stratum of the horny layer of the epidermis. They are usually preceded by an attack of itching.

Etiology and Pathology.—The disease is the result of excessive sweating, induced by any cause, as violent exercise, the elevated temperature of the summer season, flannel underclothing, vapor-baths, or hot fomentations. It not infrequently follows the hyperidrosis of systemic debility, tuberculosis, inflammatory rheumatism, and the acute infectious fevers. The vesicles may occur as symptoms of the death agony.

Robinson[1] early showed that the vesicles in this disorder are always formed in the corneous layer, and that they are in direct communication with the sweat-ducts. The rete around the vesicles is normal, and their contents always sweat. The disease, in his opinion, is therefore merely an overproduction of sweat, together with some obstruction to its escape. Török[2] found the walls of the vesicles composed purely of the corneous layer, with the sweat-pore opening at the lower border of the chamber.

Diagnosis.—No difficulty can arise in making a diagnosis if the peculiar characteristics of the sudamen be kept in view. All pustular lesions have different contents; all bullous lesions are larger, or are seated on an engorged base, or they lack the crystal clearness of the sudamen, because, however transparent the contents, they are mostly covered by a thicker and less transparent roof. The halo about the lesions of miliaria rubra, or their rosy-pink shade, will determine their character. In varicella the lesions are chambered.

Treatment.—Only the simplest treatment is required. Alkaline and bran-baths may be employed, of the temperature most grateful to the skin. Afterward the surface may be dusted with one of several of the dusting-powders, such as starch, lycopodium, or boric acid, named in the chapter on General Therapeutics. The internal treatment is that indicated by the condition of the patient.

MILIARIA RUBRA.

Synonyms.—Prickly heat, Lichen tropicus, "Heat rash," Red gum, Strophulus. Fr., *Miliaire;* Ger., *Schweissflechte.*

Definition.—This is a disorder characterized by the sudden appearance of vesicles or papules, occurring, as a rule, in hot weather; being more pronounced, therefore, in tropical countries. It is accompanied by itching and burning sensations, and has a tendency to relapse.

Symptoms.—In hot weather or in tropical countries, under the influence of a high degree of temperature, the skin may become the seat of a mild, or in some cases quite severe, disorder, which primarily originates in hyperemia of the sweat-glands. It occurs commonly in those who have been sweating profusely, and particularly in those having a sensitive skin, such as infants and young adults, invalids, the gouty, and the obese. The lesions are usually pinpoint- to pinhead-sized, discrete but closely aggregated vesicles, vesico-papules, or papules.

The eruption may cover the entire surface, but is commonly best displayed in the parts covered by clothing and which are the seat of excessive sweating. The disorder is accompanied by marked tingling, pricking, and burning sensations, which are often in a high degree distressing and may solicit rubbing of the affected part, though the scratching elicited in severe itching is not common here. Minute crusts may form after vesicles rupture. Areas of diffuse redness may develop where few elements of the eruption are visible. The attack may be mild or severe, and may last for a few days or for a few weeks or months, the result of continuous aggravation or the production of new crops of lesions after each recurrence of the cause. The affection is not rarely complicated in obese individuals by all varieties of eczema and intertrigo.

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[1] Jour. Cut. Dis., 1884, ii, p. 362.

[2] Mraček's Handbuch, Bd. i, p. 418 (with bibliography).

The disorder in the heated season of the northern zones is usually scarcely more than an annoyance, while in tropical countries it may induce a severe inflammatory process in the skin.

Etiology and Pathology.—Overheating of the body from climatic effects, high temperature of rooms, excessive use of alcoholic beverages, and sweating under the influence of opium, aspirin, or other sudorifics, are the usual causes of the disease, resulting in hyperemia of the part around the sweat-gland and pore. The disorder may occur in people wearing an excessive amount of clothing, this being exemplified in the cases termed *strophulus* or "red gum," where a unilateral eruption not infrequently occurs due to excessive sweating in an infant closely swaddled. In a histological study Robinson[1] found that the papules and vesicles in miliaria rubra were due to an exudation, composed of serum, corpuscles, and some sweat, into the rete mucosum around the orifices of the sweat-ducts. The changes in the rete were similar to those met with in eczema, and the exudation came from the vessels of the papillæ. He concluded that the disease is not an inflammatory disorder of the sweat-glands, but of the skin, especially around the duct orifices.

Pollitzer[2] described in miliaria rubra a horny layer, swollen by imbibition and slightly edematous; the rete Malpighii contained cystically dilated sweat-ducts; and the cutis was unchanged except in the papillary layer, where the bloodvessels appeared gorged. He further described some vesicles as occurring in the rete, not connected with the sweat-apparatus. These he considers an epiphenomenon. The disorder is due to occlusion of the sweat-duct, which Pollitzer suggests may be caused by the swelling of epithelial cells unprotected by sufficient oil.

Diagnosis.—The temperature to which the skin has been subjected; the sweating, local or generalized; the character of the lesions, and their close agglomeration, all point to the nature of the malady. In papular eczema there are usually patches and a serous exudate, which stiffens linen, as distinguished from sweat-moistened but unstiffened clothing. Vesicular eczema rarely exhibits uniformity and symmetry of the resulting lesions.

Treatment.—The indications are to remove the cause, as far as practicable, and to soothe the irritated skin. Lotions and powders are preferable to ointments. The parts may be washed or wiped with starch-water, almond-meal-water, or bran-water, and then dried and thoroughly dusted with a soothing powder, such as equal parts of boric acid, zinc oxid, and starch. The use of soap should be interdicted. The bowels should be regularly evacuated, and acidulated beverages (never iced) may be ingested in moderate quantity. When medicated lotions are indicated, one may use the zinc-oxid and lime-water combinations which are useful in the treatment of acute eczema. One may also employ with advantage black wash, diluted one-half with water; weak lotions of phenol, 1 part to 250 of alcohol and water; or of the biborate of sodium, 1 part to 200. When a dusting-powder is used talcum, the stearate of zinc, or acetanilid, 1 part to 30 each of boric acid and talcum, may be employed with advantage.

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[1] Jour. Cut. Dis., 1884, ii, p. 362.

[2] Ibid., 1893, xi, p. 50.

MILIARY FEVER.

Synonyms.—"Sweating sickness." Fr., *Suette miliaire.*

Definition.—This is an epidemic disorder, accompanied by sweating and a cutaneous exanthem. Pineau[1] gives a description of the disease as it occurred in epidemic form on the island of Oléron, in France, where, of 1000 patients affected, between 150 and 200 perished. The eruption appeared in the form of hyperemic maculæ, disappearing under pressure, after which there rapidly formed myriads of reddish or whitish, grouped, unequal-sized, acuminate papules, rising from a whitish and macerated surface. Among these papules were interspersed lesions of sudamina. The region of the face was not spared, and the conjunctivæ were occasionally affected. In the course of from two to four days pinhead- to bean-sized, varioliform but non-umbilicated pustules formed in the site of some of the papules, the contents of which disappeared by resorption. The final lesions presented were large, flat, reddish papules, the skin of the face particularly becoming reddened and swollen. In the course of from ten to twelve days general desquamation ensued, with extensive palmar and plantar losses. Relapses occurred in some cases, with diffuse redness of the surface or with the eruption of crops of reddish plaques, or yet again with the occurrence of furuncles. The sensation was that of myriads of needles thrust into the skin.

The exanthem was accompanied in some cases by fever. In the fatal cases death resulted from exhaustion.

Geber and other writers believe that the lesions described are not peculiar to any special disease, and they deny the possibility of an independent miliary fever.

HYDROCYSTOMA.

Synonyms.—Hidrocystoma, Cysts of the coil-ducts.

Definition.—Hydrocystoma is a chronic, non-inflammatory disorder, characterized by the presence on the face of scattered, isolated, deep-seated, persistent, clear vesicles. Robinson[2] has published a report of his studies in this affection, which he first described in 1882. Reports of cases and studies of the disease have been made also by Hutchinson, Jackson, Jamieson, Rosenthal, Hallopeau, Tebel,[1] and others.

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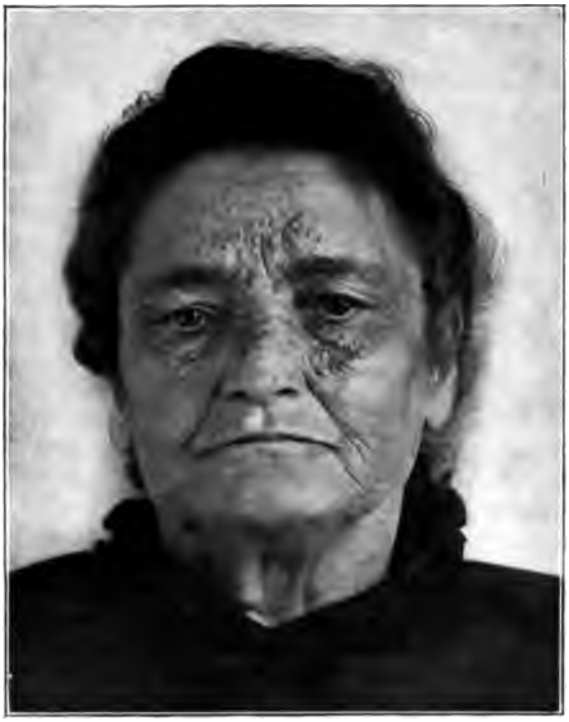
[1] Arch. gén. de Méd., January, 1882, p. 25.

[2] Jour. Cut. Dis., 1893, xi, p. 293.

Symptoms.—The lesions are discrete or closely set, few or exceedingly numerous, tense, well-developed, clear, shining, rounded or oval, pinhead- to pea-sized vesicles, non-inflammatory, and never superficially seated—that is, never so near to the surface as the vesicles of miliaria, because the base of all hydrocystomata is to be found in the corium. They have no tendency to rupture spontaneously. The lesions are whitish in color, or, when of greater age and size, are dark bluish, especially at the periphery, some resembling boiled sago-grains. The signs of inflammation are absent; occasionally a mild hyperemia becomes evident at the periphery of a single cyst. The contents are neutral or slightly acid in reaction, and pellucid, never changing to a yellowish hue. When uninjured, the lesions resolve in time by desiccation, leaving a short-lived pigmentation. They occur chiefly

FIG. 394



Hydrocystoma. (Howard Fox.)

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[1] Annales, 1903, s. iv, iv, p. 273.

upon the face, especially the brow, cheeks, and nose, in symmetrical distribution, and may prevail for weeks or months, or disappear in cool weather. They are always accompanied by very free sweating.

Etiology.—The disease occurs almost invariably in middle-aged women, more often in those engaged as laundresses, who have been sweating freely at their work, the face being simultaneously exposed to warm vapor. Men are very rarely affected. There is usually aggravation of the disorder in summer and either complete or partial relief in winter. Aggravation has been noted at the time of the menstrual period. One of Hutchinson's patients exhibited lesions on one side of the face only. The patients seen by us have been usually of the dispensary class, and were women who worked much over the wash-tub. Hyde and McEwen[1] reported a typical case occurring in a woman past the menopause, where sweating was one of the symptoms of exophthalmic goiter. Hallopeau describes exacerbation of the symptoms at the menstrual period, and he believes the disease is much influenced by the condition of the nervous system.

Pathology.—The epidermis, hair-follicles, and sebaceous glands are in all parts normal; the papillary layers are involved only when the cyst approaches the upper part of the corium, where "a thin plate of flattened papillary body" is found above. Below, in places, the lumen of the sweat-duct is found enlarged and distended with liquid and a granular material. The enlargement in the duct begins above the coil of the gland, and usually in the lower part of the corium. There is some perivascular leukocytosis in progress here and there in the vicinity of the vessels, but this was not a marked feature in any one of the several sections examined by Robinson. The cavities of each duct were found lined with epithelial cells. Adam[2] believed that the cyst developed in the coil portion of the gland, but the finding of Robinson, that the duct immediately above the gland proper is the part involved, has been confirmed by the researches of several authorities.[3]

Diagnosis.—The lesions of sudamen and pompholyx are readily distinguished by their superficial character and their situation, as they are rarely discovered upon the face. The vesicles of eczema are short-lived and inflammatory. In adenoma of the sweat-glands the lesions are often painful and usually firmer and larger than in hydrocystoma.

Treatment.—The lesions can be made to disappear by puncturing each, thus permitting the escape of the imprisoned fluid. A weak spirit lotion may then be applied, and this may be followed by the application of dusting-powders. Due care should be taken to avoid the effective causes of the malady.

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[1] Amer. Jour. Med. Sci., June, 1903, n.s., cxxv, p. 1000.

[2] Brit. Jour. Derm., 1895, vii, p. 169.

[3] Jarish, Hautkrankheiten, Wien, 1908; Lebel, Annales, 1903, s. iv, iv, p. 273; Pinkus, Zeitschrift, 1904, xi, p. 642; Schidachi, Archiv, 1907, lxxxiii, p. 3 (experimental production of hydrocystoma); Török, Mraček's Handbuch, Bd. i, pp. 423–426 (with excellent bibliography).

HYDRADENITIS SUPPURATIVA.

This disorder was described in 1864 by Verneuil, and since then many writers have recorded different disorders under the same name, while other observers have described similar cases under different titles. The disorder under consideration is essentially a suppurative inflammation involving the sweat-apparatus. Some cases of *folliclis* have been included in these descriptions on account of involvement in these cases of the sweat-glands.

Symptoms.—In the disorder under consideration, the common sites of the lesions are the regions of the axillæ, anus, nipples, scrotum, and labia majora. In these parts the lesions may be single or multiple. They may attack other regions, but avoid the palms and soles. The lesions resemble indolent furuncles without pilo-sebaceous involvement. Occasionally, several of the nodules coalesce and form a flat tumor with a number of openings. The disorder is chronic.

Etiology and Pathology.—As predisposing causes should be counted all conditions, general and local, which tend to lower the vitality of the tissues. The origin of the disorder is unknown, though it should probably be sought in local infection or in the action of some toxic agent excreted by the coil-glands.

The process has been shown to be a diffuse inflammation of the coil-glands and periglandular tissue, usually terminating in necrotic suppuration and destruction of the glands. Primarily, the coil-epithelium undergoes changes which are responsible for the cellular infiltration of the peripheral tissue. No microörganisms have been recognized in the cases examined.

Treatment.—The general condition of the patient should furnish the indications for treatment of each case. Locally, the nodules should be opened and dressed antiseptically. The disease is stubborn but eventually terminates in recovery.

GRANULOSIS RUBRA NASI.

The above title was given by Jadassohn[1] to a peculiar affection of the nose in children. This name is accepted today, but the disorder has been described under other headings, as: "A peculiar form of acne with changes in the sweat-glands" (Luithlen); "Perisyringitis chronica nasi," and "Dermatitis micropapulosa erythematosa hyperidrotica nasi" (Jadassohn); "A peculiar inflammatory dermatitis of the nose of young individuals, with sweating" (Herrmann); "False acne rosacea in children" (Audry); "A form of chronic erythema of the nose in children" (Dubreuilh). Since first described by Luithlen, in 1900, the number of reported cases is constantly increasing.[2]

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[1] Archiv, 1901, lviii, p. 145.

[2] For report of cases and discussion of the disease in general see: Audry, Jour. Mal. Cut., 1903, xv, pp. 809-811; Baumer, Zeitschrift, 1904, xi, p. 640; Mirolubow, Deutsch. Med. Zeitung, 1906, Nos. 62 and 63; Malherbe, Jour. Mal. Cut., 1906, xvii, p. 96; Pick, Archiv, 1902, lxii, p. 105; Herrman, ibid., 1902, lx, p. 77; Lebet, Annales, 1903, s. iv, iv, pp. 273 282; Pinkus, Zeitschrift, 1904, xi, p. 642; MacLeod, Brit. Jour. Derm., 1903, xv, p. 197; and ibid., 1906, xviii, pp. 342 353 (excellent *résumé*); Ormsby, Jour. Cut. Dis., 1905, xxiii, p. 183. Barber, Brit. Jour. Derm., 1919, xxxi, p. 106. Granulosis rubra nasi in boys, two cases.

Symptoms.—The disease is a chronic inflammation of the skin covering the cartilaginous portions of the nose, and is characterized by a more or less sharply defined area of redness, on which are scattered irregularly, without definite arrangement, pinpoint- to pinhead-sized, dark-red macules and papules. Interspersed among these lesions are beads of perspiration; often the rounded papules are tipped with a droplet of sweat. The hyperidrosis of the nose is a striking feature of the disease, and there is in some cases a coincident hyperidrosis of the face and hands. Occasionally, vesicles are found containing a clear droplet of fluid; when these are large and deeply seated, they are not to be distinguished from the lesions of hydrocystoma. The redness of the area involved ranges from pale pink to a purplish hue; the color of the lesions fades on pressure. The nose is cold to the touch, but subjective symptoms, aside from slight itching, are absent. There are usually to be found evidences elsewhere of impaired peripheral circulation.

Etiology and Pathology.—The patients are usually children ranging in age from six months to sixteen years. A few cases have been reported in adults. Both sexes are about equally affected, the individuals being for the most part delicate children with poor circulation. Hereditary influences have been traced in some instances.

The histological changes are those of inflammation situated in the corium. The superficial bloodvessels are dilated; about them, and also about the sweat-ducts, is an infiltration consisting of leukocytes, connective-tissue cells, plasma-cells, and occasionally mast-cells. A few leukocytes may be found in the dilated interepithelial lymph-spaces of the rete. The pilo-sebaceous follicles are usually normal. If the infiltration about the sweat-duct is marked, a cyst of the proximal portion of the duct may be found, as in hydrocystoma.[1]

The essential factors in the production of the disorder are not positively recognized. It is probable that vascular disturbances are the primary elements in causation. Hallopeau[2] regards the disease as a neuro-hyperidrosis, due to disturbances of the vasodilators and vasoconstrictor nerves.

Diagnosis.—The disease has been mistaken for lupus vulgaris, because of the gross appearance of the papules. In the disorder under discussion the lesions disappear under pressure, there is no tendency to ulceration, and marked hyperidrosis is present. From acne vulgaris and rosacea it may be differentiated by the absence of involvement of hair-follicles and sebaceous glands.

Treatment.—Treatment is usually not very effectual. The general health should be improved by proper hygiene and the administration of indicated internal medication. Locally, astringent powders, lotions or pastes containing resorcin, salicylic acid, or ichthyol, may be tried. Tannoform as a dusting-powder has been used with considerable suc-

[1] Lebet, Pinkus, loc. cit.

[2] XV Internat. Med. Cong., Lisbon, 1906. Ref. in Zeitschrift, 1906, xiii, p. 573.

cess. Relief by these applications is usually temporary, but complete disappearance of the disease may be looked for when the patient attains maturity.

DISEASES OF THE SEBACEOUS GLANDS.

SEBORRHEA.

Synonyms.—Steatorrhea, Acne sebacea, Dandruff, Seborrhagia, Sebaceous flux, Stearrhea. Ger., *Schmeerfluss;* Fr., *Séborrhée.*

Definition.—The clinical phenomena which should be included under the title "seborrhea" are at present matters over which differences of opinion exist. Since the time when Unna first placed under the caption of "eczema seborrhoicum" a number of conditions which had previously been classed with seborrhea, the tendency has been toward a constant increase in the former category at the expense of the latter. In the recent work of Sabouraud, which has been accepted by many investigators, the only conditions described under the latter term are those which are clinically non-inflammatory. All seborrheic disorders which clearly show evidences of inflammation are considered under the term "dermatitis seborrhoica."

Seborrhea has heretofore been described as occurring in two varieties: seborrhea oleosa and seborrhea sicca, the latter being subdivided into the dry, scaling, and crusting forms. The simplest division from a clinical standpoint, and, if the conclusions of Sabouraud are confirmed, also from an etiological standpoint, would be to separate the symptoms formerly described under seborrhea sicca into two divisions, namely: pityriasis simplex and pityriasis steatoides, which would leave seborrhea oleosa as the sole representative of the seborrheas. The term "pityriasis," however, hardly seems compatible with some forms of the disease.

Seborrhea Oleosa.—This form of seborrhea, variously known as hyperidrosis oleosa (Brocq), seborrhea simplex (Unna), stearrhea simplex (Wilson), and *acné sébacée fluente,* is characterized by the presence of an excessive amount of oily secretion on the scalp, face, and some other regions of the body. The sebaceous secretion is exuded as an oily fluid upon the surface both of the hairy and non-hairy parts of the skin. In the former situation, both in adults and infants, the free oily substance is seen to cover as a coating both skin and hairs, and, especially in adults who have suffered much loss of hair as a result of the sebaceous disorder, to produce a glistening and shining appearance of the scalp. This is the form of seborrhea responsible for permanent baldness. In women with long hair the locks are often matted together in a glue-like paste. The same greasy layer may be seen over the non-hairy portions of the skin, especially about the nose, forehead and cheeks. Free drops of oil can occasionally be wiped from such surfaces with a handkerchief. The ducts of the sebaceous follicles here are either patulous or plugged with sebum, which may be squeezed out, appearing as worm-like bodies. The skin-surface may be slightly reddened or be pallid, but it is usually cold to the touch. The oily substance serves to entrap particles of dust and soot floating in the air; thus a peculiarly dirty or even blackish hue of the face is often produced. This form of seborrhea, though most common on the face and scalp, may occur on the chest, the back, the pubes, the genitals, and rarely on the other parts of the body. In the negro, in whom the sebaceous glands are usually well developed and active, the oily form of seborrhea is common, and the flux at times is practically physiological. Subjective symptoms in seborrhea oleosa are usually slight, though a moderate amount of itching is commonly present.

Seborrhea Sicca.—Seborrhea sicca varies greatly in its manifestations, but in general its features may be divided into the scaling and the crusting forms of the disease. The scaling form, variously known as *seborrhea furfuracea* or *pityriasiformis, pityriasis simplex, eczema seborrheicum,* and *eczema squamosum,* is most common on the scalp, in which region it is popularly known as "dandruff." Seborrhea capitis in its commonest form (pityriasis simplex) is recognized in the adult by the formation on the scalp of fine, branny, slightly greasy, white or grayish scales, which may be so abundantly shed as to fall freely and cover the shoulders of the patient whenever the hair is brushed or otherwise disturbed. At other times these fatty scales are more or less adherent to the scalp-surface, or are piled up in laminæ one upon another. These scales may mat the hairs to the scalp or be disseminated through the mass of the hair, some of the hairs penetrating a flattened, greasy scale, as a twig might be passed through the center of a leaf. In consequence of their deprivation of unguent, the hairs to which the affected glands are accessory become dry and lusterless.

The affection may be circumscribed, and exhibited in conspicuous patches covered by scales; or it may extend uniformly over the entire surface of the scalp; or, as is frequently noticed, may fringe the brow at the line of the hairs and then extend chiefly over the vertex, being conspicuous at the line where the hairs are parted from vertex to brow. Beneath the scales or crusts of dried sebum the scalp is usually lusterless and of a slate-gray color. The disease not infrequently extends from the scalp to the adjacent portions of the face, neck, and ears. In these situations the skin may be slightly reddened, while the scales are thin, adherent, and not very abundant. The eyebrows, the region covered by the beard, and the pubic region may be affected, although less frequently, in the manner described above. In the latter region the itching is often more severe than occurs when the disease is limited to the scalp. The disorder may appear on the portions of the face more distant from the scalp, and on other parts of the body, in the form of dry, roughened patches, which scale more or less, but which are only slightly, if at all, reddened. On any of these surfaces the condition may shade insensibly into those described under dermatitis seborrheica.

The crusting forms of seborrhea may occur on any of the hairy or non-hairy parts of the body, but are most common on the scalp and face. In the scalp large, waxy, greasy, yellowish scales and crusts, associated with itching and alopecia (*pityriasis steatoides*), may occur. The so-called "waxy" form is represented by the physiological *vernix caseosa* of the newborn infant, and by the more or less adherent, dirty-yellowish cap often long surviving upon the vertex of young infants. Occurring later in infancy, the disease is known as "milk-crust," or as *crusta lactea*. This may merely be persistence of the dried *vernix caseosa* about the vertex in the newborn, or it may occur in scalps which have been perfectly cleansed after birth. The crust differs somewhat in color with the tint of the child's complexion, and may vary from a light yellow to a dark brown; it may be thick, greasy, and mat the hairs; or be thin, dry, and friable. Inflammatory complications are very prone to develop from decomposition of the material making up the crust, in which event the disorder becomes properly a dermatitis seborrheica. The region of the brow, the surface covered by the beard of the male, and the pubic region may be involved in this type of the disease, though less frequently than in the furfuraceous form.

On the face, this form of seborrhea is characterized chiefly by the accumulation of thick, dirty-yellowish and even yellowish-black accumulations of sebaceous matter, often adherent to the surface and disfiguring the features by the mask produced. This condition is conspicuous about the nose, where the disease is at times symmetrically disposed. The crusts once removed, the skin beneath is generally found to be pallid or slightly reddened, with the orifices of the sebaceous ducts patulous; while the under surface of the separated crust is seen to project downward in corresponding delicate prolongations, comparable to stalactites. The crusts rapidly reform when the disease is not arrested. They are found in the furrows on either side of the nostrils, on the brows, the cheeks, and the pavilion of the pinna of the ear. They are most common at the puberal epoch in both sexes, when the sebaceous glands of the skin are in a state of developmental activity.

Seborrhea may affect the eyelids, in which situation a mild dermatitis seborrheica usually supervenes, owing to the trauma of rubbing and the incessant movement of the skin in winking. The lids are then reddened, slightly swollen, and in various degrees covered with minute crusts (less frequently with scales). The eyelashes often fall, and in cases of long standing their loss may be permanent, owing to atrophy of the follicles.

Seborrhea of the umbilicus assumes special features in that the fatty matters in this region are remarkable for their tendency to speedy decomposition, with the production of an exceedingly fetid odor and a mild degree of seborrheic dermatitis.

Seborrhea of the genitals in men is usually located in the sulcus behind the corona glandis, though in individuals with a tight or a redundant prepuce it may become more extended. In women, the accumulation occurs about the clitoris and vestibulum, though the external labia may be covered with the secretion in various degrees of fluidity. The smegma preputii, supplied by the glands of Tyson, may thus be a source of trouble either by its retention, or by its secretion in abnormal quantity or quality. In either event, the tendency is to decomposition, fetid odor, and subsequent irritation, which may lead to an inflammation of severe grade.

Etiology.—Seborrhea, except that form which appears in infancy, is most frequent at the age of puberty or adolescence—that is, at the time of greatest activity of the glands. It may appear, however, at any age. It occurs about equally in both sexes. The commonest seats of the disease are: the scalp, the face, the genital region, the dorsum of the body between the scapulæ, and the anterior surface of the chest; all of these are regions where the oil-glands are especially numerous and well developed. Seborrhea oleosa is found more frequently in persons of dark complexion, while seborrhea sicca is more common in blondes. A family tendency to seborrhea of the scalp, with the resulting alopecia, may often be noted.

Among the predisposing causes may be counted all systemic disturbances which lower the vitality and general nutrition. Seborrhea may thus follow acute infectious diseases, and frequently appears during the course of chronic exhausting diseases, such as syphilis or tuberculosis. Constipation, indigestion, sedentary habits, and the excessive use of alcohol and tobacco, may be classed as predisposing factors. The disease occurs, however, in individuals who are apparently in excellent health. Among the local predisposing causes are the wearing of stiff, heavy, and ill-ventilated hats, and the failure properly to care for the scalp. Women with long hair are generally obliged to bestow special attention upon the scalp. Men with short hair attend chiefly to its disposition upon the head, and because this is so easily accomplished often neglect the care of the scalp. Such neglect is followed frequently by seborrhea sicca (*Pityriasis simplex and Pityriasis steatoides*). Both varieties of seborrhea are frequently found, often with inflammatory complications, in nuns, with whom the scalp, ears, and neck are encased snugly in stiff, unventilated headdresses.

While there is much, both in clinical experience and in laboratory findings, to commend the theory that the disease is of parasitic origin, the definite organisms have not been decided upon. Those thus far isolated and described have not been universally accepted.

Pathology.—As a rule, at puberty, and in some individuals throughout their lives, the quantity of oily sebum excreted is larger than usual, and it is not always possible to draw sharply dividing lines between the physiological and the pathological process. In the dry form of seborrhea, the secretion is dryer than usual and mixed with cells exfoliated from the ducts of the glands and hair-follicles, and with imperfectly metamorphosed cells from the glands themselves. Unna[1] believes that the skin is lubricated by oil from the coil-glands, and that in seborrhea oleosa (which he terms *Hyperidrosis oleosa*) the secretion is practically all furnished by them, the sebaceous glands being involved, if at all, secondarily. Beatty[1], on the contrary, states that the coil-glands do not furnish the oily secretion in this condition. Sabouraud[2] concludes that seborrhea oleosa (also comedo, acne, and alopecia areata) is due to an inflammation of the sebaceous glands, caused by a definite microbacillus, which is found within a cocoon-shaped mass of epithelium at the neck of the follicle.

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[1] Brit. Jour. Derm., 1894, vi, p. 257; and Histopathology, p. 222.

It is generally believed that the coil-glands secrete fat, but how much they supply and what part they play in seborrhea are unsettled questions. The fact that seborrhea is most frequent and most pronounced in regions where the sebaceous glands are largest and most numerous is fairly good evidence that these glands, more than the sweat-glands, are active in the production of the disease.

According to Sabouraud,[3] the sequence of events is as follows: Pityriasis simplex capitis is produced by the bottle bacillus (Unna) or spores of Malassez.[4] In this form, occurring in children, there is a dry, scaling process (dandruff), in which the scales fall abundantly, and the hair but little. At this time the scales show the process to be hyperkeratotic, and not inflammatory. At puberty, when hyperactivity of the sebaceous glands is present, a second factor is added, the polymorphous coccus, producing gray colonies (Unna's morococcus). The process now becomes more greasy, the scales form adherent, yellowish, waxy crusts, and the condition is transformed into the second-grade pityriasis steatoides. Now there is added from a pathological standpoint a perivascular cellular infiltration in the corium, with edema and parakeratosis. Finally, a third factor is added, the microbacillus of Unna and Sabouraud, and an oily seborrhea is the result. This form terminates in marked loss of the hair. At this time the sebaceous glands show hypertrophy, with degeneration of the hair-follicle, which accounts for the hair-loss.

Seborrhea oleosa may occur independently of the other processes above described, and the sequence of events above noted may also occur at different ages.

As to the specificity of the organisms, no uniformity of opinion exists. Jackson and McMurtry[5] are not convinced that the microbacillus is responsible for seborrhea, and Whitfield[6] is of the same opinion. Moreover, the sebum retained in the follicles furnishes an excellent culture-medium for an unusual development of the microorganisms which may be found on the scalp in normal conditions. Schamberg[1] has demonstrated Sabouraud's microbacillus in the follicles of individuals having no signs of seborrhea or of other diseases of the sebaceous glands.

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[1] Brit. Jour. Derm., 1894, vi, p. 161.

[2] Brit. Med. Jour., 1901, ii, p. 858; and Les Maladies du Cuir chevelu, Paris, 1902.

[3] Loc. cit. *Cf.* excellent description by Jackson and McMurtry, Jour. Cut. Dis., 1912, xxx, p. 608; also, The Diseases of the Hair, pp. 298–323.

[4] Kraus (Archiv, cxvi, p. 723, with plates showing the bacillus as a fungus; abstr. Brit. Jour. Derm., 1913, xxv, p. 293) believes the organisms described as bottle bacilli are not of one type. Some forms appear to be true moulds, while others seem to be sporoidal elements of mycelial fungi.

[5] Loc. cit.

[6] Allbutt and Rolleston's System, 1911, p. 8.

Unna and Elliot state that the microscope shows inflammation to be present in all but the simple oily form of seborrhea. As stated before, it is difficult to draw sharply dividing lines between the types here described, which clinically show little or no evidence of inflammation, and the distinctly inflammatory forms described as dermatitis seborrheica.

Diagnosis.—Seborrhea is to be distinguished from eczema, dermatitis seborrheica, ichthyosis, impetigo, psoriasis, syphilis, and trichophytosis capitis. The distinctly inflammatory character of the first two serves to differentiate them. In ichthyosis, the scale is dry and non-greasy; and the disease is congenital, and usually involves the entire body, whereas seborrhea is generally acquired and is rarely universal. Crusting impetigo of the scalp might be confused with seborrhea; the former is an acute disease; its lesions are comparatively small, circumscribed, and isolated; the crusts differ in character from the sebaceous matter formed in seborrhea; and the skin beneath is reddened and evidently the seat of an exudation. Psoriasis of the scalp may be recognized by the presence of typical patches upon the body; the scales are lustrous, larger, and not greasy unless some fatty application has been made to soften them; they cover a reddened integument; and alopecia is not produced even in persistent cases. Some of the pustular syphilodermata located upon the scalp and face, if observed only in the stage of crusting, might be confounded with seborrhea; but the history of the case, the discovery of other signs of syphilis, such as adenopathy, or mucous patches, the character of the secretion, the condition of the surface beneath the crusts, and the small size, more definite outline, and characteristic grouping of the lesions, should point to the identity of the disease. Trichophytosis capitis may be recognized by the non-greasy character of the scales upon the affected patches, which are usually circumscribed and circular; the hairs in the areas involved are fragile and often appear as broken-off stumps, in which the presence of the parasite is shown by the microscope.

Treatment.—The general and internal treatment of seborrhea should be varied to meet the requirements of the individual case. The preparations most often indicated are: iron in the case of anemic young women, cathartics in sluggishness of the bowels, and cod-liver oil and the bitter tonics when there is impairment of nutrition. Arsenic, employed in the manner suggested by Sir Erasmus Wilson, is praised by Hebra:

℞—Vin. ferri,		f℥jss;	45	
Liq. potass. arsenit.,				
Syrup.,	āā	fʒij;	8	
Aq. dest.,		f℥ij;	60	M.

Sig.—A teaspoonful to be taken three times daily with the meal.

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[1] Jour. Cut. Dis., 1902, xx, p. 99.

In many cases the acid-iron mixture of Startin, or some modification of it, admirably meets the indications:

℞—Magnes. sulph.,		℥ij;	60	
Ferri sulphat.,		℈ss–℈j;	0.66–1	33
Acid. sulph. dilut.,		fʒij–fʒiv;	8–16	
Infus. quassiæ,	ad	f℥iv;	120	M.

Sig.—A teaspoonful in water, to be taken through a tube after eating.

Throughout the treatment the physician should insure a careful observance of the laws of hygiene. Sunlight, nutritious food, and open-air exercise are not to be disregarded. When the scalp is involved, the patient should be encouraged to discard the hat as far as possible consistently with the circumstances in which he may be placed, and thus promote the favorable action of light and air upon the part affected. In cases in which it can be tolerated, daily cool salt-and-water sponging of the entire body-surface, followed by brisk friction, as described under the treatment of acne, may be employed with great advantage.

The first indication to be met by local treatment in seborrhea is the removal of the crusts and the fatty matters accumulated upon the surface. It is always well to warn patients, especially if the disorder be upon the scalp in an aggravated form, that a considerable loss of hair will result. Many of the hair-filaments are so impoverished by the disease and so loosened in their follicles that a complete cleansing of the scalp-surface will bring the hairs away in quantities sufficient to threaten speedy baldness; and it is not rarely the case that patients attribute this to the treatment rather than to the disease. The fatty accumulations are first to be soaked with some oily fluid to facilitate their removal; for this purpose olive oil, vaselin, almond oil, glycerin, or lard is usually employed. The substance selected should be used in quantity sufficient to permeate all crusts. It may be poured over or be rubbed into the scalp several times in the twenty-four hours, and at night a flannel or other cap should be worn. In the case of children and infants, gentleness is required in thus treating the scalp, especially in the subsequent washings, lest the surface be irritated. In women it is rarely necessary to cut the hair. As soon as the soaking with oil is complete, the crusts are to be removed by washing with soap and water, though when the accumulations are bulky, the masses may be gently removed with the fingers or a comb. When the scalp is tender, ordinary toilet or Sarg's glycerin soap may be applied with warm water; but it is usual, in the case of adults, to employ the well-known tincture of sapo mollis. The surface should be thoroughly sponged with the tincture, and then warm water added until with gentle rubbing a lather is abundantly produced over the scalp, when an excess of water is finally used to cleanse the part of crusts, oil, and soap. The scalp and hairs are then thoroughly dried and anointed with some bland, fatty substance, if the exposed surface be tender and irritable; if not, with some stimulating pomade or lotion.

In cases in which milder effects are required, the scalp may be washed with water containing such alkaline substances as borax, ammonia, or potassium carbonate. The popular prejudice against these articles is based upon the abuse of strong alkaline lotions in the hands of inexperienced persons. Such lotions may readily be tested by the tongue before use upon the scalp. They should in all cases be followed by an oily or greasy application, medicated to meet the requirements of the case. Tar-soap may be used at times with advantage for cleansing the scalp.

After the use of soap and water, which may be repeated as often as need be (daily, at intervals of several days, or once a week), the scalp is to be thoroughly anointed. For this purpose olive oil, cod-liver oil properly scented, almond oil, vaselin, or glycerin and water may be used. Van Harlingen recommends, as a substitute for other oils, the oleum sesami (oil of benne), since it does not dry and clog as do the former. An ounce (30.) of this oil, rubbed up with 5 grains (0.33) of powdered benzoin, and digested for three hours over a water-bath, with the addition of 3 drops of absolute alcohol, and filtered, furnishes an excellent basis for oily mixtures to be used on the scalp. Any of these applications can be made conveniently with a medicine-dropper. Crocker advocates the use of a lotion containing acetic acid prior to the application of oily preparations to the scalp, the object being to aid the penetration of the remedy.

In the place of oils after these ablutions, pomades are often used with more advantage. For this purpose vaselin, lanolin, lard, and cold-cream ointment furnish the best bases. To obtain the desired consistency, any one of these may be used alone or in combination with the others or with an oil.

Of the many substances employed and recommended as medicaments for these pomades, sulphur, resorcin, salicylic acid, the red oxid, red sulphid of mercury and the ammoniated mercury are most serviceable.

Sulphur is of great value in the treatment of all sebaceous-gland disorders: in the form of an ointment, 15 grains (1.) to a drachm (4.) to the ounce (30.) of vaselin or other ointment-base. One-half or the same quantity of resorcin may often be added with advantage to the pomade. The alterative effect of the mercurials is also as evident in seborrhea as in many other cutaneous disorders. At the head of the list, for this special purpose, stands the red mercuric oxid in strength of from 2 to 4 grains (0.133–0.266) to the ounce (30.) of ointment; but ammoniated mercury, and calomel in the proportion of from 5 to 10 grains (0.33–0.66) to the ounce (30.), may be often substituted for the former with advantage. The tars are useful in many obstinate cases; oleum rusci may be added in the strength of from 1 to 10 parts to any of the salves recommended above. Ichthyol in ointments in the strength of from 5 to 10 per cent.

has also proved efficacious. An excellent formula for the scalp is the following:

℞—Sulphur. præcipit.,		ʒj;	4	
Lanolin. anhydrous,				
Glycerin.,				
Aq. rosæ,	āā	ʒijss;	10	
Saponis,		℈ss;		66 M.
Sig.—Ointment for scalp.				

Lotions are well adapted to some cases. They are cleanly and easy of application, and are more pleasing to most patients, especially to women with long hair. Their efficacy is often enhanced by the addition of a small amount of oil. Mercuric chlorid is admirably adapted for use in lotions; so also are the tinctures of cantharides, capsicum, nux vomica, and the salts of quinin. A good formula is as follows:

℞—Resorcin.,		ʒijss;	10	
Hydrarg. chlorid. cor.,		gr. ij;		133
Ol. amygdal. dulc.,		ʒij;	8	
Tinct. cantharid.,		ʒij;	8	
Alcoholis,		℥ij;	60	
Aq. dest.,	q. s. ad	f℥vj;	180	M.
Sig.—To be rubbed into the scalp.				

For this may be substituted ½ ounce (15.) of resorcin in 2 ounces (60.) of alcohol and 6 ounces (180.) of rosewater.

Often the combined use of a pomade and a lotion is advantageous. A convenient procedure is to have the patient shampoo the scalp once a week; after the hair is dry a pomade sutably medicated is applied with the finger-tips, and on each of the intervening six days before the next shampoo a lotion is employed upon the scalp with the aid of a medicine-dropper.

Repeated applications and patient care of the scalp are necessary to secure complete relief in the case of a disease as essentially chronic as seborrhea. At times the local treatment may be changed with advantage. Not infrequently, too vigorous treatment is followed by a more or less acute dermatitis. In this case stimulating preparations should be replaced by soothing ointments or lotions until the induced inflammation has subsided.

The treatment outlined above for the hairy portions of the body may be used with success also for the relief of seborrhea of the non-hairy portions, especially the face. Here, it will be observed, the crusts have a tendency to reform, and the most persistent treatment is necessary to secure permanent relief. Occasionally, after cleansing the surface with soap and spirit-lotions according to the indications of each case, it is of advantage to apply the ointment selected for subsequent application, not only by gently smearing it on the parts with the tips of the fingers (always the most effective method), but also by spreading it on a compress, which, for the night at least, may be fixed in contact with the part. Unna's lead-plaster mulls, used for this purpose in Germany, may be fairly well imitated by drawing strips of cheesecloth through heated diachylon ointment and then smoothly smearing them with the same material. When the tendency to reformation of the crust is abated, one or more of the dusting-powders may at times be employed with advantage for the purpose of protecting the skin or of exercising upon it an astringent effect. Sulphur and salicylic acid are especially valuable in these dusting-powder combinations.

Seborrhea oleosa is best treated with lotions or with powders. Should the skin become irritated under these applications, ointments may be substituted for a time.

Astringent lotions or powders containing tannin, zinc sulphate, zinc oxid, bismuth subnitrate, sulphur, salicylic acid, or tannoform are often serviceable.

The local treatment of seborrhea of the genitals is somewhat different. Ointments rarely answer well in disorders of the mucous surfaces, and green soap is too irritating for similar employment. Here washing with a good toilet-soap and warm water is sufficient for the purposes of cleanliness, and diluted lotions containing alcohol, or aromatic wine, suffice. These lotions can be made astringent with tannin, alum, or zinc sulphate, and when there is pain or tenderness opium may be added. In this form of the disease, as also in seborrhea of the umbilicus, phenol or chlorinated soda may be necessary to correct fetor. After the employment of these lotions boric acid with talc (1 to 4) or zinc oxid (1 to 8) may be dusted over the part.

Prognosis.—In forming a prognosis in cases of seborrhea of the scalp, it must be remembered that the disease is frequently obstinate, and shows a decided tendency to recur unless some treatment be continued for weeks or months after the scalp is apparently well. The resulting loss of hair, if symmetrical, may be remediless, but much may be done in the way of saving the hair which is left. Facial seborrhea is much more amenable to treatment; seborrhea of the genitals and the umbilicus is an entirely manageable disease.

DERMATITIS SEBORRHEICA.

Synonym.—Eczema seborrhoicum.

Duhring was the first observer to show that a type of inflammation of the skin, to which he gave the name seborrhea corporis, was closely allied and usually consecutive to seborrhea capitis. Later, Unna[1] advanced the theory that a single morbid process, to which he gave the name *Eczema seborrheicum*, was responsible for a number of varied clinical manifestations which had previously been considered separate disorders. Under this title he included seborrhea sicca (or pityriasis) of the scalp, face, and body, some chronic circumscribed forms of eczema, and many cases which most observers still believe are forms of psoriasis. In America Elliot has furnished an excellent presentation of the subject.[2]

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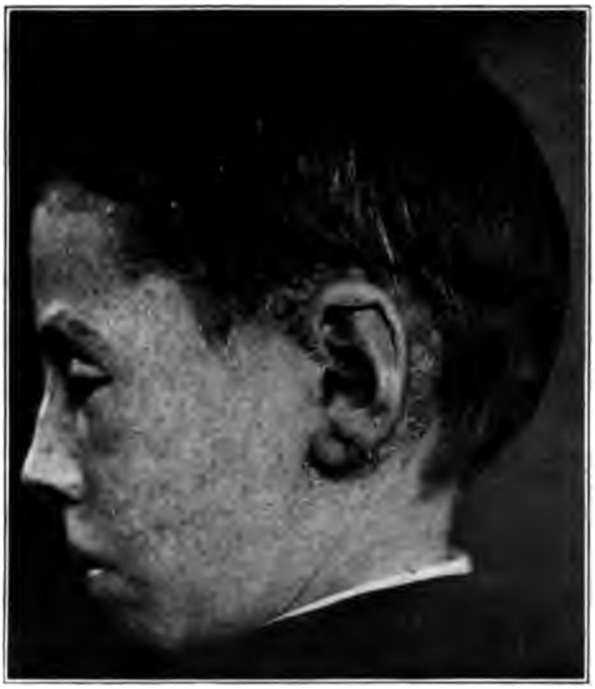
[1] Monatshefte, 1887, vii, and Histopathology.

[2] Morrow's System, iii, p. 273.

Though Unna gave eczema seborrheicum a wider range than is accepted by the majority of dermatologists, there is little doubt that the most of the phenomena he describes under that title are intimately related etiologically and pathologically. It must be said that the tendency today is toward his position, though it is doubtful if his views in their entirety will ever attain full acceptance. Following Unna, the term by many authorities is made to include certain seborrhoic disorders which are non-inflammatory, in the sense that the clinical signs of inflammation are absent. These same disorders, however, may show histologically distinct, though perhaps slight, evidences that the pathological process is inflammatory. It is manifestly not easy to mark accurately the dividing line between the inflammatory and the functional in these instances without the aid of the microscope. As the term itself implies an inflammatory complex, the expedient course would seem to be to make the division between seborrhea and eczema seborrhoicum purely clinical; in other words, to place in the former group those disorders which show no clinical sign of inflammation, reserving for the latter group those which are manifestly inflammatory.

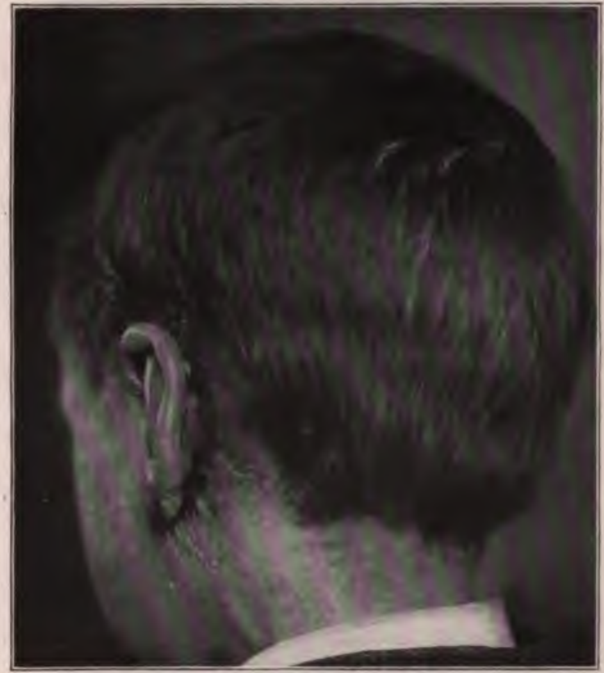
FIG. 395



Dermatitis seborrheica.

Many of the conditions described under eczema seborrheicum arise from the implantation of an inflammation upon an ordinary seborrhea, as the result of some form of external irritation. Prominent among these exciting factors must be mentioned the decomposition of the excessive sebaceous secretion upon the uncleansed or improperly cleansed skin; but it cannot be denied that any dermatitis, produced by whatever irritant, may, in individuals with a tendency to functional disturbances of the sebaceous glands, take on a seborrheic character. This fact, viewed in the light of the present-day conception that the difference between eczema and dermatitis is largely etiological, argues in favor of a change in nomenclature from eczema seborrheicum to dermatitis seborrheica.

Fig. 396



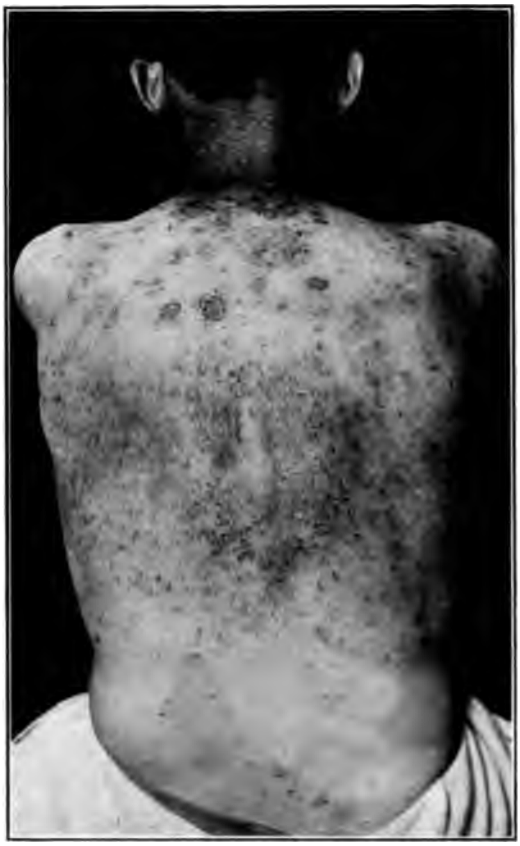
Dermatitis seborrheica.

The eczema seborrhoicum of Unna, therefore, narrowed by the elimination of clinically non-inflammatory forms, is here described under the name Dermatitis seborrheica.

Symptoms.—Dermatitis seborrheica almost invariably begins on the scalp, and often remains limited to this region, though frequently it extends to the ears, temples, forehead, neck, and adjacent parts. The disease is not uncommon on other parts of the body where the sebaceous glands are large and abundant, as in the sternal, interscapular, inguino-scrotal, axillary, and umbilical regions. It may appear, however, on any part of the body, and in rare instances is universal. The disease is extremely variable in its course and mode of extension.

It may remain confined to the scalp for years and then extend to adjacent surfaces, or appear on portions of the body distant from the scalp, leaving the intervening surfaces unaffected. Such spreading of the disease may be very rapid, or so slow as to be almost inappreciable; while the lesions may be numerous, extensive, and acute in type, or few, scattered, and indolent in character.

FIG. 397

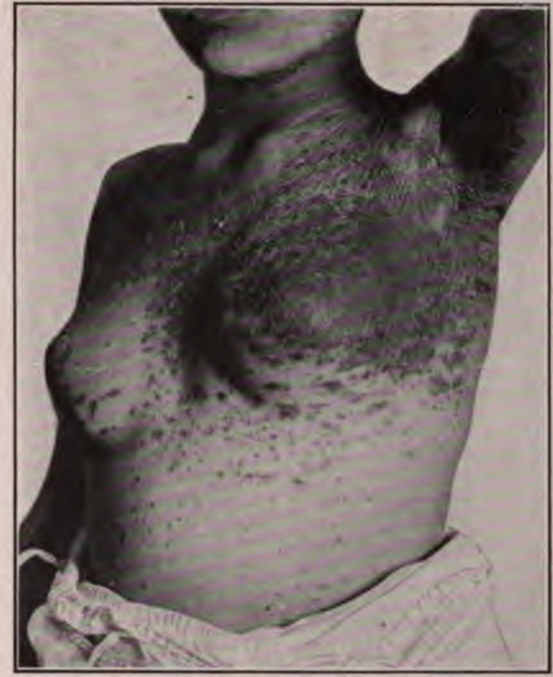


Dermatitis seborrheica.

The affection varies considerably in appearance in its different phases, and especially in different regions. In the scaly form, which is the most common, there is a scanty or abundant formation of fine, branny scales; the skin is somewhat reddened, and often has the peculiar yellowish color which is characteristic of the disease. The scales may be large and abundant, and heaped up in dry, adherent masses, simulating those sometimes seen in psoriasis; but in such cases the scales are usually somewhat fatty. Frequently, there is a coexisting seborrhea oleosa, with the formation of yellowish to brownish, soft, greasy, and non-adherent masses, suggesting crusts rather than scales, under which the skin is more or less reddened and the mouths of the follicles patulous.

The disease often appears in the form of oval or rounded macules and patches, or as small, scale-capped papules, which may remain discrete or may coalesce to form slightly elevated plaques. The macules, papules, and plaques are sharply outlined, and patches that are spreading peripherally frequently present a circinate border with a fading yellowish center. By the coalescence of several such areas polycyclic, gyrate bands may be produced. The color of the lesions is reddish or pinkish, modified by the yellow tinge that is nearly always present. Scaling and crusting in varying degrees are usually present, as in the more diffuse forms described above. The lesions may occasionally be moist over all or part of their surface, but the characteristic vesicles and pustules of eczema are absent, and the discharge when present is usually distinctly greasy. Of the varied manifestations of the disease, the scaling forms are the most common, but in a given case the type may change gradually or rapidly, and multiformity of lesions is not unusual. Itching is usually slight and may be absent.

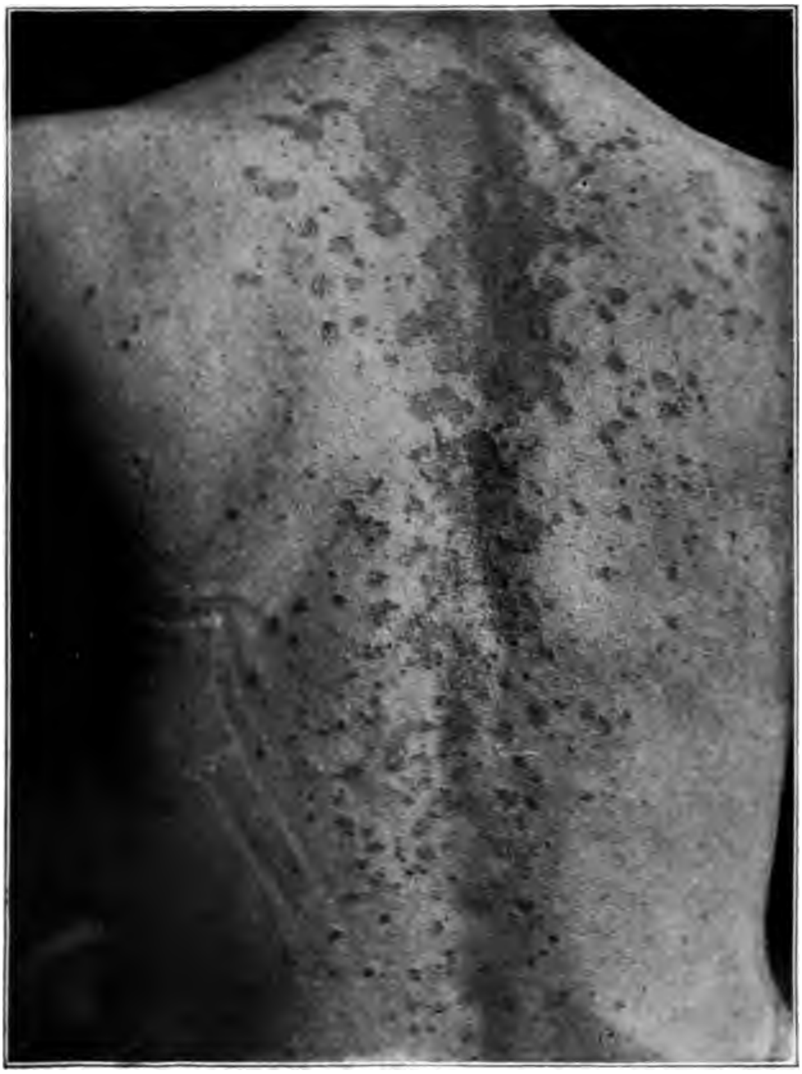
Fig. 398



Dermatitis seborrheica in a young girl.

On the scalp the onset of the disorder is particularly insidious and often unnoticed until attention is attracted to it by a thinning of the hair, moderate or really annoying itching, and a scanty or abundant formation of scales over more or less of the surface. In the early and mild forms the condition is practically that described under seborrhea sicca; the point of emergence from the latter condition into a dermatitis seborrheica is here considered as marked by the appearance of

Fig. 399



Dermatitis seborrhoica. (Stopford Taylor.) (Photo by Oram).

the clinical characteristics of inflammation. The vertex is the usual site of the affection, but the entire scalp may be involved. The scales may appear in any of the forms described above, but are usually fine, dry, grayish, and slightly greasy. The lowest layers of the scales are usually firmly attached to the underlying surface, which, at first dry, lusterless, and pale, becomes more or less hyperemic. After the condition has existed for a time, alopecia is noticed, while the hairs of the affected regions are dry and lustreless. The condition may persist for months or years with but slight change. In more severe forms the heavier masses of scales and crusts described above may occur upon distinctly reddened or moist patches. Seborrhea oleosa may complicate the process, with its characteristic greasy crusts and oily condition of scalp and hair. Itching is usually quite severe, and the inflammatory features of the condition are aggravated by the trauma of scratching. In infants and occasionally in adults a genuinely acute dermatitis may supervene, involving portions or all of the scalp and usually extending to the adjacent parts of the face. The condition known as "milk-crust" (described under Seborrhea) passes into a form of dermatitis seborrheica when inflammation is produced by irritation from decomposition products in the material accumulated upon the scalp. In adults circumscribed, oval or circinate, reddened, and scaling, moist, or crusted patches may appear, chiefly at the nape of the neck and about the temporal and parietal regions, often extending to the ears and portions of the face. Occasionally, a sharply defined, red band, more or less covered with scales or small crusts, may be seen at the margin of the hair, especially on the forehead and on the neck. Such bands closely resemble those of psoriasis, but usually have a more regular and even outline, much less infiltration and thickening of the skin, and they lack the characteristic scales and outlying separate lesions of psoriasis.

The ears and the surfaces surrounding them are, after the scalp, more frequently involved than other parts of the body. Any of the above-described types of the disease may be seen in this region, the moist and crusting forms being quite common, especially back of the ears, where fissures frequently occur. The disorder not rarely affects to a very marked degree the lining of the external meatus of the ear, blocking it with crusts and interfering seriously with hearing.

The beard, moustache, eyebrows, and pubes may present symptoms differing but slightly from those in the scalp. The disorder may linger about the verge of the moustache or other parts of the beard, showing its grease and scales even at a distance from the line of hairs, with a well-defined, reddened surface beneath. The same occurs about the line of the eyebrows. Alopecia is uncommon in any of these regions except the eyebrows.

On the face the pityriasic forms, with a moderate degree of redness, are common on the nose and adjoining portions of the cheeks, the eyebrows and the regions between, and the eyelids and their margins, and may be exhibited on any part of the face. The more inflammatory moist and crusting types are most frequent along the junction of the alæ of the nose with the cheeks, but may involve the entire nose and other parts of the face. The macular and papular types above described are most common on the cheeks.

Seborrhea Corporis.—Upon the trunk is frequently found Unna's "flower-leaf" or "petaloid" type of the eruption, which was first described by Duhring and to which have been assigned by different authors the titles *Seborrhea corporis*, *Seborrhea papulosa* or *lichenoides* (Crocker), *Lichen circumscriptus* (Willan), *Lichen annulatus et serpiginosus* (Wilson), and *"flannel-rash."* Its favorite sites are the sternum and interscapular region, but rarely it spreads in more extensive areas on other parts of the trunk. In a well-marked case the lesions appear in the form of sharply outlined circles or segments of circles, which enlarge centrifugally, often coalescing to form patches with irregularly circinate outlines. The extreme borders, which represent the early stage of the lesions, are made up of very small, red papules, usually covered with fine, whitish or yellowish, dry or fatty scales. As the border progresses, the center undergoes involution, so that from without inward the patch may display varying shades of red, brown, and yellow, while the whole surface is often the seat of a furfuraceous desquamation. Round or oval, somewhat elevated, solid lesions are frequent, and may scale slightly or be covered with yellow, greasy crusts. In less perfectly developed cases and in those modified by friction of the clothing or frequent bathing, there may be simply yellowish, finely scaling patches, with slightly reddened, more or less irregular borders.

The eruption also occurs upon the trunk and extremities in the form of macules, papules, and reddened patches, which by coalescence of individual lesions may become quite large. These lesions may present any degree of scaling or crusting, though there is usually a narrow, uncovered, reddened margin. The affected areas may be dry, and in form, distribution, and general appearance closely simulate psoriasis; or they may be somewhat moist and, as a result of irritation or of excessive exudation, may undergo a transformation to a condition indistinguishable from that of eczema. In most cases the yellowish color of the lesions is conspicuous, being most marked when the eruption is fading. While the dorsal surfaces of the hands and feet may be involved, it is very doubtful if seborrhoic dermatitis ever affects the palms and soles.

In the axilla and groin the eruption often begins as an erythema intertrigo, and owing to the influence of heat, moisture, and friction in these regions secreting patches are common. From these points the disease often spreads to the adjoining surfaces, the advancing margin of the eruption always being sharply outlined and usually of circinate contour.

A special seborrheide of the face has been described by Pringle,[1]

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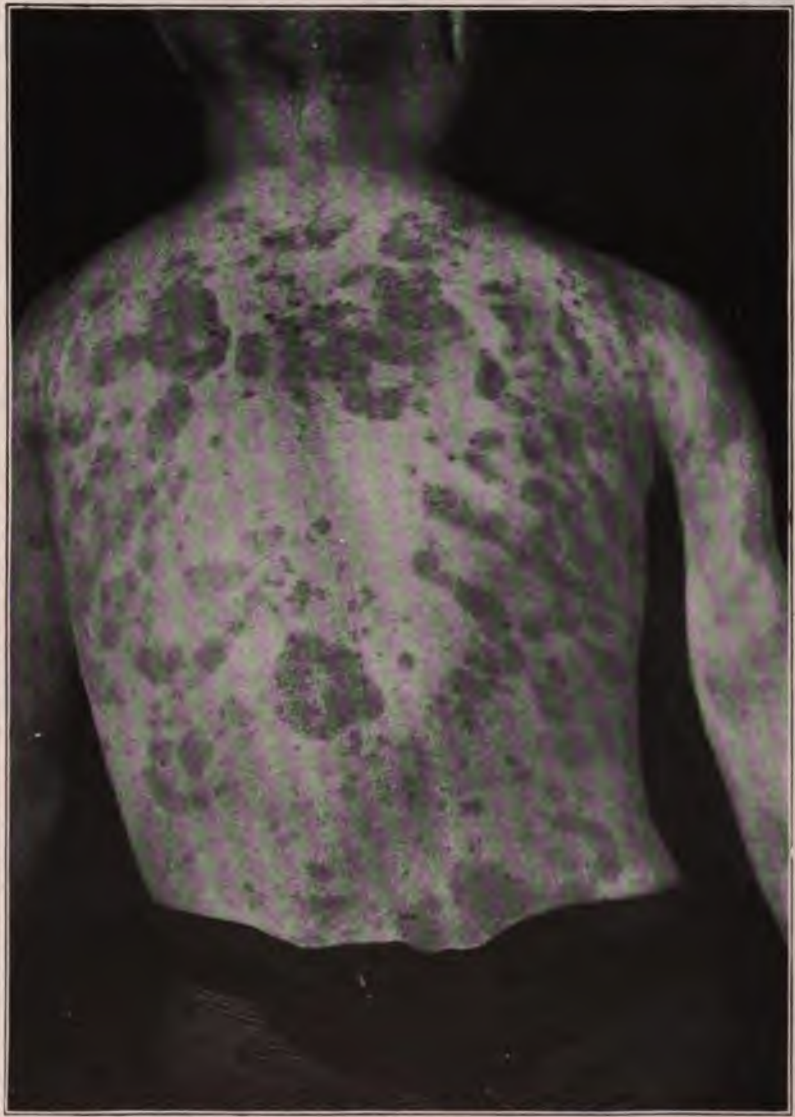
[1] Brit. Jour. Derm., 1903, xv, p. 41 (A rare seborrheide of the face).

Fox[1] and Little.[2] It is an uncommon eruption, accompanied by flushing in the region, and characterized by large numbers of red, aggregated, miliary, indolent, perifollicular papules. They may acquire a deeper tint, and occasionally present minute atrophic spots. Under the diascope a citron-yellow base is seen.

FIG. 400



Dermatitis seborrheica. (Stopford Taylor.) (Photo by Oram).

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[1] Quoted by Pringle, Brit. Jour. Derm., 1894, vi, p. 368.

[2] Ibid., 1903, xv, p. 252.

Etiology.—In his first description of eczema seborrheicum, Unna claimed for it a parasitic origin. He has described three varieties of diplococci which he found in the lesions of this disease, beside several varieties of bacilli which were occasionally present. Of these he considered a mulberry-shaped coccus, which he called the *Morococcus*, of special importance, and on occasions has produced with it, by the inoculation of pure cultures, one or more vesicles, but without reproduction of a patch of true eczema seborrhoicum. He also found Malassez's flask-shaped bacillus in the scales.

Elliot[1] reports on a bacteriological study by W. H. Merrill of 50 cases of eczema seborrheicum. In all but two cases (on which a solution of resorcin had been freely used) bacteria of some kind were found. Merrill describes two varieties of diplococci and a bacillus, all three of which were present in 31 cases, while one or two of them were found in most of the remaining cases. Twelve inoculation-experiments were made, of which seven were successful. From pure cultures of the cocci typical lesions of the disease were produced, from which in each case the special coccus was recovered and cultivated. One of these cocci was decided to be chromogenic and the cause of the yellowish color characteristic of the disease. Positive evidence of the transmission of the disease from one individual to another is difficult to get, though a history of probable contagion is obtained frequently.

Locally, heat, moisture, friction, and other forms of irritation may act as predisposing causes and favor the origin and spread of the disease. On the body it is often found in those who perspire freely and who wear wool next the skin. On the scalp it is common in those who keep the head covered much of the time. Elliot reports that the majority of his cases occurred in people who lived for the most part indoors, and that the affection is unusual among those who live largely in the open. His explanation of the greater prevalence of the disease in winter than in summer is that in the former season most people live indoors, with poorer ventilation, and bathe less than in summer.

The systemic conditions favoring the development of the disease are practically those named as predisposing causes of seborrhea.

Pathology.—Even in the mildest grades of the affection, corresponding to the condition known as pityriasis capitis, Elliot[2] found slight inflammatory infiltration about the papillary vessels and the ascending branches from the subpapillary plexus, and along the hair-follicles, while in the rete there were some vacuole-like formations in the basal layer, and a few wandering cells. In moderate grades the inflammatory infiltration extended to the subpapillary plexus, and in severe grades to the entire cutis, which was then somewhat edematous. In the rete, vacuoles were numerous and their origin could be traced to a nuclear degeneration. Many wandering cells were present, also karyokinetic figures and areas of cell-degeneration. The horny layer was thickened and easily detached from the interfollicular spaces, but densely packed in the dilated openings and necks of the follicles. The sebaceous glands were apparently normal. The coil-glands in many instances were dilated and contained cast-off epithelial cells mixed with a granular débris, while mitosis and cell-degeneration were seen frequently. Elliot found no appearance that would warrant him in believing the coil-glands to be the source of the fatty hypersecretion. Unna, on the other hand, found fat in the coil-glands, and believes them to be the source of most of the fatty secretion characteristic of the disease. He also describes an infiltration of small, free globules of fat through all parts of the cutis and rete, inside the lymph-sacs. Elliot found no evidences of such infiltration; but Ledermann announces that he has recognized it in normal epithelium.

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[1] New York Med. Jour., 1895, lxii, p. 528. A subsequent report by Merrill, ibid., 1897, lxv, p. 322, confirms these findings.

[2] Morrow's System, iii, p. 282.

Unna and Elliot agree in considering all stages of the process an inflammation of a catarrhal nature, the immediate cause of which is to be found in one or more specific microörganisms. (See also Seborrhea.)

Diagnosis.—From other forms of dermatitis and from simple eczema, dermatitis seborrheica may be distinguished by its origin on the scalp, its oily secretion and crusts, the yellowish color and sharp outline of its lesions, its tendency to spread peripherally in circinate outlines, and by its lack of marked subjective sensations.

In some forms of the disease the diagnosis from psoriasis is difficult, but the location of the lesions on the flexor rather than on the extensor surfaces, the oily character of the scales and crusts, the yellowish color, the greasy and scaly center of circinate lesions undergoing involution, and the general course of the eruption, will usually suffice to distinguish the disease.

Pityriasis rosea may present appearances similar to those of dermatitis seborrheica of the trunk and extremities. The lesions in the former disease, however, do not appear on the scalp, usually have ill-defined, frayed-out borders, and the enlarging rings present a dry, fawn-colored center, which is free from greasy scales. The affection, moreover, runs an acute course, rarely lasting more than six or eight weeks.

Lupus erythematosus occurs chiefly upon the face; it is rarer upon the scalp and body. The scales of lupus are tenacious and dry, and require scraping for their removal; the contour of the lesions is well defined, and scars are produced as the condition resolves. Erythematous lupus is far less amenable to treatment, and persistency of lesions after a faithful trial of the remedies usually effective for seborrheic dermatitis should always suggest a reconsideration of diagnosis. This fact holds especially true of lesions upon the scalp.

Trichophytosis corporis often appears upon the face, and might be mistaken for seborrhoic dermatitis; but the tendency to clear in the center as the margin advances, the distinct elevation of the active edge, and the discovery of the fungus will establish the diagnosis. The same criteria hold for eczema marginatum, in which the fungus invades the crural region; in this, however, the fading of the central portion is less pronounced than in ringworm elsewhere, and more dependence must be placed on the well-defined, elevated, advancing margin, and the demonstration of the organism.

Treatment.—Sulphur, resorcin, salicylic acid, white precipitate, and other preparations of mercury are remedies most useful in the treatment of all stages of the disease. For the earlier and dry forms, stronger and more stimulating preparations may be used, together with more frequent washings of the skin, than in the acute, moist forms, which must be treated more in accordance with the principles laid down for the treatment of the corresponding stages of eczema. For the scalp and other hairy portions of the body, lotions are usually better than ointments. The lotion recommended by Elliot, containing 3 to 20 per cent. of resorcin in equal parts of alcohol and water, is one of the best, and should be applied two or three times daily. For the dry forms of the disease a small amount of oil (preferably the oil of sweet almonds), to prevent the disagreeable drying effect of the lotion alone, may be added. Instead of thus combining the oil with the liquid, a thin ointment containing resorcin or sulphur may be substituted for or applied after the lotion. After soap-and-water washings, which should be used often enough to prevent accumulation of scales and crusts, an oily or fatty application is always desirable.

The most serviceable ointment in the majority of cases is one containing from 1 scruple to 2 drachms (1.33 to 8.) of sublimated or precipitated sulphur, 10 minims (0.66) of balsam of Peru, and 1 ounce (30.) of vaselin. Instead of sulphur, resorcin or ammoniated mercury may be used. In some chronic cases with much infiltration, sulphur, resorcin, and salicylic acid may with advantage be combined in the same ointment, while in a few instances the tars, pyrogallol, or chrysarobin may succeed after the above-named preparations have failed. In acute forms, in which the symptoms are more those of an acute eczema, pastes and ointments containing salicylic or boric acid are valuable until the acute inflammatory condition has subsided, when preparations containing sulphur or resorcin should be used.

The disease is usually more amenable to treatment than eczema, though recurrences are common.

ASTEATOSIS.[1]

Synonyms.—Xerosis. Ger., *Asteatose;* Fr., *Astéatose.*

Definition.—Asteatosis is that condition of the skin in which there is absolute or relative deficiency of the sebaceous secretion.

Symptoms.—Insufficient lubrication of the skin by its natural unguent may be either general or partial, and occur as an idiopathic or a symptomatic disorder. It is produced artificially by any agents which continually withdraw the fatty substance from the skin-surface, as in those trades necessitating the constant immersion of any part of the body in strong alkaline solutions or in waters strongly impregnated with calcium and potassium salts. As an idiopathic affection it is of rare occurrence, but it is not an infrequent accompaniment of other local or constitutional diseases, such as psoriasis, lepra, xeroderma pigmentosum and ichthyosis. In these cases the skin becomes dry, often thickened and indurated, and, as a consequence, friable, and prone to desquamation, fissures, and chaps. To the touch, the absence of sebaceous secretion is noticeable in the objective sensation produced. Asteatosis is a well-marked feature of the marasmus of old age. Some authors have described under this title the dry thickening and induration of the palm of the hand accompanied by curving of the fingers toward the plane of their flexor tendons, a condition that is occasionally to be observed in laundresses.

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[1] Varney, Trans. Sect. Derm. Amer. Med. Assoc., 1919, p. 141. Deranged Sebaceous Secretion as an Etiologic Factor in Diseases of the Skin.

Treatment.—No internal medicaments are known to have the power especially of stimulating the sebaceous secretion. None, indeed, could be capable of having such action when, as is often the case in the disorders characterized by asteatosis, there has resulted an atrophy of the sebaceous glands. For external application cod-liver oil, almond oil, lanolin, palm oil, vaseline, lard, or butter may be employed. Vaseline is in many cases to be preferred, as the other articles named are liable to become rancid after oxidation, and thus act as irritants. Elliot prefers liquid albolene or benzol. With such partial or general lubrications, however, a warm bath of soap and water should be ordered every second or third day; immediately after the bath the inunction may be repeated.

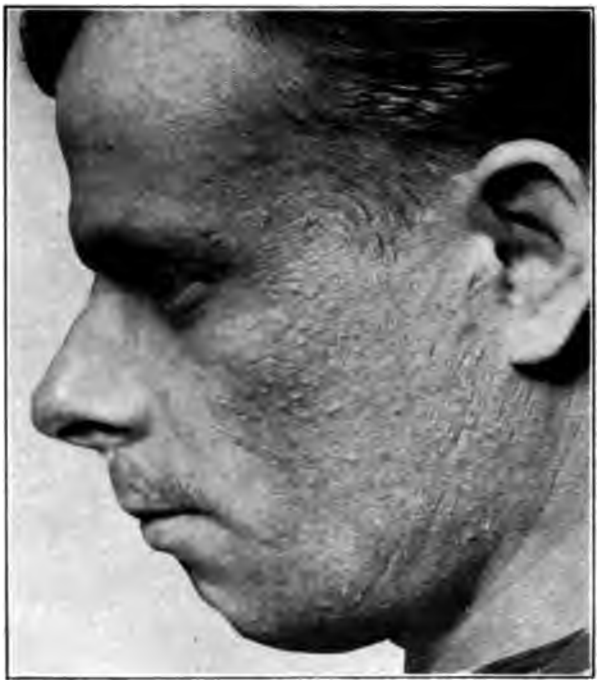
Prognosis.—In all cases in which the asteatosis is induced by agents operating externally upon the surface, a reasonable hope of recovery may be entertained after withdrawal of the cause. Persistence of the latter is likely to be succeeded by the occurrence of eczema or dermatitis medicamentosa. A complete cure can scarcely be expected when this condition is a symptom of one of the disorders already named.

MILIUM.

Synonyms.—Grutum, Strophulus albidus, Tuberculum sebaceum, Acne albida. Fr., *Acné miliaire.*

Symptoms.—Milia occur upon and about the eyelids, the cheeks, the forehead, the temples; the penis, scrotum, and corona glandis of men; and the internal face of the labia minora of women. They are millet-seed- to pinhead-sized, pearly-white, occasionally symmetrically placed, globoid masses, rarely attaining the dimensions of a coffee-bean, and showing within the epidermis as though portions of kernels of rice were lying immediately beneath a translucent layer of tissue. They occasionally project from the surface to such an extent as to resemble small-sized vesicles having milky contents. In color they are yellowish and whitish. They are often congenital, and may be recognized about the lids and temples of the newborn infant; they are also seen in middle life, when they develop very slowly, and sometimes persist for years. They occasion no subjective sensation and are commonly so insignificant as to constitute no deformity. They never degenerate by ulcerative processes, but in the course of years, when not artificially removed, are exfoliated in the natural processes of physiological desquamation. In rare instances the deposition within the milia of the salts of lime renders them as hard as cartilage (*cutaneous calculi*). These are usually of larger size and present a faint yellowish hue.

FIG. 401



Milia.

Etiology.—Milia may be of embryonic origin and occur in the newborn; they are common in infancy and early adult life, and are rare in middle life, though occasionally they develop after the thirtieth year. They are at times produced mechanically; as when the stroke of a knife-blade, accidentally or in the processes of surgery, separates one or more of the acini of a sebaceous gland from the main body. The contracting bands of a cicatrix, after destruction of tissue from any cause, may operate in a similar way with precisely the same result; and they may thus follow the lesions of tuberculosis, syphilis, erysipelas, pemphigus, and epidermolysis bullosa.

Pathology.—When a milium is incised externally, a spherical body of nearly corresponding size may be expressed, though it may require tearing from a minute pedicle below, which represents the attachment to the hair-follicle. The small mass thus extracted is seen to be a horny cyst composed of several thin envelopes, suggesting the capsules of the onion, and representing cornified epithelia which have not undergone fatty metamorphosis. In the center of the cyst is a fatty nucleus. There is never any lobular formation. Each of these horny cysts is developed in connection with the lanugo hair-follicles, distending the latter, as Unna has shown, irregularly and on one side. Virchow and Rindfleisch believed the process to represent a hyperkeratosis of the epithelium of the hair-follicles, though it is believed by others that the milium represents a retention-product of the sebaceous glands. Robinson believes that milia originate from miscarried embryonic epithelia from hair-follicles or from the mucous layer of the epidermis.

Diagnosis.—Milia might be mistaken for minute vesicles containing a milky fluid, but puncture of a lesion, with expulsion of its contents, at once discloses its character. Comedones with blackish external points, surrounded by the patulous orifice of the excretory duct and prolonged more deeply into the substance of the skin, could scarcely be confounded with milia. The most minute of the lesions of xanthoma have a yellowish color, and cannot so readily be scraped away from the subjacent tissue as can milia.

Treatment.—Milia rarely require treatment, as they are usually relatively few in number, and produce neither subjective sensation nor deformity. If desired, they may be opened with a fine milium-needle and their contents turned out, or they may be scraped off with a curette. To insure their non-recurrence, the little sac left after the operation may be entered with a needle which has been dipped in a 50 per cent. solution of chromic acid. This operation may have to be repeated in the rare cases in which the lesions exhibit a tendency to recur.

The convenient method of removing these and many similar-sized lesions of the skin is by electrolysis. With from four to six cells in the circuit, the negative pole is connected with a fine needle, which is introduced within and beneath the lesion, while the moistened sponge of the positive pole is in contact with the skin of the patient. This operation is bloodless and effectual, insignificant scars resulting.

Occasionally, milia upon the scrotum give rise to sexual hypochondriasis, which may demand attention; suggestive, rather than active, operative treatment is needed in these cases.

Prognosis.—The prognosis is always favorable.

Milium Congenitalé (*en plaques*) has been described by Crocker,[1] Hans Hebra, Wilson, and Colcott Fox as a congenital condition in which occurs a reddish-yellow patch (destitute of hair when existing on the scalp), with well-defined border and a granular surface, constituted of minute yellowish papules, with comedones at the periphery and elsewhere.

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[1] Diseases of the Skin, 3d ed., p. 1131.

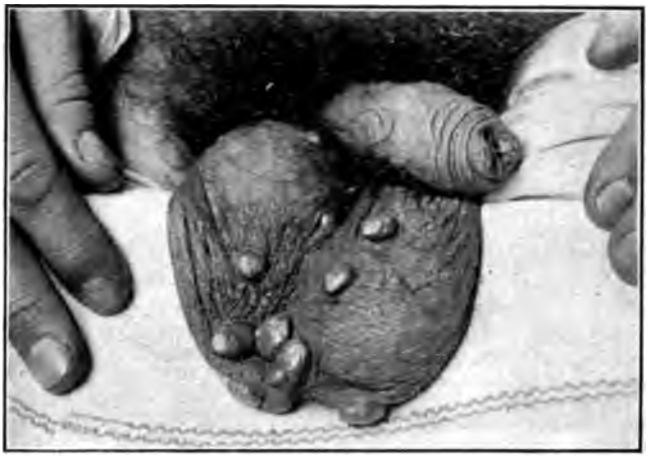
STEATOMA.

Synonyms.—Wen, Atheroma, Pseudo-atheroma, Sebaceous cysts, Sebaceous tumor. Fr., *Stéatome, Kyste Sébacé;* Ger., *Balggeschwulst, Grützbeutel.*

Definition.—This disease is characterized by the occurrence of one or several roundish, globular, elevated, soft or firm tumors, situated in or beneath the skin, and unaccompanied by subjective symptoms.

Symptoms.—Sebaceous cysts are usually of slow growth, and unattended by subjective sensations. They occur as single or multiple, elevated, occasionally flattened, fixed or movable tumors on the head, the trunk, or the genitals; and vary in size from that of a pea to a hen's egg, or larger. They are situated beneath, within, or upon the skin; usually are unattached to the deeper contiguous tissues; and develop into irregularly globular, occasionally large, button-shaped masses, covered by an integument usually unprovided with hairs. The skin covering them may be normal in hue, or unnaturally whitish from pressure; or, especially upon the bald scalp of certain fleshy men of middle years, reddened, shining, and greasy in appearance. In a certain proportion the duct remains patulous and is closed with a black, horny plug, suggesting a giant comedone. In these the semisolid, cheesy, and milky contents often emit a nauseous odor. Occasionally, a cutaneous horn develops in connection with this type. These tumors vary in size from time to time, according to the amount of escape of the contents. At times the cysts are to be distinguished only by passing the fingers through the long hairs of the scalp beneath which they are hidden; at other times they are so conspicuous, in consequence of physiological alopecia, as to occasion considerable

FIG. 402



Multiple sebaceous cysts of scrotum. Twelve years duration.

disfigurement. They vary greatly in consistency, but usually produce to the touch a certain feeling of elasticity, especially if the cyst be distended tensely. They may persist for years without producing inconvenience, save that resulting from their bulk and the consequent disfigurement; but may be attacked by inflammation, which may result in suppuration and ulceration. In rare instances in elderly people epitheliomatous degeneration may supervene in an infected sebaceous cyst.

Usually but one tumor is present, though two or more are occasionally seen.[1] As a rule, the multiple cysts are dermoid in character. These occur generally distributed, and resemble multiple fibromata or xanthoma. Such cases are recorded by Pollitzer,[2] Jamieson,[3] Maclaren,[4] and Chiari.[5]

Chalazion is a term employed to describe pinhead- to small-nut-sized cystic tumors occurring on the eyelids in connection with the Meibomian follicles, and there may be one or several. They are of slow growth, and present no symptoms other than enlargement, unless secondarily infected. By some authorities they are considered neoplasms.

Etiology and Pathology.—The cause of sebaceous cysts is not definitely known. By some they are thought to be produced in the same manner as the comedo-plug. Virchow considered them retention cysts. Török,[6] after a careful study of 26 cases, found papillæ in most of them, and concluded that the major portion of sebaceous cysts were dermoids, which confirmed the previous findings of Franke. This would place their genesis in embryonic remnants in the skin. Török also stated that such cysts contained no fat.

Dubreuilh and Tribondeau[7] found in certain cases budding occurring from primary cysts, which produced secondary cysts, and at times produced multilocular tumors. These they termed "proliferating wens."

Ehrman and Fick[8] suggest that the pathogenesis may be explained by anomalies in the life-history of the cells of the sebaceous glands, whereby they undergo horny rather than fatty metamorphosis, thus leading to retention and cyst-formation.

Histologically, the capsule is composed of fibrous tissue, which may be thin, or hard and dense, poorly supplied with connective-tissue nuclei and bloodvessels, and lined with epithelium. The contents vary in consistency and character, and may be semisolid, curdy, cheesy, and granular, or fluid, honey-like, or milky, and consist of epithelial cells undergoing fatty degeneration, fat, cholesterin crystals, and occasionally lanugo hairs.

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[1] Kingsbury, Jour. Cut. Dis., 1913, xxxi, p. 784.

[2] Jour. Cut. Dis., 1891, ix, p. 281 (Lesions resembling xanthoma).

[3] Edin. Med. Jour., September, 1873, p. 223.

[4] Brit. Med. Jour., October, 1886 (Lesions resembling fibromata).

[5] Zeitschr. f. Heilkunde, 1891, xii, p. 189.

[6] Monatshefte, 1891, Bd. xii, Nos. 10 and 11; abstr. Brit. Jour. Derm., 1891, iii, p. 365.

[7] Annales, 1910, i, Nos. 8 and 9, p. 417; abstr. Jour. Cut. Dis., 1911, xxix, p. 185.

[8] Kompendium der Speziellen Histopathologie der Haut, Wien, 1906.

Diagnosis.—Steatomata are to be distinguished from fatty tumors, the latter, however, are observed more commonly about the scapulæ, loins, buttocks, and extremities; whereas wens are very rarely found except about the scalp and neck. They lack also the peculiar "pillowy" feel of fatty tumors. Suppurating wens in the regions named may readily be mistaken for circumscribed abscesses, the result of other causes. The history of the characteristic tumor long preceding the abscess makes the distinction. Syphilitic nodes and gummata of the same parts are usually both tender and painful. Osteomata are solid growths and immovable.

Treatment.—The removal of a wen is accomplished by excision, care being taken to remove the sac *in toto*. With aseptic precautions, ablation of these lesions from any part of the body may be regarded as unattended with great risk. As the incision required for removal of the wen necessarily must extend some distance on either side of the tumor, there results a linear scar, which on the bald scalp is often a very conspicuous relic of the lesion. In consequence of the possibility of danger, many surgeons prefer destruction of a prominent section of the mass with acid or alkali, leaving the sac, after expulsion of its contents, to wither gradually, though it may then often be withdrawn with forceps.

Complete obliteration is sometimes effected by puncture, expression of the contents, and subsequent induction of artificial inflammation in the walls of the cyst by injection of tincture of iodin, ether, or other irritating fluid, as in the operation for relief of hydrocele.

Prognosis.—As a rule, a sebaceous cysts causes nothing more than disfigurement and slight inconvenience. The removal of the wall of the cyst is not followed by a return of the lesion. In debilitated and cachectic patients there may be spontaneous ulceration and sloughing, with or without surgical interference. Bryant[1] reported a carcinoma following the removal of a steatoma from the buttock of a woman sixty-three years of age. The rare occurrence of malignant change arising in elderly people is to be remembered.

COMEDO.

Synonyms.—Black-head. Ger., *Mitesser;* Fr., *Acné ponctuée, Acné comédon.*

Definition.—Comedones are collections of sebaceous mtaerial retained in the excretory duct of the hair follicle or sebaceous gland.

Symptoms.—Comedones are yellowish, grayish or blackish dots or points scattered irregularly over the surface of the skin, each point representing the end of a plug of sebaceous material lodged in one of the excretory ducts. They may project slightly above the general level of the integument, or be depressed. They commonly occur upon the face, in which situation they are found about the nose, forehead, cheeks and chin. They also occur on the front and back of the neck, chiefly the latter, and on the back and front of the body, largely on its upper portion. Occasionally they are found in other situations. The visible portion of the comedone ranges in size from pinhead to pinpoint. They are readily removed from the follicles in which they are lodged, and when removed are seen to be whitish molds of inspissated sebum, one or two or more lines in length, the exposed extremity of each comedone being dark colored through a deposit of pigment. On account of this appearance these lesions have been termed both "blackheads" and "skin worms." Comedones may be few or numerous. They are unaccompanied by subjective symptoms. The course of the disorder is chronic with occasional disappearance of lesions and development of new ones. They are frequently associated with oily seborrhea and acne. Occasionally double comedones[1] occur, in which case they have a common base with two points of exit. When expressed from the skin the plug of inspissated sebum in this variety has both

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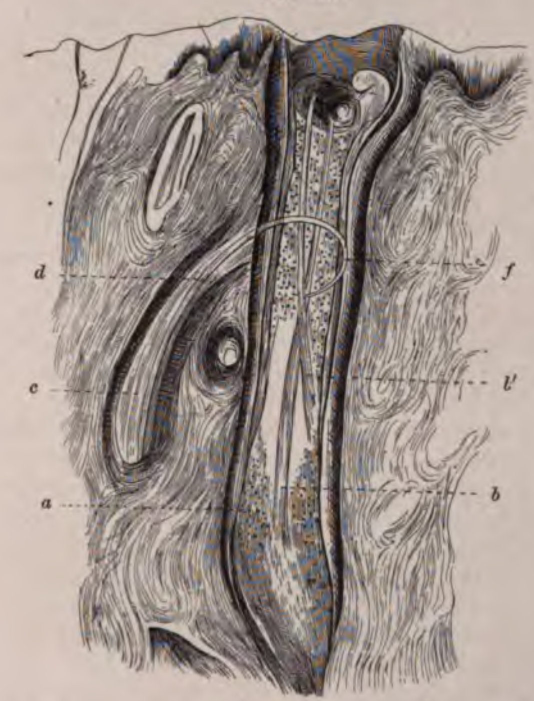
[1] Brit. Med. Jour., 1884, i, p. 1044.

Fig. 403



Section of a comedo: *a*, excretory duct of a sebaceous gland filled with a comedo; it contains also two small hairs with brush-like inferior extremities; into it opens a small hair-follicle (*c*); the contained hair (*d*), after touching the opposite wall of the duct, curves downward, at *f*. (After Kaposi.)

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[1] Ormsby, Jour. Cut. Dis., 1915, xxxiii, p. 394: "Acne vulgaris with double comedones."

extremities discolored. This type is frequently followed by marked scar formation.

Grouped comedones were first described by Thin[1] and are commonly symmetrically disposed on the forehead and cheeks. They are smaller than the ordinary variety, are not associated with acne and show no tendency to inflammation or suppuration. According to Crocker, they occur chiefly on the flush area of the face, and are commonly associated with gastric disturbance. Grouped comedones in children were first recorded by Crocker.[2] In these the lesions occur chiefly on the cheeks in infants and on the temples, forehead and occiput in older children.

The lesions are densely packed, firmer than the ordinary variety, and according to Crocker, appear to be excited by local irritation, warmth and moisture.

Cases occur in which, in addition, lesions are seen on the chest or upper part of the trunk.[3] Scar comedones, single, double or grouped have been noted in certain forms of follicular atrophy (Lang, Selhorst, and Thibierge). Large and numerous lesions of this type have been reported after kerion (Crocker) and variola (de Coquet).

Etiology.—Comedones occur most commonly at the time the pilosebaceous structures are rapidly developing. The disorder is somewhat more frequent in coarse-skinned brunettes and those having an oily seborrhea. In certain instances there seems to be a connection between the disorder and the general condition. Anemia, chlorosis, gastric and intestinal disturbances, particularly indigestion and constipation, menstrual derangements and cachexia, are sometimes predisposing causes. It not infrequently happens that when the above conditions are corrected the comedones disappear. The microbacillus of Unna and Sabouraud found in the comedone plugs is accepted by the major portion of investigators as the direct cause of the disorder. *Demodex folliculorum* (*Cf.* this section) is also found in the comedo but appears to play no role in the etiology of the disorder. Grouped comedones are believed to be due to some form of local infection, the exact nature of which is not determined. Grouped comedones occur in infants and children and are commonly due to local irritation. Dore[4] and MacLeod[5] have reported cases of grouped comedones in children produced by camphorated oil. The common use of a family shawl has been suggested as a cause by Harries.[6] Multiple comedones of the frontal region produced by brilliantine is recorded by Pernet.[7]

Pathology.—The comedo is a dense collection of concentrically arranged epithelial cells in the center of which are fragments of cells which have undergone partial fatty degeneration, dried sebum, and lanugo hairs. It is located either in the excretory duct of the sebaceous gland or in a pouch-shaped canal common to the sebaceous gland and the hair follicle. It is induced by hyperkeratosis occurring about the neck of a follicle causing retention of the fatty material which should ordinarily be thrown off. The cause of the hyperkeratosis is believed by some to be the irritation induced by the microbacillus of Unna and Sabouraud. The latter believes that comedo always follows an oily seborrhea and has the same cause. Whitfield[1] and other observers believe there is an etiological connection between this microbacillus and the comedone. The discoloration of the head of the comedo is due largely to pigment formed by a degeneration or some other change occurring in the epithelial cells. This pigment is soluble in nitric acid

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[1] Lancet, 1888, ii, p. 712.

[2] Lancet, April 19, 1884.

[3] Little, Brit. Jour. Derm., 1903, xv, p. 253; MacLeod, ibid, p. 453.

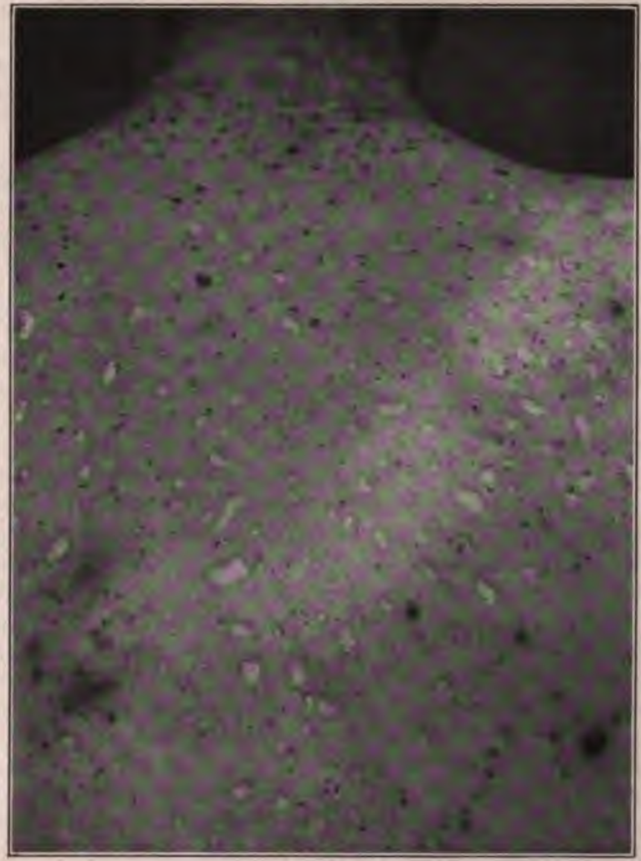
[4] Brit. Jour. Derm., 1909, xxi, p. 220

[5] Ibid., 1918, xxx, p. 181.

[6] Ibid., 1911, xxiii, p. 5.

[7] Ibid., 1916, xxviii, p. 136.

FIG. 404



Double comedo.

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[1] System of Medicine, Allbutt and Rolleston, 1911, ix, p. 8.

or hydrochloric acid and in hydrogen peroxid. Bennians[1] attributes the formation of pigment in the epithelial cells to changes induced by the growth of the acne bacillus in the situation. The double comedo is believed to arise from the merging of two glands in close proximity by pressure atrophy of the tissues which ordinarily separate them, thus forming one cavity with two ducts.

Diagnosis.—The recognition of the disorder presents no difficulty. In most cases it is apparently of secondary importance to the individual, as the acne commonly accompanying the condition is the one of chief concern to the patient. True comedones sometimes have to be distinguished from discolorations produced at the mouth of the follicles by certain proprietary preparations used for cosmetic purposes, and by the black sulphid of mercury which sometimes is formed when mercury and sulphur are applied simultaneously upon the face.

Treatment.—The internal treatment of the disorder is that described in connection with acne. The local treatment consists in the mechanical removal of the comedones as well as applications which have a specific effect on the sebaceous glands. Comedo extractors, such as described in the section on general treatment are efficient. Better results are obtained if some preliminary treatment is given the area. An ointment containing 3 to 5 per cent. resorcin applied each evening for several days preceding the removal greatly facilitates this operation.

In addition to their mechanical removal, sulphur, resorcin and the mercurials are used to advantage. Sulphur in the strength of 10 grains (0.66) to 1 dram (4.0) to 1 ounce (30.0) of equal parts of petrolatum and adeps, or in a lotion in combination with alcohol or glycerin is serviceable. Piffard suggests equal parts of sublimed sulphur, alcohol, compound tincture of lavender, glycerin, and camphor water. Another serviceable lotion consists in the following: sulphur 2 drams (8.0), camphor 1 dram (4.0), liquor calcis 4 ounces (120.0). Resorcin, in lotion or ointment form, is also beneficial. In some instances ammoniated mercury in the form of an ointment, and in others mercuric chlorid in the strength of 1 to 2 grains (0.066–0.133) to the ounce (30.0) of glycerin and rose water may be employed as an exfoliating agent.

Good results are frequently obtained by the interrupted employment of a sapo mollis shampoo. This may be used for two or three nights in succession after bathing the face with hot water, then rest for ten days and repeat the shampoo. A powder containing 10 to 15 per cent. sulphur may be dusted on after the use of the shampoo and adds to its value.

An ointment recommended by Van Harlingen[2] containing 4 parts of kaolin, 3 of glycerin, and 2 of acetic acid, with or without the addition of a small quantity of ethereal oil, is applied at night a few times in succession, the eyes being carefully protected. At the end of this time the black points of the lesions are removed and the comedones are readily extracted.

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[1] Brit. Jour. Derm., 1915, xxvii, pp. 393-436; "The relation of the staphylococcus albus and the acne bacillus to the epidermis and the excretions of the skin: with special reference to the lesions of acne vulgaris."

[2] Handbook of Skin Diseases.

Prognosis.—The disease naturally tends to spontaneous disappearance, though it may continue over a long period. Double comedones induce scars and all except those occurring in infants and children are apt to be the beginning of acne. In rare instances the comedo in elderly people is the initial process in the formation of an epithelioma.

ACNE.

Synonyms.—Acne vulgaris, Varus; Fr., *Acné;* Ger., *Hautfinne, Akne.*

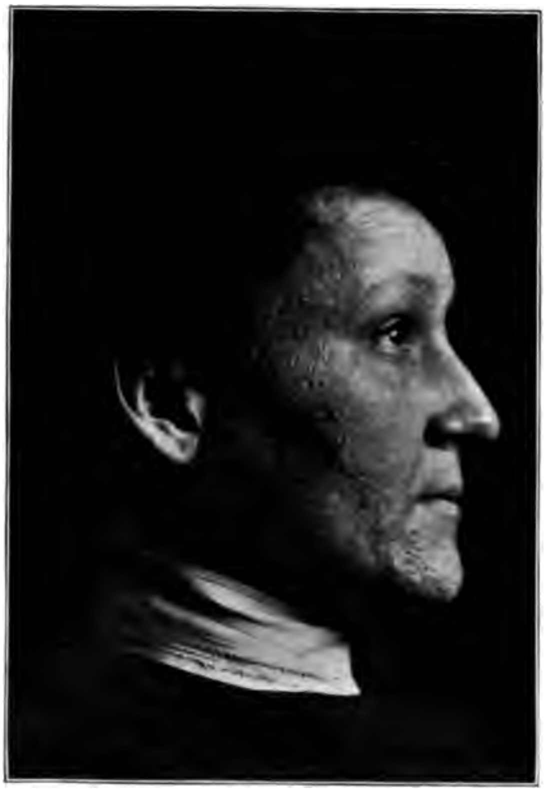
Definition.—Acne is a chronic inflammatory disease of the skin involving chiefly the pilo-sebaceous structures, characterized by papules, nodules, and pustules, and often associated with comedones and oily seborrhea.

Symptoms.—The lesions occur chiefly over the face, neck, upper part of the chest and back and over the shoulders. In exceptional cases in other areas. They may be superficial or deep and consist of papules, nodules, pustules, abscesses and mild inflammatory thickenings of the skin. In most cases there is associated oily seborrhea and comedones. While, as a rule, the eruptive manifestations are multiple in character some type of lesion predominates. The lesions may be few and irregularly distributed, or numerous and occupy a large portion of the areas of predilection. In most cases there are no accompanying subjective symptoms, though some of the lesions may be tender or painful. The course of the disease is chronic, showing periods of quiescence, and in mild cases the lesions may almost entirely disappear, only to be followed by periods of increased activity. In mild cases only a few lesions, chiefly papules and pustules originating in comedones continue with more or less activity for long periods. In more severe examples deeper pustular lesions and abscesses occur. Exceptionally several deep abscess cavities form, thereby producing lesions an inch or more in length, which may contain a teaspoonful of pus. These often remain for long periods before undergoing resolution, unless evacuated surgically. Comedones are usually present and most of the acne lesions develop from these. Not all of the comedones undergo this transformation. When the disorder is undergoing resolution, reddened and pigmented areas may remain for a time, while in some instances scar formation results, some of which exceptionally undergo keloidal changes. Several forms of the disease are described, and while many cases do not conform to these but present a combination of two or more, a study of the types explains the evolution of the lesions.

Acne Punctata is a superficial type, the lesions of which are induced by a mild inflammatory reaction occurring in the comedones, producing slight elevation and hypertrophy, in the center of which the black point of the comedone is seen.

Acne Papulosa is a form in which papules occur. It is best seen in pure form on the forehead of young subjects, where numerous small rigid papular elevations occur commonly about comedones. In other cases the papules range in size from a pinhead to pea-size or larger, and exhibit different shades of red. Not infrequently they are situated on an inflammatory base. They represent a more marked inflammatory process than the punctate lesions, the inflammation involving the tissues around the pilo-sebaceous follicle. They may exhibit in the center a comedone or the uncolored end of the sebaceous plug.

FIG. 405



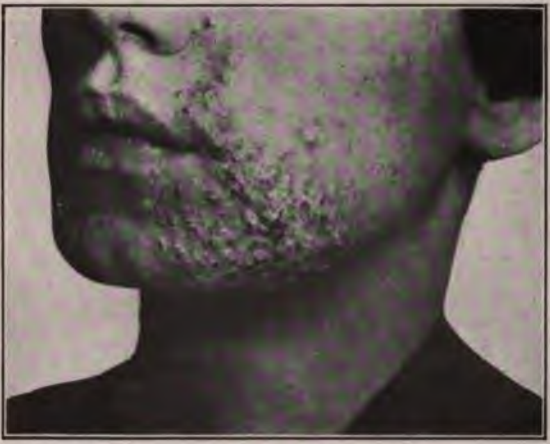
Scars following acne vulgaris (untreated).

Acne Pustolosa is that form in which the inflammatory process is of more severe grade owing to the action of secondary invading micro-organisms inducing suppuration in the center of the lesions. The pustules thus induced may be pea-sized or larger, and they may be superficial when originating in papules, or deep-seated when the suppurative process begins more deeply.

Acne Indurata is a pustular type in which the inflammatory process begins deeply, produces nodules which enlarge and progress toward the surface. The nodules are of various sizes and their color is light to dark bluish or purplish-red. They may be level with the surface, when their outline is discovered by palpation, or they may project as conical or rounded elevations. They are pus cavities covered with a thick roof wall, and may remain for long periods or undergo resolution by absorption, or may rupture and discharge their contents and heal or refill. The base of these lesions is infiltrated and continues to present this feature even when the major portion of the nodule has undergone suppuration.

Acne Atrophica is characterized by small scars and atrophic pit-like depressions resulting from the destruction of the gland tissue usually following the papular or small pustular lesions, though any of the lesions may induce the condition.

Fig. 406



Pustular exacerbation in acne vulgaris.

On the body, particularly over the back of the neck and chest, the disease not infrequently exhibits in addition to the ordinary lesions above described large, bluish-red nodules, deeply situated, sluggish abscesses, some of which may be large and associated with large comedones, many of which are double, the whole process being followed by deep and disfiguring scars and in this situation keloidal growths[1] not infrequently occur.

Acne Cachecticorum or Scrofulosorum is a variety that occurs in cachectic individuals, or those suffering with anemia, tuberculosis, or other constitutional disorder. The lesions are seen more frequently over the trunk and extremities than on the face and are indolent pinhead to bean-size, livid, purplish-red, or violaceous papulo-pustules or nodules. The lesions are usually soft, rarely indurated and contain pus and blood. They are sluggish and persistent and, upon involution, leave small scars. Colcott Fox describes acne scrofulosorum as it occurs in infants. (*Cf.* section on tuberculides.)

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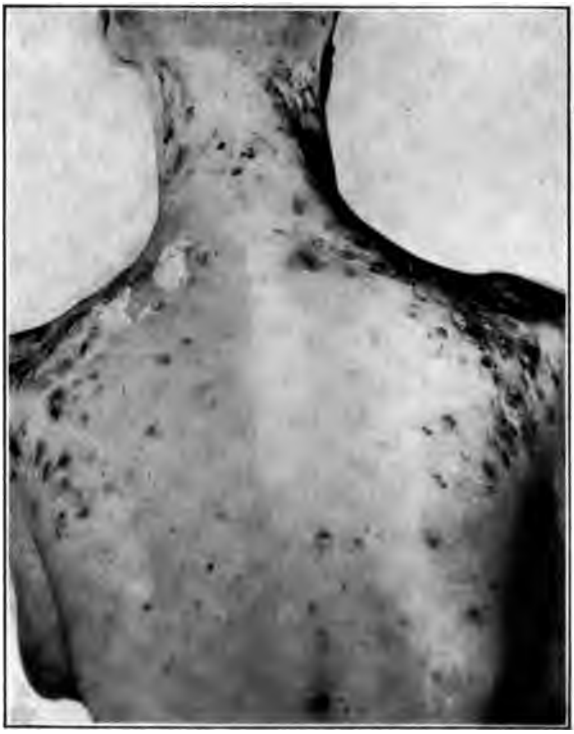
[1] Morris and Dore, Brit. Jour. Derm., 1909, xxi, p. 329. Williams, Jour. Cut. Dis., 1909, xxvii, p. 462. Little, Brit. Jour. Derm., 1910, xxii, p. 266.

Acne Agregata sue Conglobata is a form described by Reitman[1] which he found not uncommon in Riehl's clinic, occurring in men considerably after puberty. These cases were characterized by the presence of innumerable comedones, many double comedones and comedo scars together with follicular and perifollicular inflammatory infiltrations often confluent, some of which had softened and were open. They were brownish and suggested tuberculous processes. In addition, many scars were present following the resolution of older lesions.

FIG. 407



Acne cachecticorum.

Hartzell[2] described a case having on the shoulders, back, buttocks, thighs, the chest, the neck and the face, hundreds of sebaceous cysts and comedones and acne papulo-pustules. The reddened cysts were hazel-nut to English-walnut-size, and resembled fibromata in appearance. Some had reddened surfaces. On the neck were band-like keloidal lesions and inflammatory scars suggesting broken-down tuberculous glands.

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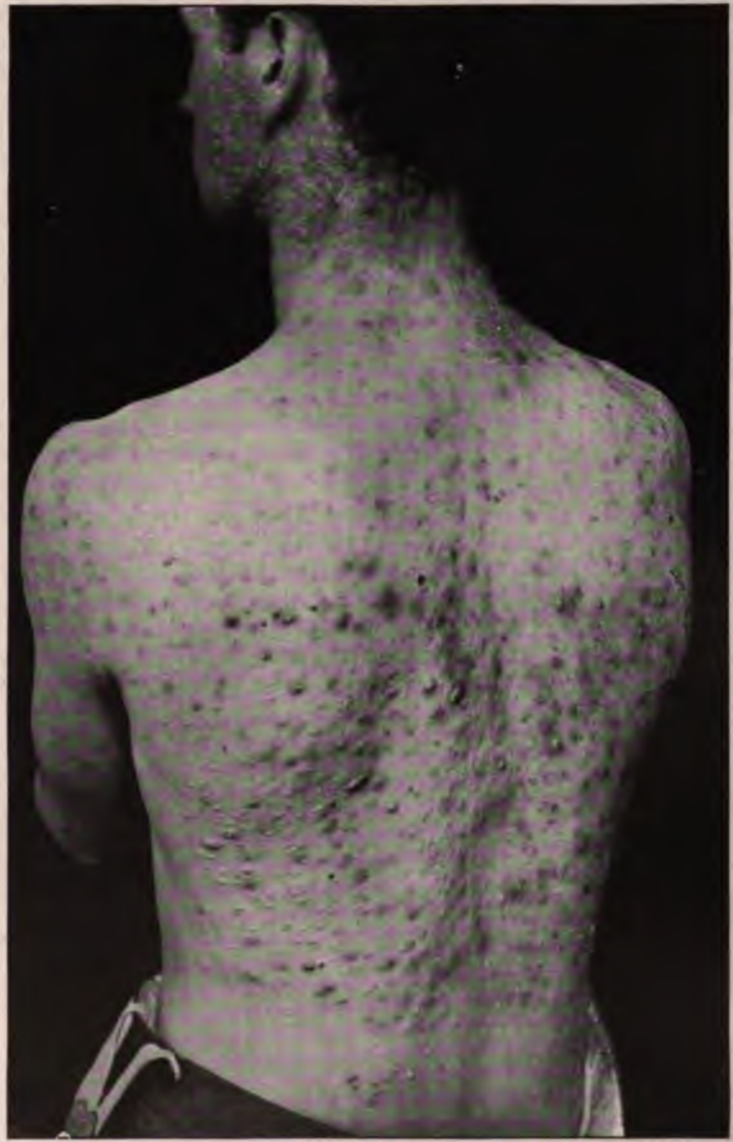
[1] Archiv, xc, p. 249, abstr., Jour. Cut. Dis., 1909, xxvii, p. 138.

[2] Jour. Cut. Dis., 1909, xxvii, p. 262.

Fig. 408



Acne-keloid of the back.

Etiology.—Acne is a more common disorder than its statistical position reveals. Many cases do not apply for relief on account of the prevailing opinion that the disease will disappear spontaneously after the individual matures. It occurs about equally in the sexes and usually during the adolescent period, though it occasionally begins at a later date. Various general and local conditions have been cited as causative factors. The consensus seems to indicate that the disease is due to the local action of different organisms appearing in a soil prepared by diverse general conditions. Among predisposing factors gastro-intestinal disturbances are often found. Pathological conditions sufficient to cause gastric and intestinal stasis followed by toxic absorption were found in 60 per cent. of acne patients examined by Ketron and King.[1] Constipation and intestinal putrefactive changes are commonly associated. Kapp[2] found indican, phenol and cresol in the urine in a large number of cases at puberty. Relative to intestinal disturbance, work recently done by Strickler, Kolmer and Schamberg[3] is important. In a series of cases of acne serological tests gave a positive reaction with the colon bacillus in a large percentage of the cases. The part played by this organism in the pathogenesis of the disease was not determined. Starches and sugars inducing fermentation have also been considered contributory factors.[4]

Disturbances of metabolism found in goiter are cited as causative by Montgomery and Culver[5] and Chalmers.[6] Indigestible articles of diet, over-eating and the excessive use of stimulants are all factors. Chlorosis and menstrual disturbances in women are contributory factors. It must be said, however, that in many individuals suffering from acne no defect in the general health can be discovered.

Bacteria.[7]—The part played by bacteria in the cause of acne and also comedo is a matter which has caused much discussion. Practically uniformly a microbacillus is present in the comedone. A similar organism is usually present in all acne lesions. In addition to this organism, *Staphylococcus pyogenes albus* and *aureus*, and a staphylococcus which cannot be recognized as identical with these, are also described. Unna, in 1893, first described a microbacillus which he termed acne bacillus. These bacilli occur as small rods, 1¼ to 1½ microns in length and ⅓ to ½ micron in width. They often have an end-to-end arrangement, like tubercle bacilli. In 1897, Sabouraud[8] described a similar organism. To this organism Sabouraud attributed the oily seborrhea as well as the comedone. He usually found a white coccus in association with the bacillus, to which organism he attributed the suppuration. In 1899, Gilchrist isolated a special bacillus in smears from 96 pustules from 56 patients, which he named *Bacillus acnes*. This bacillus is described by him as being short and thick in smears; longer, thicker, and branched in cultures. Inoculation experiments were successful, and in addition agglutination tests in the dilution of from 1 to 10 to 1 to 100 gave a positive reaction. Agglutination tests carried out by others have shown both positive and negative results. Whitfield[1] concludes that the microbacillus is concerned in the production of the comedo, but not in the subsequent acne pustule. The rôle played by the microbacillus of Unna and Sabouraud is undecided, and the question as to whether the organism described by the various investigators is identical is also undecided. Various minor differences as to its growth have caused much confusion. It has been grown by some anaërobically, and apparently aërobically by others. Again, it has been found abundantly in patients exhibiting no symptoms. Schamberg[2] found a bacillus, which was apparently identical with that of Unna and Sabouraud, in the sebaceous follicles of 90 per cent. of individuals. Haase[3] believes the organism to be similar, but observed in different phases. In 1908, Unna[4] gave to the coccus which always gives a milk-white color an important place in the etiology of acne. This observation was confirmed by Schwenter-Trachsler,[5] who reproduced comedones with the organism. A diplococcus culturally resembling *Staphylococcus albus*, but distinguished from it by agglutination tests, was isolated by Varney and Clark[6] from persistent acne-like lesions occurring after the usual acne age.

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[1] Trans. Sec. on Derm., Amer. Med. Assoc., 1916, p. 301; "Gastro-intestinal findings in acne vulgaris, especially fluoroscopic."

[2] Ther. Monats., 1907, xxi, p. 26.

[3] Jour. Cut. Dis., 1916, xxxiv, p. 166.

[4] MacDonald, Jour. Cut. Dis., 1917, xxxv, p. 90.

[5] Ibid., 1912, xxx, p. 189.

[6] University of Toronto Med. Bull., December, 1912, i, No. 2, p. 47; abstr., Jour. Cut. Dis., 1913, xxxi, p. 694.

[7] For additional information concerning research in acne, see Gilchrist, Trans. Amer. Derm. Assoc., 1902, p. 105; Jour. Cut. Dis., 1903, xxi, p. 107, with a review of work done in this field by other observers; Flemming, Lancet, London, 1909, i, p. 1035; and Brit. Med. Jour., 1910, i, p. 1382; Western, Brit. Jour. Derm., 1910, xxii, p. 6; Whitfield, International Clinic, 1909, xi, p. 183; Molesworth, Brit. Med. Jour., 1910, p. 1227; Engman, Jour. Cut. Dis., 1910, xxviii, p. 553; Lovejoy and Hastings, ibid., 1911, xxix, p. 80; Morris and Dore, Brit. Jour. Derm., 1911, xxiii, p. 311; Haase, Jour. Cut. Dis., 1913, xxxi, p. 1015.

[8] Ann. de l'Inst. Pasteur, 1897, p. 134.

Strickler, Kolmer and Schamberg obtained positive reactions with acne bacillus, and while this microörganism appeared to be etiologically concerned it was not specific, as positive reactions were obtained in other disorders.

Pathology.—In the study of the lesions in various stages of development Gilchrist[7] found in the comedo stage a widely dilated hyperkeratotic follicle filled with many layers of a keratinized substance. The sebaceous gland was pushed downward and its cells had largely lost their glandular appearance. A slight collection of lymph cells occurred below and around the comedo and there was no inflammatory reaction. In the more advanced stage the comedo was made up of horny material, polynuclear leukocytes, and nuclear detritus, and the sebaceous gland had disappeared. Some inflammatory reaction surrounded the lesions, evidenced by a collection of lymphoid cells, young connective-tissue cells, and some plasma cells. In the more advanced stage the lesion was deeper, the follicle was greatly dilated, its cells hypertrophied, and it contained masses of horny material, numerous polynuclear leukocytes, mononuclear cells and nuclear detritus. In the surrounding corium the bloodvessels and lymphatics were dilated and surrounded by many polynuclear leukocytes, which in places were massed, producing the appearance of a miliary abscess. In addition, there was a large number of plasma and variously sized giant cells. Many of the plasma cells had undergone pigmentary degeneration and pigment granules were seen lying free between the cells. Many phagocytes were noted.

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[1] Allbutt and Rolleston's System, 1911, p. 8.

[2] Jour. Cut. Dis., 1902, xx, p. 99.

[3] Jour. Amer. Med. Assoc., 1912, lix, p. 504 (a good review of research work in acne.)

[4] Med. Klin., 1908, p. 1747.

[5] Derm. Studien (Unna, Festschr., 1911, p. 311).

[6] Jour. Cut. Dis., 1912, xxx, p. 72.

[7] Ibid., 1903, xx, p. 107; "The etiology of acne vulgaris."

Diagnosis.—The recognition of the ordinary case of acne vulgaris is readily accomplished. In rare instances the pustular syphiloderm of the face is characterized by lesions which simulate those occurring in acne, but it is readily differentiated by the accompanying symptoms, some of which are usually present. In addition, the lesions are deeper and not infrequently accompanied by ulceration. Variola occasionally simulates acne, but may be differentiated by its accompanying general symptoms, although in mild cases brief observation may be necessary before a decision can be reached. Acneform lesions induced chiefly by potassium iodid, but occasionally by one of the salts of bromin, are to be distinguished from ordinary acne. These eruptions are more acute and frequently more inflammatory, and usually occur at a later period in life. The sudden appearance of an acneform eruption, or an acute exacerbation of the ordinary acne, suggests inquiry as to the possible ingestion of the iodids or bromids.

Treatment.[1]—The treatment of acne includes the general care of the patient and local treatment of the lesions. The general treatment must be guided by the findings made after a careful and exhaustive study of the general condition of the patient. Any pathological conditions found in the gastro-intestinal tract, or other organs, is to be corrected according to the principles of general medicine. The diet is, as a rule, of importance, even though positive findings cannot be made that suggest past errors. The quantity and quality of food has to be arranged to fit the specific indications. A farm laborer requires different food from a school-boy or girl, or an individual having a sedentary occupation. As a rule, fats should be reduced and starches and sweets eliminated as far as possible. Confectionery, highly seasoned food, pastry, hot breads and cakes, sugars and fried articles are excluded with advantage. Alcoholic stimulants and tobacco are generally to be prohibited.

As constipation is a frequent accompaniment, increased elimination is advisable. For this purpose a blue mass pill may be given every four or five nights, followed in the morning by a saline laxative. Or, in the beginning, small doses of calomel may be given for several consecutive nights, followed by a saline laxative the following morning. If the constipation cannot be overcome by proper regulation of the diet, then some laxatives may be continued. Some preparation of cascara is usually recommended. In certain cases a modification of Startin's acid mixture, such as the following, may be employed:

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[1] For a discussion of the treatment of acne before the American Dermatological Association, see Trans., 1902, p. 119; and G. H. Fox, Jour. Cut. Dis., 1903, xxi, p. 136.

℞—Ferri sulph.,		grs. v;	33
Tinc. cardamom. co.,		ʒj;	4
Magnes. sulphat.,		℥ij;	60
Acid. sulphur. dil.,		ʒij;	8
Sodii chlorid.,		ʒj;	4
Aq. dest.,	ad	℥viij;	240

Misce et filtra.
Sig.—A tablespoonful in a tumblerful of water before breakfast.

The mineral waters, such as Hathorn, Karlsbad, Hunyadi János, Racoczy, of Kissingen, taken before breakfast each morning are valuable aids. Milk of magnesia in dessertspoonful doses with water before meals, or a mixture of rhubarb and bicarbonate of sodium are serviceable. Salol in a dosage of 5 grains, or ichthyol in a dosage of 3 grains after each meal are recommended for some patients for their effect on the intestines. To promote further elimination water should be taken freely between meals, but iced drinks of all sorts are to be avoided. In certain instances the bitter tonics, dilute hydrochloric acid, pepsin and other agents commonly used where there is digestive disturbance are of service.

An important part of the treatment in every case of acne consists in proper bathing. A cool bath followed by a good rub should be taken each morning. Salt may be added to the water if desirable; this, however, is not necessary. A warm bath with soap is suggested twice weekly for hygienic purposes, but hot baths are to be avoided. The muscular tone should be improved by outdoor exercise and a proper amount of fresh air. The general treatment of the disease includes that specifically indicated by each individual case. Anemia, chlorosis, pelvic disease in women, and the various gastro-intestinal disturbances have to be managed according to the pathological findings.

Lindeman[1] recorded the rapid disappearance of a long standing and severe acne in two patients following blood transfusion employed in one instance for tuberculosis, in the other for gas poisoning.

Local Treatment.—The removal of comedones with the proper evacuation of the contents of pustules is important. The directions for removing comedones are given in the section devoted to that subject. Pustules should, as a rule, be opened with a needle or a narrow-bladed bistoury. The contents of the lesions should be pressed out with great care, as undue trauma aggravates the disorder. It is well before the lesions have been opened to bathe the face with hot water and after the pus has been evacuated the face should be thoroughly bathed with a hot antiseptic solution. Repeated operations of this sort produce marked improvement and if particular attention is given to comedones the development of acne lesions is greatly limited.

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[1] Jour. Amer. Med. Assoc., 1919, lxxiii, p. 896; Critical periods in disease treated by blood transfusion.

Of the local remedial agents employed in acne, sulphur heads the list. This may be used in the form of an ointment, lotion or powder. It is used in a strength of 2 to 6 or 8 per cent. in ointments. These are of more value when an animal fat, such as lanolin or adeps, constitutes at least 50 per cent. of the base. Some sulphur lotions follow:

℞—Sodii borat.,		ʒij;	8	
Sulphuris loti,		ʒiij;	12	
Glycerin.,		ʒvj;	24	
Aq. dest.,	q. s. ad	℥vj;	180	M,

Sig.—Shake well and apply freely, leaving a thin film of powder over the face.

℞—Sulphur precip.,	ʒij;	8	
Glycerin.,	ʒij;	8	
Alcoholis,	℥j;	30	
Liq. calcis,	℥j;	30	
Aq. rosæ,	℥ij;	60	M.

Sig.—Shake the vial before using.

Another useful lotion is the following formula:

℞—Camphor.,		4	
Sulphur precip.,		8	
Liq. calcis.,	q. s. ad	120	M.

Sig.—Shake and apply.

Vleminckx's solution may be employed in a strength of 15 to 60 drops in a tablespoonful of hot water and gently rubbed in.

Resorcin, next to sulphur, is probably the most valuable remedy employed in the treatment of acne. It may be used in a strength of 2 to 10 per cent. in ointment form, or incorporated in the lotions above suggested in place of sulphur. Ichthyol is somewhat similar in its action to sulphur and is frequently of much service. It may be used in lotions or ointments. The discoloration it produces is readily removed, as the preparation is soluble in water. One of the most valuable methods of local treatment consists in first stimulating the sluggish skin until slight dermatitis is produced, after which soothing applications are made until the reaction has subsided. By thus alternately stimulating and soothing the diseased portion of the skin distinct improvement occurs in a relatively short time. To accomplish this the patient is directed to bathe the face with hot water for several minutes on each of three or four successive nights. During the bathing a shampoo of the tincture of sapo mollis or other soap may be employed. After rinsing and drying the face an ointment containing sulphur or resorcin, or both, in a strength of 5 grains (0.33) to 1 dram (4.0) to 1 ounce (30.0) of equal parts of adeps and vaseline, or lanolin and cold cream salve is applied. In the morning the face may be washed with hot water followed by cold. When moderate reaction occurs, which will be evidenced by a feeling of stiffness, or the appearance of redness and scaling, this treatment is suspended and soothing applications, such as the zinc oxid and aqua calcis lotion, or the calamine lotion, or a soothing ointment are substituted. After the subsidence of the reaction the stimulating treatment is repeated. As improvement occurs the vigor of the treatment may be lessened, the shampoo discontinued, later the hot applications, and finally reducing the strength of the sulphur and resorcin ointments.

In certain instances exfoliation of the epidermis is required. This may be accomplished by the employment of a stimulating paste such as is recommended by Lassar. This contains 1 part of betanaphthol, 2½ parts each of vaseline and sapo mollis, and 5 parts of precipitated sulphur. This is spread over the skin and allowed to remain for fifteen to twenty minutes and then wiped off, when the surface is dusted with a simple powder. Soothing applications may be used to advantage before the second application.

Vaccines.[1]—Since the introduction of this method of treatment, a very large number of cases have been treated by it all over the world. The results have been at once pleasing and disappointing. The principle of the treatment appears to be sound, but the technic of its employment has not been worked out. The best results recorded are those by Engman,[2] who employs the following technic: Using a vaccine prepared with the acne bacillus alone, he employs as an initial dose three million. On the third day the comedones are expressed, all lesions opened, and hot applications made to produce local hyperemia. The latter are continued until the fifth or seventh day, when new lesions begin to appear, signifying a stage of depression, which is the indication for a second injection. From three to five million are then given. After several doses new lesions cease to appear. If after a few injections new lesions continue to appear, after the third day the dosage is increased to from seven to ten million, but this is rarely necessary.

Gilchrist[3] in the beginning used from one hundred to two hundred million, but finding this too large a dose has since decreased the amount. He now recommends that the initial dose be ten to twenty million, after which one should wait for the negative phase, which occurs anywhere from the third to the eighth day and is indicated by the appearance of new lesions. When these subside, another injection is given, and so on for three or four injections, after which a period of rest is suggested. When relapses occur, one or two injections a week apart are given. He further states that there is no doubt about the great value of *Bacillus acnes* vaccine in the inveterate nodular type of acne, in which the bacillus exists in pure culture.

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[1] For results and technic, see the following papers: Flemming, Lancet, 1909, i, p. 1035; Lassueur, Annales, July, 1910, p. 377; Engman, Interstate Med. Jour., xvii, No. 12; Western, Brit. Jour. Derm., 1910, xxii, p. 6; Lovejoy, Amer. Jour. Med. Sci., May, 1912, cxliii, p. 693; Whitfield, Trans. XVII Internat. Cong. of Med., London, 1913, Sec. 13, Part 1, p. 193; Smiley, Jour. Amer. Med. Sci., 1912, lviii, p. 1274. Whitfield, Brit. Jour. Derm., 1919, xxxi, p. 79. Sequeria and Western, ibid., p. 83, and MacLeod and Topley, ibid., p. 90. Vaccines in the Treatment of Cutaneous Diseases.

[2] Jour. Cut. Dis., 1910, xxviii, p. 553.

[3] Ibid., p. 568, and Trans. XVII Internat. Cong. of Med., London, 1913, Sec. 13, Part 2, p. 405.

Morris and Dore[1] state that they can claim nothing more for vaccine therapy in acne than that it is a useful adjuvant of ordinary forms of treatment. In MacLeod's[2] experience, vaccine treatment in acne has been disappointing. The major portion of investigators have concluded that vaccine made from the acne bacillus is indicated in the lesions in which suppuration is not yet actively progressing; while vaccine made from either the particular staphylococcus found in these cases or the ordinary *Staphylococcus albus* or *aureus* should be employed when that stage is reached. In a large personal experience, vaccine therapy has shown both brilliant and negative results. Improvement follows sufficiently often to demonstrate the value of the method when a proper technic shall have been worked out. It is, therefore, recommended in its present state as one of the methods of treatment to be employed in selected cases and in association with older, well-recognized procedures.

Radiotherapy in acne still holds a prominent place, notwithstanding the fact that a large number who formerly used this method have given it up. With the present control over dosage, there is no reason why it cannot be given entirely within the bounds of safety. It is recommended only in selected cases, and in these should be given in moderate dosage, and only for reasonable periods of time. Sufficient treatment to produce a dermatitis or a subsequent telangiectasia, or atrophy, should never be employed, and is not necessary to achieve the object of the treatment, which is the elimination of the disease.

Prognosis.—The major portion of patients, even when untreated, finally recover. Appropriate treatment facilitates this end. It also lessens the amount of scarring in those prediposed to this unfortunate condition. A certain amount of scar formation necessarily occurs in severe cases, and the subsequent development of keloids is seen as a rare sequel. It must be remembered that the disease is chronic, and that relapses are apt to occur in spite of treatment.

Acne Rosacea.—**Synonyms.**—Rosacea, Gutta rosea, Telangiectasis faciei, Nevus araneus, "Brandy-nose," "Copper-nose." Fr. *Acné rosacée, Couperose;* Ger., *Kupferrose, Kupferfinne.*

Definition.—Acne rosacea is a chronic disease of the face characterized by hypertrophy and telangiectasia, often associated with acneform lesions, and terminating in rare instances with marked hypertrophy of the tissues, particularly of the nose.

Symptoms.—The disease occurs exclusively on the face and is usually most marked over the nose and contiguous regions, although in some instances the whole face, forehead and chin may be invaded. Early there occurs erythema, diffuse or in localized patches, which is transient and induced, or made more marked by exposure to cold or heat, digestive disturbances, or the ingestion of hot liquids, such as soup, coffee or tea. The color of the skin in the affected areas varies from bright to dull red, or may even be purplish. The redness may last for but a few hours at first, but later, as recurrences continue it persists longer and finally becomes permanent. During this time the bloodvessels finally become dilated and new ones develop until telangiectasia of varying grades becomes a part of the process. This is more marked on the nose, especially on the alæ, but develops also upon the face. Under pressure with the diascope the color disappears, and while the area appears inflammatory the surface is cool rather than hot. There is often an associated oily seborrhea and the orifices of the sebaceous glands may be patulous or plugged with dry sebaceous secretion. Small red papules are associated and often the ordinary lesions of acne, exhibited as nodules and pustules, are added. This may begin coincidently with the early erythema, or may not occur until the redness is permanent. In the beginning they are usually few in number, but later may become numerous and add greatly to the disfigurement. Occasionally the process is initiated with the appearance of small, red patches accompanied by moderate scaling. Frequently a mild seborrheic dermatitis is added, and in these cases the skin is more irritable and sensitive to external influences. Occasionally an associated conjunctivitis[1] or keratitis[2] accompanies the cutaneous symptoms, which persists until the latter have been eradicated. A fully developed case exhibits all the features above described, and may continue with periods of lessened activity indefinitely. Exacerbations occur at the menstrual period in women, and in both sexes on exposure to outside irritants and gastro-intestinal disturbance, functional or organic.

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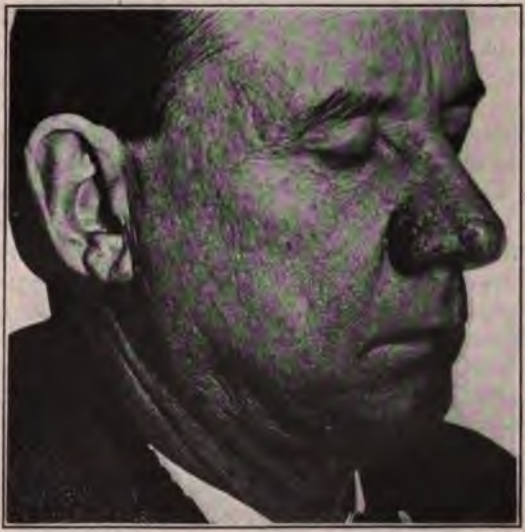
[1] Brit. Jour. Derm., 1911, xxiii, p. 311 (with bibliography).

[2] Trans. XVII Internat. Cong. of Med., London, 1913, Sec. 13, Part 2, p. 423.

FIG. 409



Acne rosacea showing dilated vessels.

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[1] Little, Brit. Jour. Derm., 1908, xv, p. 265.

[2] Schamberg, Jour. Cut. Dis., 1913, xxxi, p. 504.

Only exceptionally the disorder continues to what is termed the third stage. This is characterized by hypertrophy of the tissues, particularly of the nose, which becomes enlarged, the glandular openings become conspicuous, oily seborrhea is marked, and the bloodvessels are increased in number and size. The nose presents a dull red or purplish hue, which renders it conspicuous. Coincidentally the tissue in the immediate vicinity and on the chin may be thickened and the acneform lesions of the earlier stages be present over other portions of the face. In exceptional instances marked hypertrophy of the tissues of the nose occurs, producing lobulated masses varying in size from a pea to a cherry. Rarely pendulous masses are produced. These lesions may be limited in number or numerous and closely packed, and when highly developed a markedly disfiguring condition results, to which the term *rhinophyma* is applied.

The course of the disease is chronic and in most instances continues in the milder phases above described. As a rule, no subjective symptoms are present. Occasionally, mild itching and tenderness of the skin occur.

Under the title *rosacea acuminata*, Crocker[1] described a rare form consisting of minute or pinhead-size convex, red papules, sometimes presenting a sero-pustular apex, occurring chiefly on the cheeks but also over the forehead and lower portions of the face. The lesions may be few or numerous, may present no particular grouping, and are not accompanied by the congestion seen in other types. Several examples have been recorded by English observers. Galloway and Goadby[2] isolated an unusual bacillus from this type.

Etiology.—The disease occurs more frequently in women than men and usually after the twenty-fifth year. Stelwagon says that this is more apparent than real, owing to the fact that women more frequently apply for treatment. Extreme cases are more frequently seen in men. Gastro-intestinal disturbances are important etiologically. This may be induced by errors in diet, such as the excessive use of meats, sweets or alcohol. The connection between gastric and hepatic disease and rosacea is well shown in the cases of chronic alcoholism, where inflammatory changes in the stomach and liver are not infrequently associated with dilated vessels on the nose and face. Similar conditions induced by articles other than alcohol will produce the same results. Improper assimilation of food and lessened elimination through constipation produce toxic substances which are factors. In women functional or organic disease of the generative organs act as predisposing as well as contributory causes. Rosacea is usually more active at the menstrual period, even in the absence of demonstrable pathological conditions. Local inflammatory conditions of the nasal mucosa, including the pustular folliculitis seen about the hairs near the nasal orifices, induce hyperemia and congestion of the vessels in the vicinity and these are causative in some cases. External irritation in those much exposed to the wind, such as teamsters, cab drivers, sailors, and others, acts similarly.

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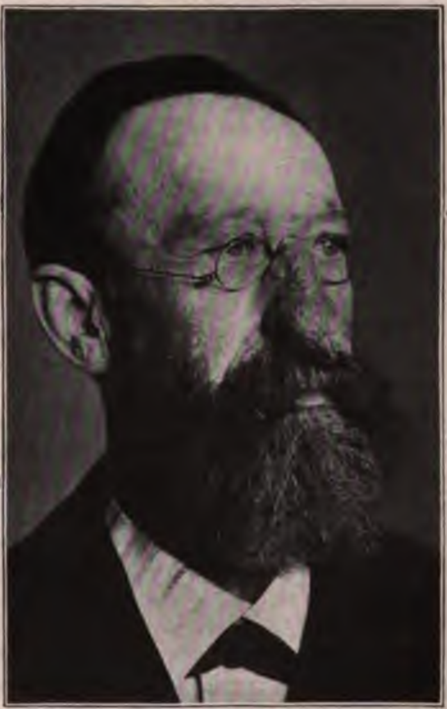
[1] Page 1149.

[2] Brit. Jour. Derm., 1908, xx, 408.

Pathology.—In the early stages vascular dilatation is found which early is temporary, but later becomes permanent. The vessels of both the deep and superficial layers of the corium are involved. Inflammatory changes are seen about the enlarged vessels and sebaceous glands, indicated by a cellular infiltration of moderate grade. In the nodules of the later stage there occurs a marked hypertrophy of the sebaceous glands and fibrous connective tissue. In some instances this hypertrophy predominates in one or the other of these tissues. The vessels are dilated sometimes to a degree suggesting angiomata. Cellular infiltration occurs about the vessels and sebaceous glands in dense foci throughout the corium, and among these many plasma cells are seen. The histology of the acneform lesions is said by some to be similar to those described in lesions of this type in acne vulgaris. Unna believes the process is seborrheic and views the histological findings differently.

FIG. 410



Rhinophyma.

Diagnosis.—The important features are erythema, telangiectasia, limitation of the disorder to the central portion of the face and nose, the age of the patient, absence of subjective symptoms and the common association of acneform lesions. In erythematous eczema there is more infiltration and scaling, the process is more actively inflammatory, and itching is usually a conspicuous symptom. Seborrheic dermatitis is likely to be less limited in its distribution, greasy scaling is present, subjective symptoms may be more marked, and telangiectasia and acneform lesions are absent. The nodular syphiloderm is distinguished by the presence of ulcers and scars, asymmetrical arrangement of the lesions and, as a rule, by the history of their involution. The nodules of lupus vulgaris are brownish-red, soft and scale covered, and in long-continued cases scar formation results. In addition, the disorder usually begins in childhood. Lupus erythematosus is distinguished by the presence of atrophy and scar formation and absence of all lesions of acneform type. Acne vulgaris occurs at an earlier period of life, is unaccompanied by the hyperemia of rosacea, the lesions are apt to be more widespread and comedones are usually abundantly present.

Treatment.—The internal treatment depends upon the general findings. Any disorder connected with the gastro-intestinal tract should receive appropriate treatment. Likewise, in women, derangements of the generative organs should be treated according to the indications presented. The diet should be simple, and alcohol, hot liquids, including soup, tea and coffee, are to be avoided, as well as sweets, highly seasoned articles, pastries, and other things capable of inducing congestion of the face. In addition, the face should be protected from outside irritants. Elimination is to be promoted by the employment of mercurial and saline cathartics. The internal treatment is limited chiefly to the general indications of individual cases. The so-called bitter tonics, including nux vomica, are frequently given in addition to hydrochloric acid and pepsin. The only remedial agents given directly for the relief of the disorder are ichthyol and ergot. The former is suggested in 3-grain doses three or four times daily, and appears to be of some service. The latter may be used in the form of fluidextract, or the alkaloid ergotine may be used in a dosage of 1 grain after each meal. This also is of some value and is supposed to lessen the facial congestion.

Local Treatment.—In the early stages a soothing lotion containing zinc oxid and aqua calcis, such as the following, is useful:

℞—Glycerini,		ʒiss;	6	
Talci,		ʒiij;	12	
Zinci oxidi,		℥ss;	15	
Liquor calcis,				
Aqua rosæ,	āā. q. s.	℥viij;	240	M.

This may be applied during the day and in the evening, and an ointment containing 2 per cent. salicylic acid, resorcin or sulphur, may be used before retiring. The following lotions are also of value and should be used for varying lengths of time. In most instances it is well to apply one until a moderate reaction occurs and then suspend its use until this reaction has subsided. During this time soothing applications may be employed. Upon the subsidence of the reaction the active preparation should again be used. A valuable lotion containing sulphur and camphor is as follows:

℞—Camphoræ,		ʒj;	4	
Sulphuris precip.,		ʒij;	8	
Liq. calcis,		℥iv;	120	M.

Van Harlingen recommends:

℞—Camphoræ pulv.,		grs. v;		33	
Tragacanthæ pulveris,		grs. x;		66	
Sulphuris precipit.,		ʒj;	4		
Liq. calcis,					
Aq. rosæ,	āā	℥j;	30		M.

Duhring's lotio alba:

℞—Zinc sulph.,		ʒj;	4	
Potas. sulphuret.,		ʒj;	4	
Glycerin,		ʒiij;	6	
Liquor calcis,	q. s. ad	℥iv;	120	M.

Liquor calcis sulphuratæ (Vleminckx's solution) is also valuable, well diluted at first and gradually increasing in strength:

℞—Calcis vivæ,	℥ss;	15
Sulphur. sublimat.,	℥j;	30
Aquæ,	℥x;	300

M.—Boil down to 180 c.c. and filter.

Occasionally ointments may be necessary. Sulphur, resorcin, salicylic acid or ichthyol may be employed in a strength of 2 per cent. In the cases showing a large number of acneform lesions with thickening of the skin, associated with oiliness, the use of sapo mollis as a shampoo, followed by an ointment containing sulphur or resorcin may be employed in the same manner that it is in the treatment of acne. (*Cf.* this section.) When the inflammatory element is well under control the telangiectatic vessels may be removed by means of electrolysis. The technic of this operation is that described in connection with the removal of superfluous hair (which see), except that the needle instead of being inserted in the hair follicle is inserted into the individual vessels. As a rule, trunk lines can be detected and if the needle is inserted into these the outlying branches are affected simultaneously. The current should be moderate and the needle left in position only until the reddened vessel becomes white and then gradually withdrawn without interrupting the current, which procedure seals the opening made by the needle, and prevents bleeding. Great improvement is accomplished by removal of these vessels. In the late stages the nodules may be removed by multiple scarification, electrolysis, surgical excision or decortication.[1]

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[1] Heineck, Jour. Amer. Med. Assoc., January 10, 1914; "Rhinophyma successfully treated by decortication."

Radiotherapy is the most valuable method for local use. There are a few cases that will not respond to this treatment but there are some recurrences.

Prognosis.—In the milder forms the disorder can ordinarily be entirely eradicated. Great improvement can be accomplished, even in the severe grades. The disease, however, may continue, in spite of treatment, over a period of years.

ACNEFORM ERUPTIONS.

Acne Artificialis.—Acneform eruptions are not infrequently seen following the administration of the iodids or bromids. These lesions commonly occur in the areas selected by acne vulgaris. Other types of lesions occur in addition. (*Cf.* section on drug eruptions.)

Several substances applied to the skin induce acneform lesions; among these may be mentioned tar, which occurs both among workers in tar and in those subjected to the action of this substance for the relief of their cutaneous disorders. Pinpoint to pea-sized reddish-brown papules form, at the apex of which is a perceptible, minute, blackish point produced by the lodgment of a particle of tar in the orifice of a sebaceous follicle.

Acneform lesions occurring on the abdomen, thighs and forearms are seen in twine workers, due to the action of the oil used to preserve the twine from being cut by insects. Such a case was recorded by the author in 1906[1] and at a later date a large group of these cases was examined in a twine factory, where it was noted that only those who came in contact with the oil were affected. Similar cases are reported by Anthony and Pusey.[2] Workers in chlorin[3] and paraffin[4] not infrequently have a widespread and severe acneform eruption situated on the trunk and extremities rather than on the face and other areas chosen by acne in its ordinary forms.

The management of these cases consists chiefly in prophylaxis. In a group seen among twine workers the disease was terminated and its recurrence prevented by thorough washing daily with soap and hot water after removing the clothes which were saturated with oil.

Acne Keratosa.—Under this title Crocker[5] reports four cases in women, in whom there appeared on the face, chiefly about the angles of the mouth, but also in other situations, firm, painful, inflammatory papules, succeeded by pustules and crusts. From the center of these lesions could be expressed short, soft, or horny plugs, which were evidently formed in the sebaceous glands or hair follicles. On removing the plugs, the lesions healed slowly, in many instances leaving a scar.

In a patient whose case was demonstrated before the Chicago

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[1] Jour. Cut. Dis., 1906, xxix, p. 82.

[2] Ibid., 1908, xxvi, p. 426.

[3] Lehman, Archiv., 1905, lxxvii, p. 265; bibliography.

[4] Schamberg, Jour. Cut. Dis., 1915, xxxiii, p. 221; "Severe acne in the paraffin worker." Case demonstration.

[5] Brit. Jour. Derm., 1899, xi, p. 1.

Dermatological Society in 1914 by the author, the presence of the sebaceous plug was a source of great irritation and there occurred an irresistible desire to work with the lesion until the plug could be removed, after which all irritation subsided. Other cases have exhibited similar symptoms. In all cases the disorder is persistent. In one of Crocker's cases it continued for forty years. These cases closely resemble the group described under the title "Neurotic Excoriations." (*Cf.* this section.)

Treatment, including radiotherapy, has only been temporarily beneficial.

Acne Urticata.—Acne urticata is described by Kaposi, Touton, Löwenbach,[1] and others, as occurring on the scalp, face, and other portions of the body. The primary lesion, which is preceded by itching and burning, is a small wheal which enlarges to the size of from 6 to 12 mm., when the center becomes paler and depressed and shows a vesicle, which dries into a crust. The crust falls, leaving a small scar, which in time becomes depressed and shining white. The full development of a wheal requires from four to six days.

The histopathology studied by Löwenbach showed a superficial inflammatory condition exhibited as edema of the papillary layer of the corium with dilatation of the bloodvessels and some cellular infiltration. Adamson classed these cases with neurotic excoriations. (*Cf.* this section.)

Acne Varioliformis.—**Synonyms:** Acne frontalis, Acne rodens, Acne necrotica, Acne atrophica, Folliculitis varioliformis, Necrotic granuloma, Impetigo rodens. Fr., *Miliaire scrofuleuse, Folliculite cicatricielle nécrotique.*

Symptoms.—Acne varioliformis is characterized by the occurrence over the brow, scalp, or other regions, of discrete, exceedingly indolent, reddish-brown, papulo-pustular, often umbilicated, lesions, which become covered with crusts, and eventually leave depressed, superficial scars, resembling those of smallpox. This disease is not to be confounded with that to which Bazin and other French writers once gave the name *Acne varioliforme,* viz., *Molluscum epitheliale* (*Molluscum verrucosum* of Kaposi).

The disease is relatively rare, and may be characterized by the development of few (but one or two) or very many lesions. In some instances the peripheral extension of a single papulopustule may produce a narrow, annular, seropurulent chamber with a depressed firm center. There is commonly a well-marked coincident seborrhea. Many of the lesions are traversed by lanugo-hairs. The subjective sensations are slight; at times there is itching. The disease tends to recur and is exceedingly chronic in course.

In exceptional cases the disorder occurs in other regions than those named above; for example, over the dorsal and sternal aspects of the trunk (where large scars are apt to follow),[2] about the nose, and within and about the concha of the ear, the interscapular region, and the extremities. In one of Dr. Hyde's patients (the subject of the accompanying illustration), the disease left very disfiguring scars on the right ala of the nose. In some cases the affected regions are invaded so thickly that the resulting scars produce a cribriform aspect in the integument. Occasionally, the arrangement of the lesions is linear or circinate.

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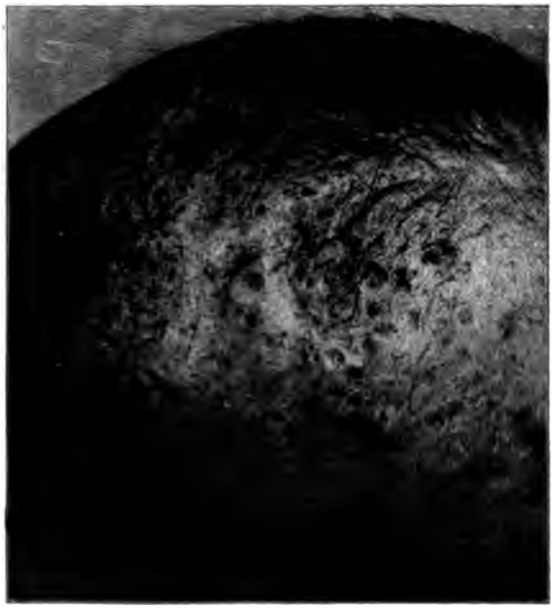
[1] Archiv, 1899, xlix, p. 29.

[2] Fox, Brit. Jour. Derm., 1909, xxi, p. 118; Little, ibid., 1913, xxv, p. 362.

The variations displayed are exceptional, but worthy of note. Severe confluent, serpiginous, and very extensive developments of the malady may be seen. According to Boeck, the hue of the papulopustule is due to minute capillary hemorrhages, which later become invisible in consequence of tumefaction of the overlying integument.

Fig. 411



Acne necrotica.

Etiology.—The sexes are represented nearly equally among the subjects of the disease, who are, as a rule, in or near middle life. Gastro-intestinal disorders are believed to have some etiologic relation to the disorder. The causes of the disease are obscure, but that its origin, as with necrotic granuloma and folliclis, is in part microbic, is well-nigh established.

Pathology.—Fordyce[1] and Sabouraud state that the disease begins in the upper part of the hair follicle, from which point it extends to the entire follicle and to the sebaceous gland. Various microörganisms are found in the lesions, but the active agent is apparently a staphylococcus. Sabouraud[1] believes the disease is always preceded by seborrhea, due to infection with his microbacillus.

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[1] Jour. Cut. Dis., 1894, xii, p. 152.

The histological changes are similar to those of ordinary acne, except that the process is limited distinctly in extent, and almost invariably terminates in a small central area of necrosis and subsequent scar-formation. Some of these cases may be due to the presence of the toxins of tubercle bacilli. Acne varioliformis occurs in typical development upon the faces of the tuberculous.[2] For further details in this connection, the paragraphs devoted to the tuberculides of the skin (necrotic granuloma, folliclis) should be consulted.

Diagnosis.—The lesions are to be distinguished from the syphiloderm named above, from acne vulgaris, and from variola. The points of distinction are: the absence of fever, present and precedent; the absence of other symptoms of syphilis; the localization of the eruption; and the absence of intermingled comedones and other symptoms of acne disseminata. The involvement of the scalp-surface is not alone sufficient to distinguish it, as syphilodermata and occasionally comedones are visible in the scalp above the brow.

Treatment.—As a rule, the disease yields readily after the use of antiseptic lotions or of ointments containing ammoniated mercury, resorcin, sulphur, mercuric chlorid, formaldehyd, or boric acid, though lesions are likely to develop after suspension of treatment. In severe cases caustics or galvano-puncture may be required. Crocker employs potassium iodid internally with happy results. Engman and Mook[3] report success following treatment with staphylococcus vaccine.

DISEASES OF THE HAIR AND HAIR FOLLICLES.

HYPERTRICHOSIS.

Synonyms.—Hypertrophy of the hair, Superfluous hair, Hariness, Hirsuties, Hypertrichiasis, Polytrichia, Trichuxis; Fr., *Hypertrichose*.

Definition.—Hypertrichosis is a condition in which hairs are developed in excess, or in regions normally occupied by lanugo hairs.

Symptoms.—Hypertrichosis may be congenital or acquired, general or partial. In congenital hypertrichosis the subject is born covered with hair which continues to grow after birth, becoming longer, darker and coarser. An example of unusual congenital hypertrichosis, the so-called Russian "Dog-faced boy," was described by Jackson[4] in

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[1] Annales, 1899, s. iii, x, p. 845.

[2] Schamberg, Jour. Cut. Dis., 1910, xxviii, p. 414 (A case of acne varioliformis, with a coincident papulo-necrotic tuberculid).

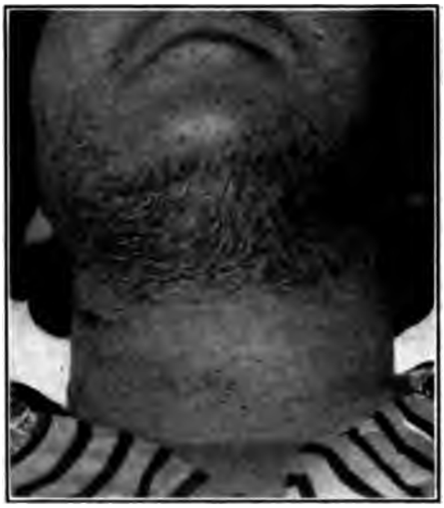
[3] Jour. Cut. Dis., 1913, xxxi, p. 269.

[4] Jackson and McMurtry, Diseases of the Hair, p. 143.

1886. Other remarkable examples are recorded. Anomalies of the teeth are not infrequently noted in these cases. Herzog[1] reported a case of general hairiness with precocious puberty in a three-year-old girl.

Partial congenital hypertrichosis is common and occurs often with nevi. Extensive cases of nevus pilosis are on record. Excessive development of hair may be one manifestation of mixed nevi.[2] Localized hypertrichosis is frequently seen in the lumbar and sacral region, and often in association with spina bifida. It is occasionally seen where neither spina bifida nor nevi are demonstrable.[3] Partial acquired hypertrichosis is seen in cases of precocious development in which a full beard grows in a young boy, or pudendal hair develops in female infants or children. Increased hairy growths not infrequently follows accidents, such as a fractured leg or injury to a nerve, and in these cases the hair often falls at a later date. Likewise, that occurring after the local application of plasters, liniments, or other continued irritation, may or may not fall. Some extraordinary cases are on record of the disappearance of superfluous hair in women after the reëstablishment of menstruation, which had been temporarily suspended. The following case was recorded by Hyde.[4] The patient had a fully developed beard and moustache of one year's duration, which began

FIG. 412



Hypertrichosis.

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[1] Münch Med. Wchnschr., 1915, lxii, pp. 184–225; brief abstr., Jour. Cut. Dis., 1915, xxxiii, pp. 654–655; *Cf.* this article for discussion of etiology.

[2] Hyde, Jour. Cut. Dis., 1885, iii, p. 193; "Nevus lipomatodes."

[3] Gilmore, Jour. Cut. Dis., 1918, xxxvi, p. 372; Vignolo Lutati, Giorn. ital., de mal. Ven. e della Pella, 1915, fasc., ii, p. 172.

[4] Treatise on Skin Diseases, 2d ed., 1888, p. 399.

at the cessation of menstruation. For two years electrolysis was used, with success; at the expiration of this time normal menstruation returned and the hypertrichosis spontaneously disappeared. A similar case treated by Gottheil and Jackson is recorded by the former.[1] Anomalous cases are seen where the hair of the scalp or beard may reach a length of several feet, or that in the genital region many inches.

The hypertrichosis to be chiefly considered here is that which occurs on the faces of women. This frequently begins from the sixteenth to the twentieth year, and becomes conspicuous from the twentieth to the thirtieth. In these cases there is usually a marked growth of downy hair which gradually develops. It may be limited to the lip or chin, or the growth may involve the entire so-called bearded region. The over-developed hairs may be moderate in number, or thousands may be present. This form also frequently occurs in older women at or after the menopause, when many coarse hairs are likely to develop. Occasionally a beard equal to that seen in man is developed in woman.

Etiology.—Several factors are cited as causing abnormal growth of hair. Heredity is a factor in many instances. The cases of universal hypertrichosis often occurred in several generations. Atavism is believed by some to account for this peculiarity. Unna[2] accounts for congenital hypertrichosis on the theory that primitive hairs of the fetus remain unchanged and continue to grow, becoming stronger and coarser, and inducing the type of growth seen in cases of universal hypertrichosis. Normally, the primitive hairs are replaced by ordinary lanugo hairs. Acquired hypertrichosis may be induced by local applications, such as cosmetics, or where liniments have been applied for long periods of time for the relief of neuralgia, and on fractured limbs which have been kept in bandages or fracture-boxes for an extended time. Hairy growth is also increased where the parts, such as the arms and chest in laborers, are much exposed to the sun and wind. In the hypertrichosis of the face heredity is also an important factor. In a series of 350 cases, Jackson[3] found 41 per cent. were hereditary. Derangement of the female generative organs is an occasional factor. The appearance of hair on the cessation of menstruation, and its disappearance on restoration of that function, suggests a connection. It is further suggested in those cases occurring at the menopause, but in many instances no such connection can be found. McEwen[4] cites two examples in which severe hypertrichosis developed coincidentally with cessation of menstruation and increase in body weight. This syndrome he attributed to derangement of the glands of internal secretion. The connection between derangements of endocrinous glands and abnormal hair growth is not understood, but that there is such a connection there appears to be no doubt.[5]

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[1] Trans., IX. Internat. Med. Congress, 1887, iv, p. 180.

[2] Seimssen's Handbuch, xiv, p. 56.

[3] Loc. cit.

[4] Jour. Cut. Dis., 1917, xxxv, p. 829; "The problem of hypertrichosis."

[5] For results of experimental work see Leopold Levi, Bull. et mem. Soc. méd. de Hôp. de Paris, 1913, 3, s., xxxi, p. 956; quoted by McEwen.

Treatment.—The removal of superfluous hair may be accomplished by electrolysis or radiotherapy. Temporary removal is accomplished by the use of depilatories. Electrolysis for this particular condition was first employed by Hardaway of St. Louis,[1] but the method was devised by Michel[2] for the removal of hairs in trichiasis. By this method extensive growths of hair may be removed with only a small percentage of regrowth. The resulting scars are practically imperceptible and are less disfiguring than hair. The instruments required are needles, needle-holders, and epilating forceps, in addition to the necessary apparatus to furnish a galvanic electric current. A sponge electrode is also necessary and this is attached to the positive pole of the battery through an insulated wire. Any good galvanic battery may be employed and should consist of a number of cells connected with a switchboard which is so arranged that a few or many of the cells may be brought into the circuit. A milliampère meter is placed in the circuit to indicate the strength of current employed. The number of cells varies with different individuals; two, four, or twenty or more may be used under different circumstances. The electric current may also be obtained through a wall-plate which has its source of supply from the general lighting system.

The needle most commonly used is made of iridoplatinum. Steel needles may be used; such an experienced operator as Jackson advised a jeweler's steel broach. It is absolutely essential that the needle be attached to the negative pole if steel needles are employed. The needles should be held with a simple insulated needle-holder, such as was recommended by Duhring, which is the shape of a lead pencil and penholder, and is about four inches in length. The handle is of hard rubber, through which passes a metallic rod acting as a conductor for transmission of the current. The needle is inserted into the needle-holder proper, which is slotted, and the needle is clamped immovably by means of a screw-nut. In the other end of the stem is an insulated pin attached to a cord leading to the battery.

Technic of the Operation.—The patient should be placed in a reclining position in a good light and have the handle of the sponge electrode connected with the positive pole of the battery in one hand ready to press the sponge into the palm of the other. In this way at the bidding of the operator the patient makes or breaks the circuit. The sponge attached to the electrode should be wet with salt solution or ordinary water. The needle, which is in the needle-holder above described, and attached to the negative pole, is gently introduced into the hair follicle along the hair shaft, being careful to follow the direction taken by hair. After the needle is thus introduced, the patient grasps the wet sponge attached to the positive pole, which closes the circuit and starts the electric current. If the needle has been inserted in a correct position, there will shortly appear around the mouth of the follicle bubbles, which indicate that the needle has entered the papilla. The current should be continued for fifteen to thirty seconds, at which time the hair will be found to be loosened and can be extracted without traction. The current should be broken by the release of the positive electrode by the patient before the needle is removed. Otherwise, pain is excited upon removal of the needle. One or two milliampères is the strength commonly employed. It is best not to remove many hairs in close proximity on account of the possible destructive effects on the skin. In extensive cases the procedure may be repeated daily in different areas but, as a rule, one to two weeks should elapse before hairs are removed from a particular area. The accompanying pain is not of such degree as to prohibit the use of this method even in sensitive individuals, and it is therefore entirely unnecessary to use any preceding analgesic applications. Transitory lesions of the skin occur at the site of the puncture, but these disappear without treatment. The removal of superfluous hair by this method is advised when the hair is coarse and conspicuous. There are many in whom a fine, downy growth is abundant, but in these cases the work should not be attempted.

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[1] St. Louis Med. and Surg. Jour., 1877, xiv, p. 291.

[2] St. Louis Clinical Record, 1875, ii, p. 145.

In 1897 Freund[1] reported that he had succeeded in removing hairs from a large, hairy nevus by the use of radiotherapy. This method was developed further by Schiff and Freund[2] and has been employed since by many, including Benedikt, Ehrmann, Juttassy, Pusey,[3] and the author. While this method is of value in the removal of hairs from nevi, the observation of patients subjected to radiotherapy for the removal of superfluous hairs has led the author entirely to abandon its use. In some cases the result is satisfactory alike to the physician and patient, but the probability of future telangiectasia is so great as to be prohibitive of its employment.[4] Hairy nevi may be removed by complete excision, but the removal of the hairs by electrolysis will sometimes result in the disappearance of the entire growth without such operations. The other methods which have been employed consist of inserting into the follicles needles previously dipped in caustic solutions, or heated to various degrees. Also circular knives have been employed to excise the hair. All these methods, however, are inferior to the electrolytic destruction of the hair papilla as described above.

Schwenter-Trachsler[5] has treated 252 cases by a new method, as follows: After removing the hair, presumably by shaving, the patient is directed to rub into the surface pumice-stone twice daily. This prevents the hair from growing and if persisted in for a year, produces atrophy of the hair follicle sufficient to produce a permanent result. After six months a rest of four weeks is given and if there is any recurrence the treatment is resumed. The skin itself is never injured. This method has apparently not received much support.

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[1] Wiener med. Wchnschr., 1897, xlvii, p. 428.

[2] Ibid., 1898, xlviii, p. 1058.

[3] Pusey-Caldwell; "The Roentgen rays in therapeutics and diagnosis," Philadelphia, 1903, p. 339. With bibliography.

[4] *Cf.* MacKee, Jour. Cut. Dis., 1917, xxxv, p. 171; "Hypertrichosis and the *x*-ray."

[5] Archiv, 1912, cxi, p. 69; abstr., Jour. Cut. Dis., 1912, xxx, p. 364.

Depilatories remove the hairs without affecting the papilla. The consequence is that the hairs are reproduced in the course of about two weeks. Most of the compounds used for this purpose contain either calcium sulphate, arsenic sulphate, or barium sulphid, made into a paste with warm water. This paste is applied over the hairy surface with a spatula and is permitted to remain until it dries, or produces a sensation of heat and burning, a period usually requiring ten minutes: it is then rapidly removed by scraping with a spatula, when the surface is thoroughly cleansed with warm water, after which the skin is anointed with cold cream salve, or other similar unguent.

Of these depilatories Duhring recommends the following:

℞—Barii sulphidi,		ʒij;		8	
Pulv. zinc. oxid.,					
Pulv. amyl.,	āā	ʒiij;	āā	12	M.

To be prepared in form of an impalpable powder, which, just before using, is to be mixed with water to form a thin paste.

The following are formulæ devised by French authors:

℞—Sodii hyposulphit.,		ʒiij;		12	
Calcis,					
Amyli pulv.,	āā	ʒx;	āā	40	M.

To be finely triturated, and, when used, to be made in a thin paste with water. (Boudet.)

℞—Calcis,	ʒj;	4	
Sodii carbon.,	ʒjss;	6	
Cerati. adipis,	℥j;	30	M.

To be applied as a depilatory in the manner of a paste.

The hairs may be rendered less conspicuous by bleaching them with frequent applications of hydrogen peroxid. Bulkley is of the opinion that the thorough use of this remedy retards the growth of fine hairs.

Trichiasis and **Districhiasis** are conditions in which hairs grow in abnormal directions.

In *trichiasis* the hairs of the eyelids are directed backward and thus irritate the ocular globe. Both eyelids may be affected; the acquired cases usually follow an inflammatory condition inducing distortion.

Districhiasis is a congenital or acquired condition in which two rows of hairs grow on the eyelid, the inner one being directed toward the eyeball, producing irritation in the same manner as that induced by trichiasis. In these two conditions electrolysis, radiotherapy or surgical procedure is employed.

Plica Polonica.—This was formerly supposed to be a disease peculiar to Poles (whence its name), but which has long been recognized as a result merely of persistent neglect, filth, the invasion by parasites, and consequent exudative disorders of the scalp. When it exists the hairs form a huge, matted mass on the crown of the head. Hebra devotes an interesting chapter to the superstitious awe with which this accumulation of hairs, lice, and filth has been regarded. The same condition has been recognized in Alaska in a number of cases observed among the natives of that region.

Plica Neuropathica.—This term is applied to cases in which there occur tangled "lumps" and "festoons" of hairs, flat, curled, looped, and intertwisted, appearing in localized areas of the scalp. Such cases have been recorded by LePage,[1] Stelwagon,[2] and a few others. In Stelwagon's case the felting hair was limited to a dollar-sized area situated below the occipital protuberance, and formed a rounded, matted, felted lock four feet long. The remainder of the hairs exhibited no tendency to felting.

The etiology of these cases is not known. In LePage's case neuralgic pains had occurred previously in the site of the growth.

ATROPHIA PILORUM PROPRIA.

Synonym.—Atrophy of the hair.

Definition.—Atrophy of the hair may be either symptomatic or idiopathic. Illustrations of the first-named condition are observed in phthisis, syphilis, seborrhea, parasitic affections of the scalp, and in almost all general diseases interfering with the nutrition of the pilary growth. The filaments then become dry, lusterless, friable in both longitudinal and transverse directions, and diminished in each dimension. Three forms of atrophy are recognized: Fragilitas crinium, Trichorrhexis nodosa, and Monilethrix.

Fragilitas Crinium.—Under this title a number of disorders due to atrophy, and which produce fragility, splitting, or curling of the hair in abnormal directions, have been described by authors. In a number of local diseases, such as ringworm, favus, and dermatitis seborrheica, the nutrition of the hairs may be interfered with to such a degree that they lose their luster and become dry, brittle, and broken. A similar condition exists in certain constitutional disorders, such as tuberculosis, certain fevers, and other cachectic disorders, which lower the general vitality. In all these cases splitting of the shafts of long hairs is not uncommon, and atrophic changes with respect to size and coloration of the hairs occur. In idiopathic examples of the disorder, such as those reported by Duhring,[3] Hyde,[4] and Parker,[5] marked splitting of the hairs occurs in various ways. Duhring describes fission taking place within the follicle, thereby producing irritation of the skin in that situation; and further describes an atrophy of the bulb and the separation of the hair-shaft into a variable number of stalks, the shaft often becoming enlarged. The curious feature, to him, was the atrophy of the bulb and the apparent hypertrophy of the shaft. In 1887 Dr. Hyde described a similar case, presenting fission of the hair of the beard extending completely to the base of the hair-follicle and there producing irritation. The involved hairs covered several square inches of the surface. The disease was more marked on the chin than on the cheeks or upper lip. Curling of some of the splinters was marked. Similar features were described in Parker's case. When the splitting of the hairs exists solely at the free margins or in the shaft, the condition is quite different from that described above.

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[1] Brit. Med. Jour., 1884, i, p. 160.

[2] Amer. Jour. Med. Sc., December, 1892.

[3] Ibid., July, 1878.

[4] Treatise on Skin Dis., 2d ed., 1888, p. 441.

[5] Brit. Med. Jour., 1888, ii, p. 1335.

Two cases presenting peculiar features were recorded by Jackson.[1] In these there occurred in the scalp well-defined patches where the hair was short and curled up close to the head. There was no sign of inflammation. In a case exhibiting short stumps of hair in a patch the size of a silver dollar, exhibited before the American Dermatological Association by the author in 1914, Dr. Jackson recognized the same condition. In this case the hair in the patch was curled and of finer texture than the rest, and in addition there was intense itching in the area.

The cause of the systemic cases above described is plain; that of the idiopathic cases is not understood. Kaposi believed the splitting occurring in the long hairs in women was due to its distance from the source of nutrition. The splitting of the shaft of long hairs in women without involvement of the point is often due to the thrusting of sharply pointed hairpins through the hair-coils on the scalp, a single thrust being thus capable of ruining a large number of hairs in a braid (Hyde). Jackson[2] believes that the drying of hair with blasts of hot air, as is done by some hairdressers, is responsible for some cases, as well as the use of hot curling-irons, and neglect of proper oiling of the scalp after washing.

Treatment.—The treatment of these conditions is primarily hygienic, as regards both the general health of the patient and the preservation of the hair from artificial methods of management (hot irons, curling, singeing, crimping, and wounding with hairpins). When associated with local or systemic disease, the latter should receive prompt treatment. In addition, local stimulation, shampooings, and inunctions with bland oils and simple applications are useful. When the region of the beard is affected, it should be shaved with regularity.

Trichorrhexis Nodosa (*Trichoptilosis* (Devergie), *Nodositas crinium, Trichoclasia, Clastothrix*).—Trichorrhexis nodosa is a disease of the hairs, first described by Wilson in 1849, since which time it has been the subject of extensive discussion. It occurs chiefly in the male beard. The diseased hairs show one or more, usually several, nodular enlargements, which on careful examination are seen to be due to partial transverse fracture of the hair-shaft. The nodes are oval or ovoid in shape, and stand out fairly prominently, presenting a grayish or whitish appearance. They frequently resemble quite markedly the ova deposited by the *Pediculus capitis*. Under the microscope, the nodes are seen to be composed of a fracture of the hair, which presents somewhat the appearance of two interlocking brushes. When the hair is completely broken, its end simply presents a brush-like appearance. The brittleness of the hair, with consequent fracture and loss, produces a certain amount of alopecia.

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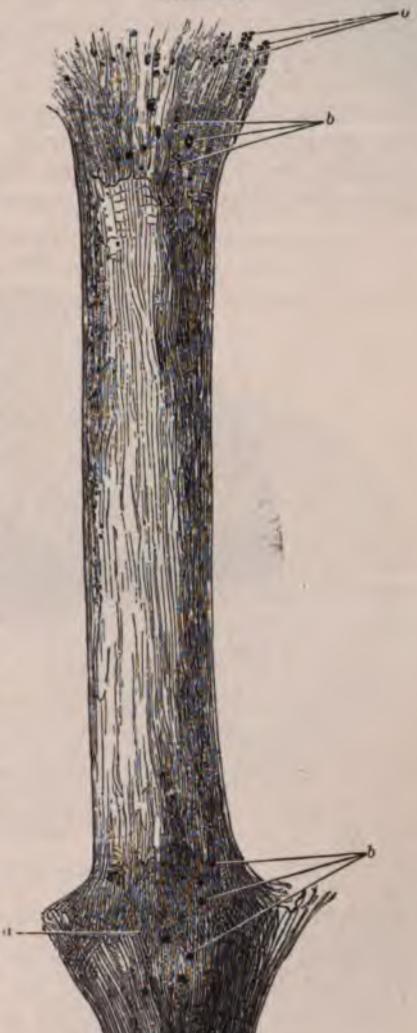
[1] Jour. Cut. Dis., 1903, xxi, p. 473.

[2] Jackson and McMurtry, Dis. of the Hair, 1912, p. 130.

FIG. 413



Trichorrhexis nodosa. (After Schwimmer.)

Etiology and Pathology.—The chief causes of the disorder are given as mechanical and parasitic. Wilson believed the nodes were the result of mechanical injury to a hair whose nutrition was below par. Adamson[1] produced typical nodes by trauma in hairs from a patient suffering with the disease. Similar experiments were made with normal hairs as control without producing the same result. He concludes that the fractures or nodes are the result of mechanical injury to a hair of damaged nutrition. Hodara[2] found a bacillus, which he termed *Bacillus multiforme*, that he cultivated, and with which he reproduced the affection by inoculation. Spiegler (quoted by Adamson) described a similar bacillus, which he believed to be identical. He also reported successful inoculations. Markusfeld[3] rubbed up hair with pumice-stone in a sterilized mortar and obtained a Gram-positive bacillus which he identified as being the same as Spiegler's. Heidingsfeld[1] believes there are two varieties, a pseudo and a true, the latter being of infrequent occurrence. Bruhns[2] found trichorrhexis nodosa common in Berne. In a study of 20 cases, in 6 of which inoculation experiments were made, the latter proved negative. He believes in the mechanical cause in hairs having an underlying nutritional change, and quotes Rauber's case, in which the disorder occurred periodically following epileptic fits. Richter[3] concludes the disease is not parasitic, but due to interference with the nutrition of the hair. Barlow,[4] after reviewing the bacteriological investigations of Raymond, Blaschko, Hodara, Spiegler, and Essen and comparing them with his own results, concludes that there is no absolute proof that the condition has a parasitic origin. He further states that the view that trichorrhexis nodosa is conveyed from diseased hairs to the bristles of brushes cannot be accepted, for the reason that the same condition is found in brushes which have not been in contact with diseased hairs. The mechanical origin of the disease must be considered. Observation shows that the condition is only found in much-used brushes and not in new ones; and, finally, it is probable that nutrition-changes in the hair play a

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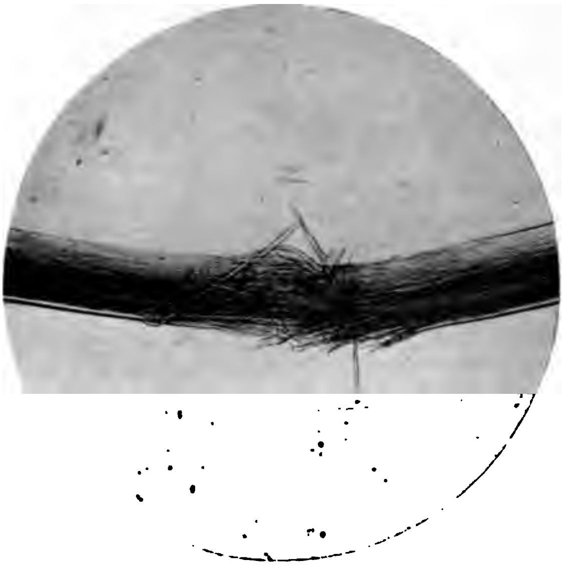
[1] Brit. Jour. Derm., 1907, xix, p. 99.

[2] Monatshefte, 1894, Bd. xix, p. 173.

[3] Archiv, Bd. xlviii, p. 146.

FIG. 414



Trichorrhexis moniliformis. (Heidingsfeld.)

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[1] Jour. Cut. Dis., 1905, xxiii, p. 246 (a report of a case and review of the literature, with extensive bibliography; illustrated with 6 plates).

[2] Archiv, 1897, xxxviii, p. 43; abstr. Brit. Jour. Derm., 1897, ix, p. 290.

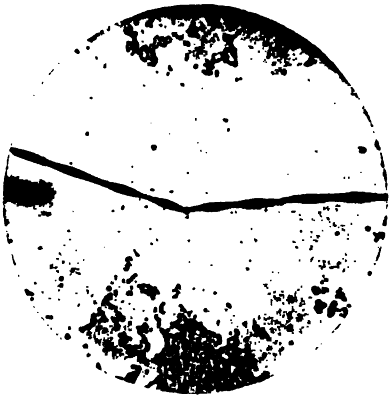
[3] Münch. med. Wochenschr., No. 40, October 6, 1896, p. 947; abstr. Brit. Jour. Derm., 1897, ix, p. 121.

[4] Münch. med. Wochenschr., No. 26, June 30, 1896, p. 615; abstr. Brit. Jour. Derm., 1897, ix, p. 121.

part in the production of the disease. Stelwagon[1] and Ravenel[2] found the disease in the bristles of shaving-brushes used by patients with the disease. The supposition was that the brushes were infected by the patients. Similar observations were later made by several other observers. By some of the later investigators the disease was not only found in brushes used by patients having the disorder, but in those used by unaffected individuals.

Treatment.—Treatment is unsatisfactory. Crocker states that change of climate has cured it in some cases, and that repeated shaving is sometimes successful in others. Richter advises the use of oils and simple ointments and an avoidance of all mechanical irritation. Those who view the disease as parasitic suggest parasiticides. The extraction of the affected hairs is recommended by most observers. General treatment in the way of tonics may be of value in certain cases, but the experience of all, no matter what their view as to etiology, points to the fact that treatment is, as a rule, disappointing.

Fig. 415



Trichorrhexis nodosa. (Heidingsfeld.)

Monilethrix.[3] — **Synonyms.** — Beaded hairs, Pili annulati; Fr., *Aplasia moniliforme intermittente;* Ger., *Ringel Haaren.*

Definition. — Monilethrix is a disorder characterized by the presence of beaded hairs, occurring usually in the scalp.

This condition was first described by Walter Smith, of Dublin, in 1879, and since by a number of observers, including Luce, Anderson, Crocker, Lesser, MacKee and Rosen, and others.

Symptoms.—The disease occurs in children as a rule, but may develop later in life. Adult cases have been recorded by Unna, Gilchrist, Morrow, Ruggles, Savatard and others. The entire scalp may be invaded, or the disease may be limited to localized areas. In the affected areas the hairs are broken, twisted, curved and lusterless, and commonly one-fourth to one inch in length. In most instances there is an associated keratosis pilaris. This may be limited to the areas where the peculiar hairs are found, or have a more extended distribution. In addition there may be an associated pityriasis in the scalp, and in long-continued cases atrophy may follow. The affected hairs, in addition to their distortion, present a succession of rings or nodes at regular intervals. By transmitted light the nodes or beads appear dark and the constrictions light; by reflected light these conditions are reversed. This peculiar change gives the hair a characteristic beaded appearance. The so-called nodes are the normal portions of the hair, whereas the constriction between the nodes represents an atrophic portion. As a rule, the disorder is limited to the scalp, but occasionally occurs elsewhere. Ruggles recorded symmetrical involvement of the legs below the knees and Gilchrist reported a similar instance. As a rule, there are no subjective symptoms and the general health is unaffected.

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[1] Quoted in Stelwagon's Diseases of the Skin, 1914, p. 976.

[2] Medical News, October 29, 1892.

[3] MacKee and Rosen, Jour. Cut. Dis., 1906, xxxiv, pp. 444 and 506; "Monilethrix: a clinical and histological study, with a report of six cases, and a review of the literature." A thorough and comprehensive study.

Etiology and Pathology.—The cause of the disorder is unknown. It is agreed by most observers to be trophoneurotic in origin. It occurs in both sexes. Nervous shock is cited by Unna and Sabouraud as a principal cause. Heredity is a factor. McCall Anderson recorded 14 cases in five generations; Sabouraud 17 cases in five generations; Hallopeau and Lefèvre 5 cases in two generations; Dore and MacKee and Rosen each 2 cases in a family with no heredity history. Many examples are seen in which heredity plays no part. Kaposi, Virchow and others explained the condition as being due to periodic aplasia of the hair follicle. Smith believes the disorder to be due to perverted nutrition in which there are alternate periods of activity and sluggishness, and that the origin of the disorder is intrafollicular. Gilchrist found the disorder had its origin in the hair follicle near the papilla, and further found strictures in the follicle corresponding to those on the hair. MacKee and Rosen found the hair follicles dilated and tortuous, and their upper portions markedly hyperkeratotic and containing horny plugs. The inner surface of the follicle presented undulations corresponding to the nodes and internodes of the hair. The upper portion of the corium showed signs of low-grade inflammation. The sebaceous glands and elastic tissue were underdeveloped. The epidermis was atrophic between the follicles but acanthosis was present near the follicle. The shaft of the hair between the nodes was composed of a thick cuticle, a reduced cortex and practically no medullary substance.

Bacteriological examinations have uniformly been negative.

Treatment and Prognosis.—No treatment has been found of much value. The disorder continues and the chief characteristic is the disfigurement.

TRICHONODOSIS.

Synonym.—Knotting hair.

A patient presenting this peculiarity was demonstrated before the London Dermatological Society in 1905 by MacLeod.[1] In 1906 Galewsky[2] recorded cases under the above title, in which there was exhibited a pseudo-knotting and fraying of the hair, associated with thinning and breaking of the hair-shaft, so that it remained short without cutting. It was further stated that the long hairs of the scalp, beard, pubes, and even the lanugo-hairs of the trunk, might be involved. MacLeod[1] describes a case in which the scalp was dry, the hairs dry and lusterless, but deeply pigmented, and the ends split up into several fibers, or pointed and atrophic, or occasionally bent up like a hook. The majority were curled up at the ends, forming one or more loops. In cross-section the hairs were seen to be flat or oval, some twisted. Many small nodes were present, but not all the hairs were involved; some had several nodes, usually on the peripheral half of the shaft. On microscopic examination the nodes proved to be various forms of knots. Kren,[2] in examining 54 women with skin disease, found 35 had nodes on the hair, the site of the node being near the middle or terminal portion of the hair.

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[1] Brit. Jour. Derm., 1905, xvii, p. 61.

[2] Archiv, 1906, lxxxi, p. 195.

Dawson[3] recorded a case of fragilitas crinium associated with knotting. In this patient the hairs were breaking off close to the scalp, and on some of them nodular swellings occurred, which on microscopic examination proved to be half-hitched knots, situated about two inches from the roots.

PIEDRA.[4]

Synonyms.—Fr., *Trichomycose nodulaire.*

Definition.—Piedra (Sp., *piedra*, a stone) is a name given to a disorder affecting chiefly the natives of certain districts in Colombia, South America. Both men and women (more frequently the latter) and persons of all races are liable to contract the disease, which involves the shaft of the hairs of the scalp chiefly, but also of the head and other hairy regions. The individual filaments are dotted at irregularly disposed points with minute nodosities, apparently as hard as stone, from which circumstance the disease has acquired its Spanish name. The nodes are pinhead-sized and gritty, so small at times as to be scarcely perceptible to the eye, though distinctly recognized on palpation. A score or more have been found on a single hair 60 centimeters in length. The affected filaments are distorted, and apt to be matted and twisted, as in plica. Each node is fastened to the hair like a sheath, though it may be implanted on one side only. It is divided readily with a sharp knife; and is colored in various shades of gray, brown or black. When a comb is passed through the hairs, a distinct crepitation is produced by friction against the dense, nit-like nodes.

MacLeod[5] describes specimens of hair from an aboriginal Indian girl, British Guiana, as follows: The hairs were characterized by the presence on the shaft of hard nodules, varying in size from minute specks like particles of grit to small pinheads, oval in shape, brownish black in color, situated either on the side of the shaft, like a nit, or forming a concretion around it. The extrafollicular portion only was affected. The nodules varied in number from one to ten or more, and were situated close to each other or at irregular intervals. They were entirely outside of the hair-shaft at first, but eventually caused a breaking up of the cuticle and fraying of the cortex, and rendered the hair liable to fracture opposite them.

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[1] Brit. Jour. Derm., 1907, xix, p. 40.

[2] Wien. klin. Wochenschr., 1907, p. 916; abstr. Jour. Cut. Dis., 1908,

[3] Brit. Jour. Derm., 1909, xxi, p. 54.

[4] Manson, Loc. cit., p. 780. Morris, London Pathological Society' p. 441 (with plate), and Medical Times and Gazette, 1879. Juhel-Rénoy s. ii, ix, p. 777, and 1890, s. iii, p. 765 (with illustrations). Trach 1896, xxii, p. 1.

[5] Brit. Jour. Derm., 1911, xxiii, p. 255.

Etiology and Pathology.—The origin of the disease is unknown, but in Colombia it is said that the node-like masses are due primarily to a species of mucilaginous oil employed by the natives for hair-dressing. According to Juhel-Rénoy, the nodes are composed of numerous spore-like bodies, recognized readily by soaking the hairs in dilute liquor potassæ after washing in ether. The spores are twice the size of those furnished by the trichophytons, are polyhedral as a result of counter-pressure, and form a species of tessellated mosaic, the elements of which are united by a greenish, soluble cement, in which are incorporated minute rods resembling bacteria.

In MacLeod's case he states that the nodules were seen to consist of large spores of a peculiar fungus, which were probably those described by Behrend as *Trichosporum giganteum*. In small nodules the spores had a strikingly regular arrangement. At the periphery of the nodule there was a palisade layer of oblique spores of from ten to twelve microns in length, arranged at right angles to the hair-shaft; while next to the hair the spores were irregular and tended to be polygonal. In some instances they had a peculiar fan-like arrangement of rows of spores radiating from a central base. They appeared to be held together in a mass with some cement substance, which formed a sort of capsule enclosing them; and they presented a yellowish appearance, due to a diffuse coloring matter in the protoplasm and the presence of pigment-granules. No definite mycelial threads were seen.

Cultures from this case are described by MacLeod[1] as follows: The culture began as a small, white knob, with a slightly woolly surface. It gradually changed, and in a week assumed a greenish-gray tint, and finally became of a brownish tinge, a little lighter than cooking chocolate.

Diagnosis.—The disease is closely allied to tinea nodosa (Crocker and Pernet) and to Piedra nostras (Unna and Behrend), but is quite distinct from lepothrix and Castellani's trichomycosis flava, nigra, and rubra, both of which affect the axillary regions chiefly.

Treatment.—Thorough cleansing with soap and water, followed by the application of a parasiticide, is recommended, with shaving if necessary.

Chignon Fungus (*Beigel's disease*).—This affection is discovered upon false hairs, which exhibit upon their shafts dirty-brownish nodes, due to masses of parasites. These organisms are said by Hallier[1] to be a species of sclerotium.

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[1] Brit. Jour. Derm., 1912, xxiv, p. 23.

Piedra Nostras (Unna); *Tinea nodosa* (Crocker).—This disease was first described by Morris and Cheadle,[2] and has since been reported by Crocker,[3] Thin,[4] and others. The disorder affects the hairs of the beard or moustache. The nodular concretions are developed irregularly along the hair-shaft, the bulb remaining unaffected, as the concretions begin some distance from the root. Crocker described the hairs as being ensheathed in a concretion which made the outline of the hair irregular and of a dark-brown color, dull and opaque. The hairs are brittle and break or split. Under the microscope the growth is recognized as an ensheathing mass, which, when the hairs are split, penetrates below the cuticle. With high power it is seen to be made up of fungus spores, somewhat smaller than those occurring in trichophytosis. In Crocker's case Pernet found the spores occurring in rows at right angles to the shaft of the hair, looking like segmented mycelium, and bound together by a cement substance secreted by the fungus.

Treatment.—The treatment advised is shaving or clipping, with the application of parasiticides.

LEPOTHRIX.

Synonyms. — Trichomycosis nodosa (Patterson), Trichomycosis palmellina (Pick).

Definition.—Lepothrix is a disorder in which nodular or diffuse masses of fungous material are found on the hairs in the axillary or genital regions.

The disorder was first described in 1869 by Paxton,[5] later by Wilson,[6] who named the disorder, Patterson, Pick, Babés, Barthelemy and others. It has recently been fully described in America by Lane.[7]

Symptoms.—The fungous material occurs on the hair in two forms; a nodular and a diffuse form. In the former individual nodes or masses occur on the hair, whereas in the diffuse form much of the shaft may be ensheathed with the growth. Crocker describes circular, well-defined nodes lying upon but not surrounding the shaft, three times the diameter of the hair and containing fibers of the cortex that had been split by concretions. In the diffuse form the masses are arranged irregularly along, and partially or entirely encircling the shaft of the hair. They are reddish-brown or yellowish, or when secondarily infected with *Bacillus prodigiosus* they are red. In addition, Lane has described a black variety.

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[1] Quoted from Jackson and McMurtry, Dis. of the Hair, 1912, p. 291.

[2] Lancet, 1879, i, p. 190.

[3] Dis. of the Skin, 3d ed., p. 1176.

[4] Lancet, 1882, ii, p. 742.

[5] Jour. Cut. Med., 1869, iii, p. 133.

[6] Quoted by Crocker.

[7] Jour. Cut. Dis., 1919, xxxvii, p. 387; "Lepothrix; including a brief consideration of Trichomycosis flava, rubra et nigra of the axillary regions (Castellani's disease);" full description, report of cases and review of the literature.

Their consistency varies; they may be soft and easily removable, or hard and adherent. The hairs are described as being brittle and easily broken, inducing brush-like fractures. The brittleness of the hair appears to depend upon the extent of its invasion with the growth and as to the presence or absence of moisture. Castellani described a yellow-black and red variety under the title "*Trichomycosis flava nigra and rubra of the axillary regions*"[1] seen by him in Ceylon, which he considered distinct from the forms occurring in temperate zones. No subjective symptoms are present, as a rule.

Etiology and Pathology.—The disease is of parasitic origin and occurs in all grades of society. Heat and moisture contribute to the growth of the microörganisms. Crocker, Behrend and others considered it common. Lane observed it in 51 out of 128 men twenty-one years of age who were being examined for military service; he also observed it in Negroes. The nodular masses are made up of microörganisms held together by a homogeneous substance. They penetrate the cortex of the hair and in their development cause its disintegration. Jackson and McMurtry[2] say the disease is caused by a species of schizomycetes forming zoöglea masses. The cocci are arranged in masses and heaps, rows, or in groups of four. Hartzell[3] found masses of micrococci which in cultures were arranged in groups of four. Castellani found a bacillary-like fungus in the yellow variety and the same microörganism associated with cocci in the black and red varieties.

Treatment.—Crocker recommended shaving and the application of mercuric chlorid (1 to 2000). Castellani recommended dabbing the hairs two or three times daily with a solution of formalin in spirit (4 grams in 180), and applying at night a 2 to 5 per cent. sulphur ointment.

TRICHOSTASIS SPINULOSA.

Under the above title Nobl[4] described a peculiar condition involving the hair-follicles, which apparently had not been recorded, but which he believed was not very rare, as he had discovered six cases in the course of a few months in his clinic. The disorder occurred in patients between the ages of twenty-four and sixty-two, and five of the six were men. In one of these the lesions were described as appearing like dark comedo-plugs seated in the hair-follicles on the shoulders, back, at the sides of the thorax, and on the upper abdomen. They were symmetrically placed, raised from 1 to 1½ mm. above the surface, and were unaccompanied by any evidence of inflammation. On microscopical examination, the lesions were found to be bundles of hairs, each being 1½ to 2 mm. in length, and containing from ten to forty hairs. There was no evidence of inflammation in the corium, and the author looked upon the condition as being a congenital dystrophic disturbance of hair-growth in the individual follicle.

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[1] Brit. Jour. Derm., 1911, xxiii, p. 342; "Trichomycosis flava, nigra et rubra of the axillary regions;" Idem., ibid., 1913, xxv, p. 14; "Further researches on Trichomycosis flava, nigra et rubra of the axillary regions."

[2] Diseases of Hair, 1912, p. 293.

[3] Diseases of the Skin, 1919, p. 653.

[4] Archiv, 1913, cxiv, p. 61.

CANITIES.

Synonyms.—Trichonosis cana, Poliothrix, Poliosis, Hoariness, Grayness or Whiteness of the hair.

Symptoms.—In this anomaly the hairs appear in all shades of whiteness, from dirty-gray or yellowish-white to a steel-gray or silvery-white. This may be either a general or a partial, congenital or acquired, physiological or pathological, prematurely, rapidly, or gradually acquired condition. General congenital whiteness of the hairs is seen in albinismus, where pigment has never colored the filaments. Partial congenital whiteness is occasionally seen in patches, limited in size and varying in color from pure white to a deeper hue, that from birth do not receive pigment in due proportion, thus contrasting with the pigmented filaments by which they are surrounded. This is sometimes a family peculiarity.

Physiological decoloration of the hairs in variable shades is the well-known result of advancing years. When premature, it may occur early in life and result from pathological causes or be due to individual or inherited peculiarities. It may occur gradually or suddenly; in the former case the hairs usually pass through varying shades of gray to white, and this at any period after (occasionally before) puberty, though commonly after middle life is reached. At first a few scattered hairs are bleached; then these multiply, and so gradually the whitening occurs; in other instances the bleaching is general, symmetrical, and uniform. In yet other cases, even in senile hoariness, the canities is at first circumscribed, the hairs of one part of the scalp blanching before others; thus the hairs of the beard whiten before the scalp, or *vice versa.* Recurrence to the darker shades is noted rarely. Leonard,[1] of Detroit, cites a number of curious instances in which changes of this sort have occurred. Generally, however, canities of advanced years is progressive and permanent, occurring earliest on the temples and the beard of man, then involving the vertex of the head. Finally, the hairs of the entire body-surface may undergo similar pigmentary loss.

The coloring of the hairs of the head is, to a greater extent than commonly is appreciated, subject to variation from the operation of external causes. Thus, washing the hair with alkaline solutions has a bleaching effect, while profuse sweating, inunction with fats, subjection to smoke, and the heat of summer have the opposite effect, the last named being associated possibly with increased sweating in the hot season.

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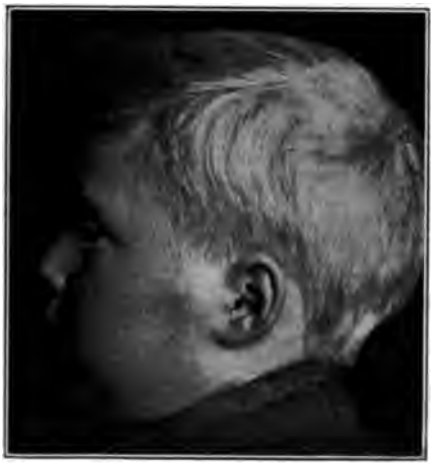
[1] The Hair: its Growth, Care, Diseases, and Treatment, Detroit, 1880.

Cases of sudden blanching of the hairs, occurring, for example, in a single night, are sufficiently numerous and well authenticated to be admitted as among the rare possibilities of a clinical experience. Nervous disorders, both centric and peripheral, such as long-continued mental depression, melancholia, paralysis, neuralgia, and traumatism of nerves or of nervous centers, may be followed by more or less rapid, general or partial, and permanent canities. The same result may follow wasting disorders, such as typhoid fever, tuberculosis, syphilis, and malarial fever, in which cases, as distinguished from the others, pigmented hairs eventually may replace those which were white. A peculiar case of canities in a child[1] was observed by the author, in which the hair was of an iron-gray hue, due to admixture of a few brown hairs with the white. At three and one-half years the patient had had complete defluvium capillorum following typhoid. The hair promptly regrew and presented the appearance described when the child was seen at the age of five years (Fig. 416). At this time the hairs of the brows and lashes were entirely white, and there were associated vitiliginous areas in different parts of the body. The general health and nutrition otherwise were perfect.

FIG. 416



Iron-gray hair.

The first hairs springing from a patch of alopecia areata in which repair is in progress are often white or gray, and are replaced later by those of normal color. The pressure of a truss or of a corset has produced patches of vitiligo and canities. Landois has shown that many instances of suddenly occurring canities depend solely upon the rapid appearance of air-bubbles in excess of the average number in the hair-shaft.

In the rare affection known as *Ringed hair* the pilary shafts present alternately white and pigmented segments.

Etiology.—Whitening of the hair may be senile in origin, in which case it is customary to declare it to be physiological; or be due to heredity; to deficient nutrition or innervation of the hair-follicles; to functional or organic nervous affections (fright, facial atrophy); or to local chemical action upon the hairs. Premature canities in young adults is often associated with the occupations of life, being much commoner in men who from necessity have the head habitually covered and who yet lead sedentary lives.

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[1] Jour. Cut. Dis., 1909, xxvii, p. 258.

Pathology.—The color of the hair is dependent upon the pigment situated in the matrix and between the horny cells, and upon the natural yellowish color of the dried horny cells. In source and character the hair-pigment is undoubtedly identical with that of the skin in general. Decoloration of the hairs may be due to failure of supply or to removal of pigment; to unevenness of the hair-surface (by which light is refracted); or to air-bubbles between the cells. In senile and presenile decolorations there is commonly actual diminution of pigment. Rapidly occurring canities is ascribed to the sudden appearance of air-bubbles in quantity in the shafts of the hair. Alterations of color in the hairs are attributed to successive periods of activity and rest in the pigment-producing cells.

Treatment.—The chief means of remedying premature canities is by the action of dyes, which are, in the main, compounded with solutions of silver nitrate, lead acetate, and ferrous sulphate. The main objections to their use are the fact that the dyed hair never has the exact hue and luster of naturally tinted filaments, and thus rarely deceives the observer, and the disagreeable coloring of the scalp which results from incautious use of the dye, and the consequent liability to irritation of the surface. These substances are not known to have a deleterious effect upon the general health. Kaposi gives the following formulæ for hair-dyes:

To obtain a black color—

℞—Argent. nitrat.,	gr. xv;	1		
Ammon. carb.,	gr. xxij;	1	5	
Unguent.,	℥j;	30		M.

Or

℞—Argent. nitrat.,	ʒj;	4		
Plumb. acetat.,	gr. xv;	1		
Aq. ros.,	ad f℥iij;	ad 90		
Aq. cologn.,	gtt. xv;	1		M.

To obtain a brown shade—

℞—Acid. pyrogal.,	gr. xv;	1		
Aq. cologn.,	ʒss;	2		
Aq. ros.,	℥jss;	45		M.

Anderson first applies a lotion of mercuric chlorid, 2 grains to the ounce (0.133 to 30.), and follows this with a solution of sodium hyposulphite, 1 drachm to the ounce (4. to 30.), for the production of a jet-black shade. In the way of constitutional treatment, he suggests, in cases of accidental presenile blanching, strict attention to the general health and arsenic internally.

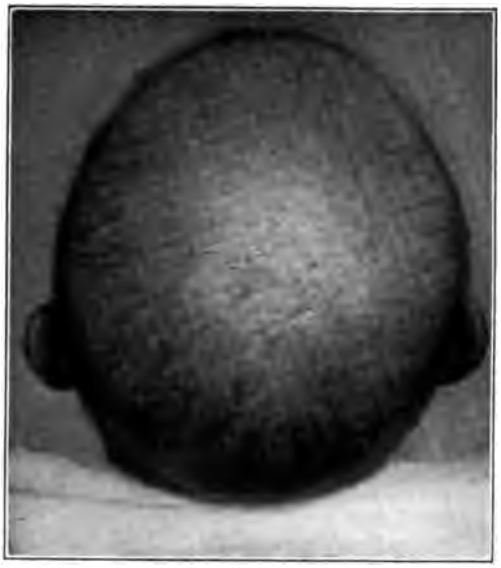
ALOPECIA.

Synonyms.—Calvities, Defluvium capilloru
Baldness. Fr., *Alopécie;* Ger., *Kahlheit.*

Definition.—The term "alopecia" denotes loss of hair. It occurs in a large number of morbid conditions. For convenience of description, the various varieties of this loss are classed under the general term alopecia, then divided into the sub-groups of congenital, premature, and senile. Alopecia areata, being distinctly different from the above affections, is considered particularly in these pages.

Alopecia Congenita[1] (*Hypotrichiasis, Alopecia adnata, Oligotrichia congenita, Congenital alopecia areata, Universal congenital atrichia.* Fr., *Alopécie congénitale*).—Under these several titles has been described a group of rare cases in which the symptoms, though possibly originating from different causes, are suggestively similar. The following conditions are described under this title:

Fig. 417



Alopecia congenita.

1. Complete and universal absence of hair at birth, not succeeded later in life by a pilary growth. This is believed to be an intrauterine atrichia due to failure of development of the hair-follicles.

2. Universal congenital hypotrichiasis, in which hairs develop in all regions of the body, but later fail to be succeeded by filaments normal in length, vigor, color, and texture. Two subvarieties of this condition have been recognized: (*a*) That in which the infant at birth is provided with the relatively long hair of most normal infants, which in due time falls and is replaced by a scanty down, which later does not develop into the normal hair of the adult; (*b*) that in

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[1] For extended discussion of this subject and bibliography, see article by Hyde: "Congenital Alopecia as an Expression of Atavism," Jour. Cut. Dis., 1909, xxvii, p. 1.

which, after birth, the hair which normally is shed persists, but later there is produced a scanty or ill-developed pilary growth.

3. Complete or partial absence of hair at birth in definitely circumscribed regions, such as the scalp, the brows, the pubes, or the axillæ.

4. Generalized or circumscribed absence of hair at birth, followed in later life by normal hair-development. This condition is believed to be due to preëxisting intrauterine disease, or to such a disease seriously involving the hair-follicles at birth, resulting in a true alopecia, evidences of which may be lacking at the time the patient comes under observation. Some cases of congenital hair-absence are reported as due to alopecia areata from intrauterine nervous shock.

5. Cases in which one or all of the anomalies cited above coexist with anomalous conditions recognized in the teeth, nails, and other organs.

Dubreuilh and Petges[1] describe the condition as occurring upon a portion of the body (chiefly the scalp) where there has been a congenital nevus; in other cases the condition results from arrested development of the skin. In these cases the plaques are situated near the posterior fontanelle, over the occiput, or on or near the median line. These authors also describe an "obstetrical" form, characterized by the irregular and variable outline of the patch, occupying the parietal or fronto-parietal regions, where the forceps of the obstetrician are applied; and a "sutural" form, produced by enlargement of the cranium before union of the bones, its seat coinciding with the lines of union of the cranial bones.

The anomalies thus outlined are rare, the rarest being those in which there is failure of development of the hair-follicles; in this case the condition is congenital, permanent, and complete. The "complete and absolute cases" described by Eshner, Shede, and Ziegler are due either to non-development of the hair-follicles or to intrauterine accident.

In cases commonly recorded under the titles given above, several generations of a single family may be similarly affected; parents and child, grandparents, uncles, cousins and cousins-german, and other near or slightly removed relations of the subject of the anomaly may exhibit both natal and postnatal hypotrichiasis. Nicolle and Halipré report the existence of this anomaly in 36 individuals occurring in six generations.

The teeth and nails are frequently altered in cases of congenital alopecia, the latter probably being more involved. In addition, there may be a defective secretion of sweat, impairment of the senses of smell and taste, and various other physiological defects. Cases are recorded with coincident webbing of the fingers and toes by both Hyde and Winfield (quoted by Hyde). Several of the subjects of the anomalies are reported as victims of alopecia areata, atrophy of the finger-tips, epidermolysis bullosa hereditaria, and xeroderma. Whitish, mosaic-like areas have been recognized in the retina, supposed to be due to retinitis albicans, but, according to Hyde, more probably the result of congenital reversion, the type of some of the lower animals whose retina exhibits areas through which the sclerotic is projected.

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[1] Annales, 1908, s. iv, ix, p. 257 (A valuable contribution to the subject of circumscribed congenital alopecia).

Etiology and Pathology.—Congenital alopecia attacks the male subject more frequently than one of the opposite sex. The number of subjects of the male sex is nearly double that of the female sex. In a certain proportion of cases heredity[1] plays a part. No demonstrable cause can be assigned for this condition. The mothers of some of the subjects of this anomaly have suffered from antenatal alopecia areata, and the disorder has been noted in families of close intermarriage.

The condition is probably due to arrested, or entire lack of, development of the hair-follicles. The arrest may occur at any period from the time when, in the embryo, a slight invagination occurs, through all the stages of the future development of the hair and follicle. The follicles may therefore be seen as slight invaginations or minute depressions, when arrested very early. Later this follicle may be more or less completely formed, with its papilla in a corresponding stage of development. Upon the stage at which the process stops will depend the future result—utter, complete absence of the hair, or rudimentary productions of hairs. In certain cases the developmental process is accomplished in varying degrees after birth, and in these cases the hairs may be produced more or less perfectly in later years.

According to the observations of Schede, Atkins, and Ziegler, the sebaceous and sudoriparous glands are normal, and the hair-follicles are represented by epithelial cylinders, composed of five or six layers of cells, with a central lumen, not containing a hair and without a papilla. These cylinders represent hair-sheaths, but are incompetent to produce evolution of a pilary filament. Certain authors, according to Brocq, have found complete absence even of rudimentary follicles.

The process appears, therefore, to be one of defective development, of unknown origin.

Treatment.—The treatment of these conditions is that which is outlined in cases of simple alopecia.

Prognosis.—It is to be remembered that many of these cases are hopeless, the hairs never developing to a normal condition, the nails always remain undeveloped, and in some cases the teeth are permanently deficient in number and size.

Alopecia Prematura.—Premature or presensile alopecia (*premature calvities*) is that form of acquired baldness which occurs in individuals who have not attained advanced years. Idiopathic and symptomatic forms are recognized.

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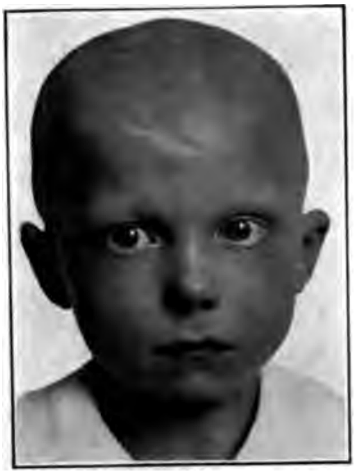
[1] Balzer and Barthélemy, Bull. Soc. fran. Derm., 1914, p. 321. (Congenital Alopecia in a Family.) A report of 4 cases in 3 generations.

Alopecia Promatura Idiopathica.—This is that variety in which diseases of the scalp or of the general economy cannot be recognized as factors in its production. It is, like the senile alopecia, more common in men than in women, and is in the former sex decidedly prevalent among those leading sedentary lives. The loss of hair may be produced either rapidly, or, more commonly, slowly, and at any time after puberty.

The process may begin with slight thinning of the hair in the affected regions. On close examination it becomes clear that the hairs which remain are relatively lusterless and lacking both in vigor and size as compared with the hairs growing on unaffected portions of the scalp. The hair-loss is always symmetrical, and at times remediless, partial alopecia being a permanent result of the process. The pilary growth may recede gradually and evenly from the forehead, or, what is more frequent, from the temples on either side of the median line, leaving a more vigorous crop extending centrally toward the root of the nose; or the loss may produce the effect of the tonsure. Often the fall of individual hairs is followed by a new growth of younger filaments, these rarely developing beyond the grade of short and slender hairs, which either soon disappear or persist without much further development. In many families there is a predisposition to this premature loss of hair, usually in the form of the receding temple, that may be recognized in the males of succeeding generations. At the time that the hair in the situations described above is either gradually or more rapidly disappearing, a growth of strong, active hairs over unaffected regions may be noted. In some persons the baldness, even before the attainment of the middle of the third decade, involves the greater part of the scalp.

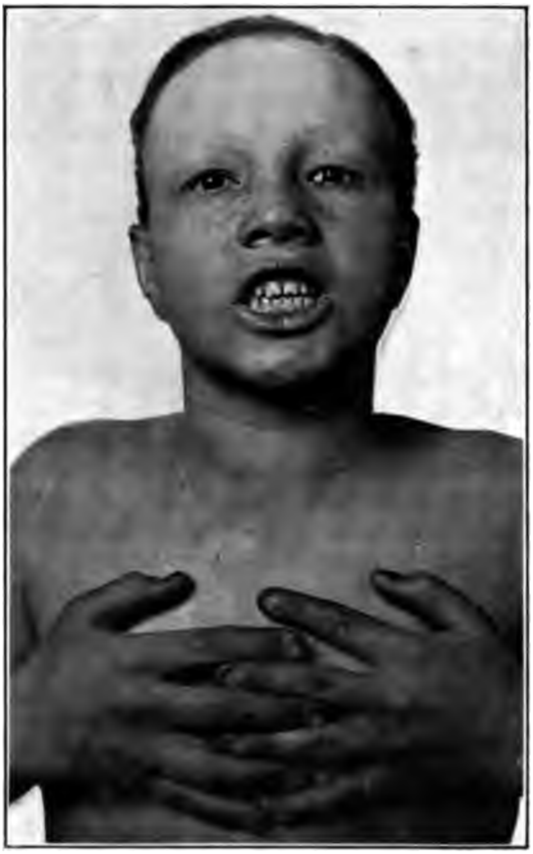
FIG. 418



Alopecia totalis.

Alopecia Prematura Symptomatica.—This variety of alopecia may result from a number of systemic and local conditions. Loss of hair (*defluvium capillorum*) is common after typhoid, eruptive, and other fevers, and after other local and systemic disorders interfering with the nutrition of the skin. Frequently, the hairs do not fall for some weeks after the patient has recovered from the constitutional disturbance, but remain in their follicles until pushed out by the new hairs, or until gradually pulled out by the use of brush and comb. In these cases there is usually a general and symmetrical thinning of the hair. The loss is usually temporary, as new hair gradually replaces that which has fallen. Not infrequently, the new hair is more luxuriant, and occasionally of entirely different quality from the old. The alopecia of the early periods of syphilis is of this class, but occurs as a rule in characteristic patches. A slower loss of hair is seen in

Fig. 419



Alopecia, spoon-nails and malformation of the teeth in a young boy, otherwise strong and well.

many cachectic conditions, such as tuberculosis, diabetes, leprosy (in which the alopecia is limited often to the eyebrows and eyelids), and myxedema.

Alopecia Pityrodes (*Alopecia furfuracea*).—Of all the local causes of alopecia pityrodes a seborrhea in some form is the most frequent. Jackson,[1] in 300 cases of premature alopecia, found 75 per cent. due to seborrhea. Elliot[2] in 346 cases found over 90 per cent. due to seborrhea. The disorder is usually manifested in early adult life, though persons of both sexes, from twelve to fifteen years of age, may display typical forms of the disease. After the condition known as "dandruff" has existed for some months or years, the subject of the affection discovers a relatively large loss of hair from the scalp, producing thinness of the growth upon the vertex, near the brow, or even the temples. The hairs, when examined, are found to be shortened, dry, harsh, lusterless, and rarely well anointed with sebum. They are rebellious to comb and brush, and project irregularly from the brushed surface. Those shed from the scalp, especially of men, are found to be nearer in type to the lanugo or downy hairs than those which fall physiologically from a vigorous growth of hair in a healthy subject; that is, they are short, thin, pointed, and often with an indistinct medulla.

At the same time the scalp is in process of incessant desquamation, the scales usually being of pityriasic type, and exceedingly abundant so long as the alopecia is not complete, after which the scaling ceases. The mealy, bran-like scales are shed in a fine shower upon the clothing of the patient, and in men, among whom the disease is more common than in women, its traces are often distinct upon the collar of the coat after the fingers have been passed through the hair. The same flour-like, whitish and grayish scales are distinct and plentiful among the hairs to which they cling, and they can also be recognized over the scalp-surface when the latter is inspected with care. Itching is often marked. The scalp may be scratched and torn by the nails, and is in some cases reddened and thickened. In certain cases there develops, sooner or later, the symptoms of dermatitis seborrhoica.

Other local causes of alopecia are found in various inflammatory disorders of the scalp, such as psoriasis and eczema; in scleroderma, morphea, lupus erythematosus, and syphilitic, tuberculous, and other destructive lesions; in some forms of folliculitis, in which the follicle and surrounding tissue are destroyed by suppuration; in ringworm, favus, and other parasitic affections of the scalp; in traumatism, which may occur as a bruise or be the result of scratching or rubbing; after drug ingestion (thallium acetate); and after exposure to the *x*-rays.

The forms of alopecia described above as encountered upon the scalp may also be found upon other portions of the body, as in the axillæ and pubes; and these also in variable degrees.

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[1] Jour. Cut. Dis., 1900, xviii, p. 352.

[2] New York Med. Jour., 1895, lxii, p. 525.

Alopecia Senilis.—The senile condition is by no means a synonym for baldness. Many men of advanced years are vigorous and have no loss of hair on the scalp, an abundant pilary growth, grayish and at times silvery white, covering the cranial surface. The alopecia occurring in old age, whether upon the vertex, so as to produce a tonsure like that of the priest, or limited to the frontal region, or so extensive as to involve nearly the entire calvarium, leaving a fringe of hairs at the occiput and temples merely, is always remarkable for its symmetry. There is, hence, a certain degree of dignity added to the appearance of the head that an asymmetrical loss of hair does not produce. It may occur at varying ages of advanced life, and is frequently traceable to an early pityriasis steatoides.

The bald surface, as a rule, is smooth and shining, the atrophy of the pilary system corresponding to that noticeable in other structures of the aged; it is occasionally the seat of a seborrhea oleosa. The hair-follicle, with its accessory sebaceous glands, and occasionally the skin itself, are often in a state of atrophy, though there may be dilatation of the sebaceous glands. There is commonly blanching of the hairs, which are shed gradually, as also of those which remain, though canities is not constant. This condition is much less frequent upon the surface covered by the beard and pubic and axillary hairs, where, according to Michaelson, the hairs in advanced years are often denser than at other periods of life.

Etiology and Pathology.—In alopecia prematura the obvious causes are assigned different weight by different authors. Indoor occupations, and the wearing of stiff hats, which operate not only by the exclusion of sunlight and air, but also by constriction of the scalp about the temples, are factors in etiology. The daily application of cold water to the scalp has not been demonstrated to be causative. A variety of alopecia in which the hair becomes dry, lusterless, and faded, and in which the scalp over the region of the occipito-frontalis aponeurosis becomes stiff and adherent, was described by Harding[1] as occurring after undue exposure to the rays of the sun without head-covering.

In alopecia prematura idiopathica heredity is a factor in many cases. Jackson found 10 per cent. only uncomplicated with pityriasis or seborrhea. In those cases not so complicated, the resemblance to senile alopecia is marked.

As alopecia pityrodes is caused by seborrhea or some form of pityriasis, the direct cause must be looked for in these diseases. The major portion of cases occur before the thirtieth year. Jackson[2] found 644 out of 1200 cases before the thirtieth and only 113 after the fortieth year. White[3] found the disease developing before the twenty-fifth year in 67 per cent. in men and 34 per cent. in women. While baldness occurs much more frequently in men, the records of all show a preponderance of women who apply for treatment for hair-loss; according to White's statistics, in the proportion of 56 to 43.

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[1] Jour. Cut. Dis., 1911, xxix, p. 166.

[2] Loc. cit.

[3] Jour. Amer. Med. Assoc., 1910, lv, p. 1074.

Alopecia has been explained by a number of writers as due to toxins elaborated in the system. Parker,[1] for example, has separated from expired air a trichotoxicon believed to originate in the residual air left in the upper portion of the lungs of men living a sedentary life. With the product obtained, this author seems to have produced alopecia in pigeons. Meyer believes that the intestinal tract furnishes a similar toxic agent.

Alopecia senilis occurs much more commonly in men than in women, probably in consequence of difference in headdress and the care bestowed upon the scalp. It is attributed to the general atrophic changes which take place in the aged, these changes being exaggerated in the scalp. This atrophy apparently does not explain the cases, often classed as senile alopecia, occurring in men under sixty or seventy years of age, who in all other respects are vigorous. The same causes operative in alopecia pityrodes play a part in some senile cases. Elliot[2] states that the area of baldness corresponds to the area occupied by the epicranial aponeurosis, a structure devoid of muscular fibers; and suggests the possibility of this arrangement being a factor, through retardation of the return flow through the veins and lymphatics, on account of the relative immobility of the structure unless mechanically exercised. Pressure of this aponeurosis is said to be a factor.[3] The histology shows varying grades of atrophy of all the structures of the skin and appendages.

Michaelson found endarteritis obliterans of the larger bloodvessels and partial obliteration of the capillaries of the hair-follicles. The follicles were diminished, appeared conical, and had patulous mouths. They were filled with loose, horny masses and lanugo hairs. The muscles showed fatty and cloudy degeneration. The sebaceous and coil-glands showed no changes, but there was a cellular infiltration about the latter. Unna largely confirms these findings, and observes little distinction between alopecia senilis and alopecia seborrheica.

Treatment.—In alopecia, the underlying conditions, local or systemic, must be treated by measures appropriate to each case. Elliot, Jackson, Kreuzfuchs, and others call attention to the great importance of prophylaxis. Early and persistent scalp-massage; permitting the hair to grow at some length, as in the case of women, who preserve the hair, as a rule, better than men; and the wearing of loosely fitting head coverings are of value. The use of brushes and combs in common by members of one family is to be forbidden. The same rule applies to these utensils in hotels, parlor-cars, public resorts of all kinds, and hairdressing establishments. Respecting the covering of the scalp with hats, writers have called attention: (1) to the consequent exclusion of light and air; (2) to pressure upon the circlet above the ears and about the temples, whereby the vascular supply of the vertex is impeded (it is here and over the temples where the thinning of the hair commonly becomes first apparent); (3) to the consequent hypersecretion of sweat (Meyer).

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[1] Jour. Amer. Med. Assoc., December 23, 1899, p. 1596.

[2] The Anatomic Factor in the Production of Baldness. George Elliot, Jour. Amer. Med. Assoc., March 29, 1902, p. 814.

[3] Jackson and McMurtry, p. 79.

In the management of alopecia, the general health should always be considered, and any condition interfering with the nutrition of the scalp should be removed. Cod-liver oil, the ferruginous tonics, and the hypophosphites are indicated in many cases.

The following general considerations are worthy of attention in many cases: Massage of the scalp, practised by the fingers once or twice daily in such a manner as to influence the subdermal structures, is useful. A pillow filled with hair or other equally firm material should be preferred to the feather pillows in common use, and in which the scalp is often too warmly and too deeply cushioned. In the case of women, the wearing of artificial hair should be interdicted, as well as the use of the "crimping-iron" and the curl-paper. Sharp hat- and hair-pins thrust deeply between the hairs are often a source of serious damage. In all patients the access of sunlight in moderation and fresh air is needful for the vigor of the hairs of the scalp. Disuse of the brush and preference for the comb in arranging the hairs on the head of women are responsible for the hair-loss in many instances. Every scalp from which the hairs are falling requires daily gentle, systemic friction with a hair-brush, the bristles of which penetrate to the scalp-surface and stimulate gently without wounding or irritating. Faradization and electricity, being as a rule less systematically available, may be regarded as useful adjuvants in the hands of the expert. Singeing the hairs is, without question, harmful. The hat should be light and well ventilated.

Local treatment is of importance in nearly all cases, and in general is directed toward stimulating the nutrition of the hair-follicle by producing in its periphery a species of transitory and artificial hyperemia. This result is accomplished by the local employment of one or more of the alcoholic, oily, alkaline, and other stimulating applications described below.

Local treatment may often be preceded by shampooing with either the Sarg fluid soap or combinations of glycerin, alcohol, and sapo mollis (tincture of green soap); or with eggs, to meet the requirements of individual cases. The shampooing may be practised every few days, once weekly, or once every two or three weeks, according to the needs of the case. The scalp after all such shampooings should be anointed with lanolin, plain or salicylated; vaseline; equal parts of lanolin, glycerin, and rose-water; the oil of benne; or scented castor oil. In obstinate cases the nail-brush may be used vigorously over insensitive scalps at the time of shampooing. A good working plan is to have a shampoo employed once in seven to twenty-one days, this to be followed by the application of the cream or ointment deemed indicated, and on each succeeding or every alternate night apply a lotion. The ointment-bases named above may often be medicated advantageously with sulphur, resorcin, chrysarobin, tar, cantharides, or mercury. Instead of ointments, lotions containing cantharides, phenol, capsicum, resorcin, mercuric chlorid, ammonia, or nux vomica may be used. White[1] finds euresol, bichlorid of mercury, tannic acid, and chloral hydrate most efficient. Care should be taken to avoid unpleasant staining or dyeing of the hair by both resorcin and chrysarobin. The former should never be compounded with ammonia. Formulæ for lotions and salves to be used in this way are appended:

℞—Hydrarg. chlorid. corros.,		gr. v;			33
Alcoholis,		℥ij;		60	
Acid. acet. dil.,		ʒij;		8	
Glycerin.,		℥ss;		15	
Aq. ros.,		℥vj;		180	M.

℞—Hydrarg. chlorid. corros.,		gr. iij;			20
Tinct. cantharid.,		℥ss;		15	
Ol. amygdal. dulc.,		ʒj;		4	
Spts. rosmarin.,		℥j;		30	
Alcoholis,		℥ij;		60	
Aq. dest.,	q. s. ad	℥vj;	q. s. ad	180	M.

℞—Sulphur. præcipit.,		ʒj;		4	
Lanolin.,					
Glycerin.,					
Aq. ros.,	āā	ʒijss;	āā	10	M.

℞—Hydrarg. chlorid. mit.,		℈iv;		5	33
Hydrarg. ammon.,		℈ij;		2	66
Vaselin.,	ad	℥j;	ad	30	M.

(Bronson.)

℞—Resorcin.,		ʒj;		4	
Quinini (alkaloid),		gr. xv;		1	
Ol. ricini,		♏x–xxx;		0	66–2
Alcoholis,	ad	℥iv;	ad	120	M.

(Stelwagon.)

℞—Cantharid. tinct.,		ʒij;		8	
Capsici tinct.,		♏xv;		1	
Alcoholis,		℥jss;		45	
Aq. ros.,	ad	℥v;	ad	150	M.

℞—Liquor carbonis detergens,		ʒij;		8	
Resorcin.,		ʒij;		8	
Alcoholis, 50%,	q. s. ad	℥viij;		240	M.

Jackson has had best results in idiopathic premature alopecia with the following:

℞—Pilocarpin. hydrochlor.,		grs. xx;	1	33
Spts. odorati.,		ʒiv;	16	
Aq. ros.,				
Alcoholis,	āā q. s. ad	℥viij;	240	M.

Where the hair is unusually dry, Saalfeld employs:

℞—Tannobromini,		gr. xv;	1	
Bals. Peruv.,		ʒss;	2	
Adipis colli equini,	ad	℥j;	30	M.

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[1] Loc. cit.

Where the hair is oily, particularly in women, Jackson endorses the following, devised by Sabouraud:

℞—Alcoholis, 90%,		℥viij;	240	
Spts. lavandulæ,				
Spts. etheris,	āā	ʒvjss;	26	
Pilocarpin. hydrochlor.,		grs. iv;		25
Liquor ammoniæ,		ʒj;	4	M.

In similar cases, Leslie Roberts (personal communication) recommends:

℞—Acid. salicylic.,		ʒss;	2	
Sulphur præcip.,		ʒj;	4	
Hydrarg. chlorid. corros.,		gr. iv;		25
Alcoholis,		℥ij;	60	
Aquae,	q. s. ad	℥iv;	120	M.

Richema and Staganovitch advise:

℞—Acid. lactic,	ʒij–iv;	8–16	
Alcoholis,	℥j;	30	
Aq. ros.,	℥j;	30	M.

To be applied with absorbent cotton, using friction until the surface is reddened.

C. J. White[1] has found the following most useful:

℞—Hydrarg. chlor. corros.,		gr. iv;		25
Euresol.,		ʒij;	8	
Spts. formicari,		℥j;	30	
Ol. ricini,		ʒj–iij;	4–12	
Alcoholis, 70%,	q. s. ad	℥viij;	240	M.

Walsh advocates: lysol, 1 part; alcohol, 8 parts; and rosewater, 25 parts, adding cantharides if desired. He also recommends a lotion composed of:

℞—Acid. salicylic.,		ʒiij;	12	
Phenolis,		ʒj;	4	
Ol. ricini.,		ʒiij;	12	
Alcoholis,	q. s. ad	℥vj;	180	M.

The addition of acetic acid to a scalp-lotion seems to favor penetration of other remedies. Further suggestions regarding the detail of treatment of alopecia and the special remedies recommended for alopecia pityrodes are given under pityriasis simplex capitis and pityriasis steatoides.

Prognosis.—The senile and many of the so-called premature alopecias are practically remediless, though in all forms further loss of hair often can be prevented or greatly retarded by proper treatment. The symptomatic alopecias, in which there is destruction of the hair-follicle, as in lupus erythematosus, syphilitic ulcers, favus, and some forms of folliculitis, are permanent. Those due to systemic disorders and local inflammations are usually temporary. In alopecia pityrodes persistent treatment will prevent further loss of hair, and in recent cases may produce a new growth.

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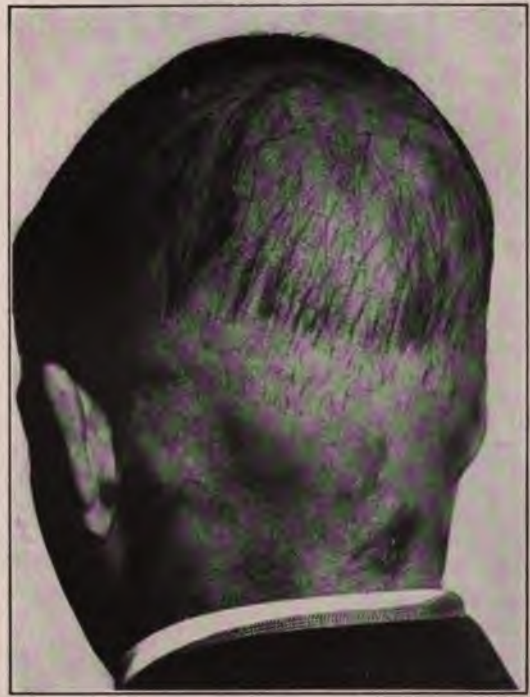
[1] Loc. cit.

Alopecia Areata.—**Synonyms**: Porrigo decalvans, Tinea decalvans, Area celsi, Area johnstoni, Alopecia circumscripta. Fr., *Pelade*.

Definition.—Alopecia areata is characterized, as a rule, by the loss of hair in circumscribed patches, the latter being round, oval, or irregular in shape, of varying size, and accompanied by little or no evidence of inflammation. Not infrequently, regions other than the scalp are affected, and rarely a total loss of hair occurs.

Fig. 420



Alopecia areata showing "exclamation point" hairs.

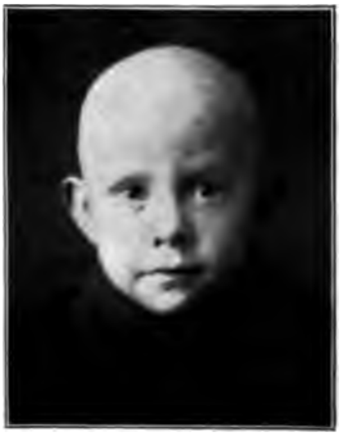
Symptoms.—Alopecia areata is a disease affecting the hairy surfaces of the body, often limited to the scalp, but at times generalized. It is characterized at the outset by the occurrence of one or several circumscribed, round, or oval areas completely devoid of hair, and exhibiting few if any changes in the affected part. The hair-loss is limited usually to the scalp, but may occur upon the beard, the genitalia, axillæ, brows, eyelids, and general surface of the body. Cases occur, especially in early childhood, in which the closest scrutiny with a glass fails to detect a single filament of hair upon any portion of the skin.

The disease commonly manifests itself by the sudden and complete

vague neurotic sensations precede the hair-loss. In yet other cases the loss of hair is gradual, the patch attaining large dimensions in the course of two or three weeks. Less frequently, an area of baldness may continue to extend peripherally for many weeks. Instead of one area, there commonly are several, which may develop simultaneously or at varying intervals.

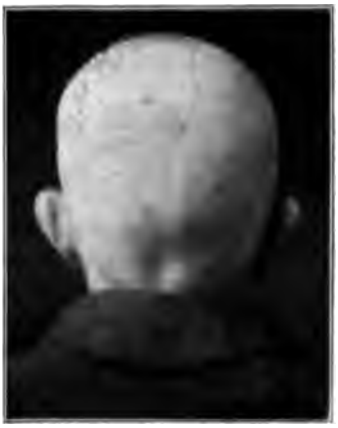
The patches may be round, oval, circinate, or irregularly shaped, and may vary in size from that of a small coin upward. They may be so numerous as to disfigure the entire scalp, but though they touch at the borders they can scarcely be said to coalesce, as the individual areas are usually recognizable. Extension, however, may occur by coalescence of patches as well as by the peripheral enlargement of single areas. The surface of the scalp is smooth, soft, whitish, and usually destitute of hair. The affected scalp may be thinner and more lax than normal, and often is depressed slightly below the level of the surrounding skin; in rare instances it is tumid and slightly reddened.

Fig. 423



Alopecia areata.

Fig. 424



Alopecia areata.

The hairs at the periphery of the patches that have attained their full development are normal in every way and are firmly implanted in their follicles, but at the borders of areas which are still spreading the hairs are loose and fragile and often broken off near the surface, thus leaving short stumps, which exhibit at the bulb a spade-like extremity or an attenuated point, the non-atrophied shaft thus contrasting with the atrophied portion implanted below the cutaneous level. Crocker likens their shape to that of the exclamation-point. Newly formed areas may be covered in greater or less degree with these characteristic hairs, and patches that are spreading show them at the margins, but by the time the process has become stationary these hairs disappear.

As a rule, there are no subjective sensations, though the affected areas may be the seat of slight or severer itching, and are nearly always less sensitive to irritating applications than the surrounding normal parts. In some cases there may be an accompanying cephalalgia, parasthesia, and formication of the skin of the scalp and other regions.

The course of the disease is variable and may persist for months or years without apparent change; or new patches may form, while those of a later date gradually regain, wholly or in part, their pilary growth, which, however, may be lost repeatedly on the same area. Shifting areas of baldness may in this manner invade the entire surface of the scalp, which yet at any moment of time may exhibit a loss of but part of its hairy covering. In other cases, patches occur in rapid succession, and in a comparatively short time the entire scalp has been swept clean. When the hairs begin to reappear, there is commonly a fine, downy growth on the affected area, later replaced by a crop of thicker and stronger, whitish hairs, which are always succeeded, in cases terminating favorably, by a growth of hairs as well colored, as vigorous and as persistent as any which were lost. An odd appearance is often presented by patients who are improving when the young and white new hairs contrast vividly with the dark shade of those on the unaffected scalp.

In young subjects the course of the disease is usually toward a favorable result. There is hope, as a rule, when even the downiest and thinnest growth, requiring a good light and a glass for recognition, can be appreciated. Even when so feebly attached that these hairs are removed with ease by the fingers or brush, and when they spontaneously fall, they may be replaced by a succeeding crop of stronger hairs, which eventually persist. In serious cases, usually after the forty-fifth year of life, and in those of long standing, there may result atrophy of the hair-follicles, in which case the baldness is permanent.

Among the unusual features of the disease may be mentioned the occurrence of alopecia in bands or streaks; at the site of an injury or along the course of a nerve; or over the entire body, removing even the finest lanugo-hairs. Universal alopecia may occur suddenly, or as the result of a gradual thinning of the hair; or it may follow the existence of the disease in characteristic areas. This variety of alopecia, which is fortunately rare, usually occurs after the middle period of life, and is termed by some writers *malignant alopecia areata;* but it may develop at any age.

Odd-looking effects are produced when in the course of the disease, with and without the development of patches in the scalp, the moustache on one side of the face falls, or the hairs of one eyebrow or one eyelid; or even when all the hairs are lost from both brows and lids on each side of the face.

In some instances alopecia areata is associated with other cutaneous diseases. It not infrequently occurs with ringworm (*Cf.* section on Ringworm for authors quoting such cases). Cases associated with vitiligo are reported by Besnier, Duhring, Freeland, Du Castel, and others.[1] Coincident dystrophy of the nails has been observed by Darier and Le Sourd,[2] Crocker, G. H. Fox,[3] Sequeira,[4] Weber,[5] Knowles[6] and others. Other conditions associated with alopecia areata are scleroderma, thyroid disease, and moniliform hair.[7] Its association with syphilis has been reported by numbers of observers, including Pusey,[8] Sabouraud,[9] Dubois,[10] Pernet,[11] Sequeira,[12] Adamson,[13] the author, and others.

There is reason to believe that the disease has a stadium of evolution and involution, though its exact limits are not known. Few individuals fully recover the hair in less than one year. The majority attain the desired end within a period of two years. These limitations, however, apply to the asymmetrical forms of the disease in the relatively young. The symmetrical alopecia areata of the middle-aged is a far more formidable affection, though in some of these cases, when the loss is recent, proper treatment will restore the hair.

Few diseases are the source of greater mental distress than those of the class under consideration. The fear that they will be suspected of having syphilis, and the social ostracism which the deformity entails produces a morbid mental state, which is especially noticeable in nervous women.

Mewborn[14] has introduced the term *Trichopathophobia* to designate the fear of disease of the hair.

Ophiasis is a form of alopecia occurring more frequently in childhood (Sabouraud states limited to childhood), in which a band two or three inches broad extends over the nape of the neck and around the lower margin of the scalp to the temples in front.

Etiology.—In the modern acceptation, the words "alopecia areata" describe merely a loss of hair occurring at first in restricted areas, which may become generalized, and which without question may spring from varying causes. The conflicting views of the nature of the disease have been concerned with the etiological factors supposed to be responsible for the results, and have demonstrated the fact of their multiplicity. The disease is probably seen more frequently in children, but may occur at any age. It affects the sexes in about equal proportions, rather more in persons having dark than in those with light hair, and in all irrespective of social condition. Of the partial and asymmetrical forms, the larger number occur in young subjects, from childhood to early adult life. The severe and generalized forms are encountered more often in middle-aged persons. In the latter class especially the disease is observed occasionally to follow the obscure disorders of the nervous centers due to undue excitation, sudden or prolonged.

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[1] Jour. Cut. Dis., 1909, xxvii, p. 465.

[2] Annales, 1898, s. iii, p. 1009.

[3] Jour. Cut. Dis., 1902, xx, p. 574.

[4] Brit. Jour. Derm., 1912, xxiv, p. 122.

[5] Ibid., p. 204.

[6] Jour. Cut. Dis., 1911, xxix, p. 27.

[7] Walsh, Brit. Med. Jour., 1902, i, pp. 812, 883.

[8] Jour. Cut. Dis., 1909, xxvii, p. 470; ibid., 1911, xxix, p. 310.

[9] Annales, November, 1910, p. 545.

[10] Ibid., p. 555.

[11] Brit. Jour. Derm., 1911, xxiii, p. 183.

[12] Ibid.

[13] Ibid., p. 354.

[14] Jour. Amer. Med. Assoc., vol. 1, p. 19.

Relative to heredity, Sabouraud[1] found the disease directly inherited in 11 out of 100 cases. It not infrequently occurs in several members of a family, such instances being recorded by Schamberg[2], Kingsbury,[3] Whitehouse,[4] the author, and many others. The three most prominent theories as to its origin are the neuropathic, the parasitic, and the toxic. The neuropathic origin of a large number of cases of alopecia areata is indisputable and verified in every clinical experience. Blows on the head, not rarely resulting in well-marked scars visible on careful inspection of the affected regions; nervous shock (fright, lightning stroke, great and prolonged anxiety, grief); traumatism of other regions than the scalp-surface; prolonged and severe toil in close apartments, these again and again have produced typical clinical symptoms of the disease. Max Joseph[5] produced baldness in the ears of cats and rabbits by excision of the second cervical ganglion. Surgical operations, for example those in severe mastoid disease, may be followed by alopecia areata. Sequeira[6] reports a case of proven nervous origin. The most important among reflex causes were brought out by Jacquet,[7] who found that alopecia areata is associated frequently with defective teeth or other sources of irritation of the cutaneous nerves. Rousseau and Decelle,[8] Sequeira,[9] and many others have confirmed Jacquet's hypothesis of reflex origin. Some observers, however, even after careful observation, have failed to discover any connection. Reflex irritation aside from the dental irritation above noted, such as errors of refraction, with consequent headache, may be associated with alopecia areata.[10] In upholding the neurotic theory, Sir Dyce Duckworth[11] states that it would be difficult to conceive either a parasitic or a toxic theory for a case of universal alopecia following shortly after an injury to the head produced by the patient being thrown out of a dog-cart. Permanent loss of all the hairs on the body of such a patient resulted. Numbers of these examples he said could be recounted.

The etiological part played by syphilis in the disorder is important. Sabouraud[12] states that extensive alopecia is more commonly due to syphilis, either acquired or hereditary, than to any other single disease. Dubois,[13] Pernet,[14] Sequeira,[15] Adamson,[16] Pusey,[17] and others have reported either the coincident occurrence of alopecia areata and syphilis, or the presence of a positive Wassermann or positive results following antisyphilitic treatment in these cases.

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[1] Annales, 1911, p. 65.

[2] Jour. Cut. Dis., 1909, xxvii, p. 219.

[3] Ibid., p. 403.

[4] Ibid., p. 457.

[5] Monatshefte, 1886, v, p. 483.

[6] Trans. XVII Internat. Cong. of Med., London, 1913, Sec. 13, Part 2, p. 142.

[7] Annales, 1902, s. iv, iii, p. 97.

[8] Bull. et. Mem. Soc. med. de. Hôp. de Paris, January 21, 1909, p. 72; ibid., 1910, p. 536.

[9] Loc. cit.

[10] Whitfield. Trans. XVII Internat. Cong. of Med., London, 1913, Sec. 13, Part 2, p. 142; and Kingsbury, Jour. Cut. Dis., 1909, xxvii, p. 211.

[11] Trans. XVII Internat. Cong. of Med., London, 1913, Sec. 13, Part 2, p. 140.

[12] Annales, November, 1910, p. 545.

[13] Ibid., p. 554.

[14] Brit. Jour. Derm., 1911, xxiii, p. 183.

[15] Loc. cit. and ibid., p. 256.

[16] Ibid., p. 354.

[17] Loc. cit.

In favor of the parasitic theory of the origin of this disorder may be mentioned those cases in which the disease begins as a small spot, and gradually and slowly spreads peripherally, with later multiple spots beginning as satellites and going through the same course. Another point in its favor is the occurrence of the disease in epidemics in public institutions and elsewhere. A remarkable example of this sort is recorded by Bowen,[1] in which 63 out of 69 children in one institution were attacked. More recently Colcott Fox[2] reported a small epidemic of alopecia areata, 21 girls between the ages of nine and fourteen being affected; and Haldin Davis[3] recorded an epidemic which recurred. In the first instance 174 girls under the age of fourteen years were attacked out of a total number of about 300 in an orphanage. Five months after the complete disappearance of the disorder, a second epidemic began, which attacked 42 of the inmates. Both epidemics yielded soon to vigorous antiseptic treatment and isolation. The distinguishing feature in the recurrent cases was that the patches were smaller than those ordinarily found in the disease. Careful examination ruled out ringworm. Other epidemics have been reported by Hillier (quoted by Colcott Fox), and others in France, Germany, and elsewhere. In a later report Bowen[4] found the scalp normal in patients previously affected with epidemic alopecia. Sabouraud[5] found much of the so-called alopecia areata in schools to be a symptomatic alopecia in patches, preceded and caused by impetigo. Eichhorst,[6] Thin,[7] von Schlen,[8] Robinson,[9] Walker and Rockwell,[10] Sabouraud,[11] and others have found various micrococci, microbacilli and *Staphylococcus epidermidis albus* (Welch) in the hair follicles of the affected patches of alopecia areata. In some of these the organisms have been cultivated, but inoculation experiments have been almost uniformly unsuccessful. Sabouraud, however, in inoculating pure cultures of his microbacillus, produced typical areas in calves, rabbits, and guinea-pigs. In large numbers of attempted inoculations with human beings, Jacquet and all others have failed to reproduce the disease. The epidemics above referred to are suggestive of the theory that there may be a small group of cases that are parasitic, but the parasite remains to be discovered.

The toxic theory of the disorder appears plausible for certain cases, and is illustrated by the production of baldness through injection of thallium.

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[1] Jour. Cut. Dis., 1899, xvii, p. 400 (Two epidemics discussed. The first was recorded by Putnam, Archives of Pediatrics, 1892, ix, p. 595).

[2] Brit. Jour. Derm., 1913, xxv, p. 51.

[3] Ibid., 1914, xxvi, p. 207.

[4] Jour. Cut. Dis., 1915, xxxiii, p. 343. Epidemic alopecia in small areas in schools, regiments, etc.

[5] La Clinique, No. 9, p. 1906.

[6] Virchow's Archives, 1899, lxxviii, p. 197.

[7] Trans. Royal Society, 1881–82, xxxiii, p. 247.

[8] Virchow's Archives, 1885, xciv, p. 327.

[9] Morrow's System, vol. iii, p. 862.

[10] Scottish Med. and Surg. Jour., 1901, viii, p. 12.

[11] Annales, 1896, s. iii, vii, p. 253.

Meacham[1] records the regrowth of hair during pregnancy in a case of total alopecia, with a relapse after the establishment of menstruation. Sabouraud,[2] in a recent study of alopecia areata, connects the chronic and severe cases with hyperthyroidism, and further calls attention to the coexistence of ovarian trouble and alopecia areata. Engman and Mook[3] record alopecia areata coincident with albuminuria and hyalin casts. Pöhlmann,[4] after a critical review, concludes that alopecia areata must be regarded as a symptom complex resulting from infection, disturbance of innervation, or intoxication.

Pathology.—The anatomical lesions in alopecia areata have been studied by many investigators, and in the main their findings agree. The interpretation placed upon these findings varies, however. Robinson[5] believes the process to be an inflammatory one, which is proven by a comprehensive study made in all stages of the disorder. In a small patch of one week's duration he found signs of inflammation in the papillary layer; while lower in the corium there were marked inflammatory changes, evidenced by considerable round-cell, perivascular infiltration in limited areas. The lymphvessels were dilated, and in some places contained a fibrous coagulum. The sebaceous and sweat-glands and subcutaneous tissue were normal. The hair-follicles were either empty, or contained normal or lanugo hairs. In some cases the hair-papillæ were absent, the lower part of the contents of the follicles consisting of pigmented epithelial cells, which formed a small hair-shaft. A lanugo hair formed farther up in the follicle. Occasionally, there was found a splitting-up and fibrillation of the shaft and an occasional stub hair. In older cases inflammatory reaction could always be observed. In one case of permanent alopecia the hair-follicles and sebaceous glands were destroyed and there was some atrophy of all the tissues of the corium. The hairs themselves show varying stages of atrophy. Giovannini[6] in the main confirms these findings. Most of the other observers have made similar findings, but the character of the cellular infiltration has different interpretations. This has been looked upon as composed variously of leukocytes (Giovannini), connective-tissue cells (Unna), lymphocytes, and mast-cells. Sabouraud believes these findings suggest a disturbance of the nutrition of the follicle through deep-seated inflammation about the hair-papillæ, which early interferes with the growth and later leads to atrophy and destruction both of the hair and of the follicle.

Diagnosis.—Alopecia areata is to be distinguished from vitiligo of the hairy portions of the surface by the preservation of the pilary growth in the disease last named, the filaments, moreover, having usually a whitened look, due to the absence of pigment.

From ringworm of the scalp the disease in question is differentiated by the suddenness of its onset; the absence of stumps of hairs, scales, crusts, and evidences of irritation in the involved area; the whiteness, smoothness, and complete baldness of the latter; and, above all, by the failure to detect with the microscope the presence of a vegetable parasite. Ringworm and alopecia areata may coexist. In cases of so-called "bald ringworm," the diagnosis must rest upon the microscopical findings.

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[1] Brit. Jour. Derm., 1912, xxiv, p. 272.

[2] Annales, 1913, iv, p. 140.

[3] Jour. Cut. Dis., 1913, xxxi, p. 268.

[4] Archiv, 1913, cxiv, p. 633.

[5] Loc. cit.

[6] Annales, 1891, s. iii, ii, p. 921.

The asymmetrical patches of pityriasis steatoides of the scalp are recognized by the presence of the fatty plates pasting the hairs to the scalp-surface, as well as by the slow and very gradual onset of the disorder.

From the patchy alopecia occurring in the early stages of syphilis, alopecia areata is differentiated by the patches in the former being less sharply defined; by the presence in the patches of tufts of hair, presenting a mangy or worm-eaten appearance; and by the general lack of luster and the low state of nutrition of all the hairs of the scalp. There may be present also the concomitant symptoms of syphilis.

The alopecia occurring in lupus erythematosus, pseudo pelade, and favus, the late stages of syphilis, and other scar-leaving disorders is readily differentiated by the presence of atrophy and scar-formation.

Treatment.—One necessarily views with distrust all treatment for that disease which in the course of months or years usually terminates in spontaneous recovery, and in the meantime may bid defiance to each and every therapeutic measure. Nevertheless, persistent and hopeful management of even apparently desperate cases is occasionally rewarded by such brilliant results that, however slight may be the foundation for a belief in the value of the therapy employed, it deserves recognition and trial.

The hygienic management of every case is a matter of importance. The general condition of the nervous system should be considered, and may call for change in the habits of working, eating, resting, and exercising. Iron, quinin, nux vomica, cod-liver oil, phosphorus and the hypophosphites, arsenic, and strychnin are often indicated and used with great benefit. Crocker advocates the administration of the nitrate of pilocarpin, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008 to 0.016) at night, a flannel night-dress being worn subsequently. Pilocarpin by hypodermic injections into the scalp, in doses from $\frac{1}{30}$ to $\frac{1}{10}$ grain (0.002 to 0.006), is also praised.

Any associated disease or morbid condition should receive appropriate attention. The number of cases presenting some evidence of syphilis makes the treatment of that disease a factor in the therapy of numerous cases of alopecia areata.

There are few patients who are not benefited by daily salt-and-water bathing of the entire body-surface, followed by brisk friction, especially over the spinal region. In the case of children, this treatment must be conducted by a skilled hand. When practicable, the cold douche is preferable.

In all cases in which the scalp is involved in either sex, and in which the special hypochondriasis of the disease is developed, a wig should be worn for the sake of its mental effect upon the sufferer. Its use, however, should be limited to social occasions, as the persistent wearing of a peruke indoors seems to lengthen the course of the disease.

The indications for local treatment are met by the employment of stimulating and antiparasitic preparations. The affected parts are to be bathed daily in water as hot as can be tolerated, then dried and rubbed with a stimulating lotion. After the lotion dries it is well to apply an oil or simple ointment. The articles usually employed are alcohol, ether, resorcin, formaldehyd, turpentine, ammonia, camphor, cantharides, phenol, oil of mace, croton oil, tincture of nux vomica, tincture of capsicum, tincture of aconite, castor oil, tar, iodin, sulphur, and the mercurials. All frequently fail. Several of these substances in combination seem at times to be of service.

The following is a formula the ingredients of which may be varied to suit the indications in different cases:

℞—Ol. ricini,	f℥ss;	15	
Phenolis,	ʒj;	4	
Tinct. cantharid.,	℥ss;	15	
Ol. rosmarin.,	gtt. xv;	1	
Alcoholis,	ad f℥iv;	ad 120	M.

Sig.—For external use over the scalp with friction.

Formaldehyd, in solution of 0.5 to 2 per cent., is sometimes efficient. It should be used with care, however, as it has occasioned severe dermatitis, and in several instances has given light hair a green color.

Jackson recommends aqua ammoniæ fortior, applied once or twice daily to the bald areas. Speedy return of hair in a patch of alopecia areata has followed the application of pure creosote and also of trikresol to the denuded surface, resulting in moderate vesication. The latter preparation may be applied in from 50 per cent. to full strength every two weeks on localized patches, usually by the physician. The spirit of turpentine, pure phenol, and acetic acid have similarly been employed; but caustic applications are to be used with caution and over limited areas at a sitting.

By many experts, having in mind the probability of a parasitic origin, epilation is practised to the extent of removing all the loosened hairs and a narrow zone of sound hairs about each patch. The remedies selected for application are of the order of parasiticides; for example, mercurials, sulphur and its compounds, chrysarobin, pyrogallol, and iodin.

Repeated blisterings of the scalp with cantharidal collodion, croton oil, spirit of green soap, and petroleum have also been employed with success. The ointment of chrysarobin has the disadvantage of staining not only the remaining hairs, but often also the face, in consequence of the frequency of a transmission to that locality through the medium of the hands. When patients, however, consent to the use of chrysarobin it is worthy of trial, as its application has been followed by a vigorous growth of new hair. Hodara[1] states that the application of a 30 per cent. preparation of chrysarobin for from two to eight weeks is followed by vascular and inflammatory changes which lead, through proliferation of prickle-cells and connective-tissue cells, to the formation of new follicles, new sebaceous glands, new papillæ, and new hairs. André employed ten hypodermic injections of pilocarpin hydrochlorid in ⅛ grain (0.008) doses, which resulted, in the case of a middle-aged woman affected with total symmetrical baldness, in an abundant growth of hair. Mercuric chlorid has similarly been employed.

Phototherapy has been used by Finsen, Forchhammer, Jersild, Leredde, Török, Schmidt, and others, including ourselves, in alopecia areata, with, on the whole, very favorable results.[2] For circumscribed areas the light-treatment gives better results apparently than are obtained by other methods, though it fails in some instances. It has been used successfully where a number of large areas were present; but in such cases the treatment is tedious, and, as a rule, does not give such good results.

Faradization of the scalp with a stiff wire-brush, pushed to the point of producing moderate hyperemia, has been followed by excellent results. Jackson[3] found treatment by means of Piffard's iron spark-gap lamp beneficial.

Wilson recommends:

℞—Ol. amygd. dulc.,	fʒj;	4	
Tinct. capsici,	fʒij;	8	
Aqua ammon. fort.,	f℥j;	30	
Spts. rosmarin.,	f℥v;	150	
Ol. limon.,	fʒj;	4	M.

Another stimulating application is:

℞—Ol. terebinth.,					
Ol. ricini,	āā	f℥ss;	āā	15	
Tinct. origani,		fʒj;		4	
Ol. camphorat.,		f℥j;		30	
Liniment. ammoniæ,	ad	f℥iij;	ad	90	M.

Sig.—For external use with a brush until the scalp is irritated.

Shaving should regularly be practised when the region of the beard is involved, as the deformity is thus rendered less conspicuous; and the bald surface should be stimulated frequently with one or several of the topical applications named above. Alcoholic solutions of resorcin (3 to 20 per cent.), or of mercuric chlorid, ½ to 1 grain (0.033 to 0.066) to the ounce (30.), are to be well rubbed over the patch or patches once or twice daily.

Prognosis.—From what precedes, it will be inferred that, as regards the relief of the baldness, the asymmetrical development of alopecia areata in youth is much more favorable than the symmetrical general disease of middle life, the latter being often remediless. In all cases the practitioner should actively persevere to the end. In no case should any encouragement be given as to complete relief within a year, though exceptionally short careers of the disease are observed at times. The prognosis of the same affection of the beard is quite favorable, the disease, in young men, usually concluding its stadium in the course of about one year, with a favorable termination.

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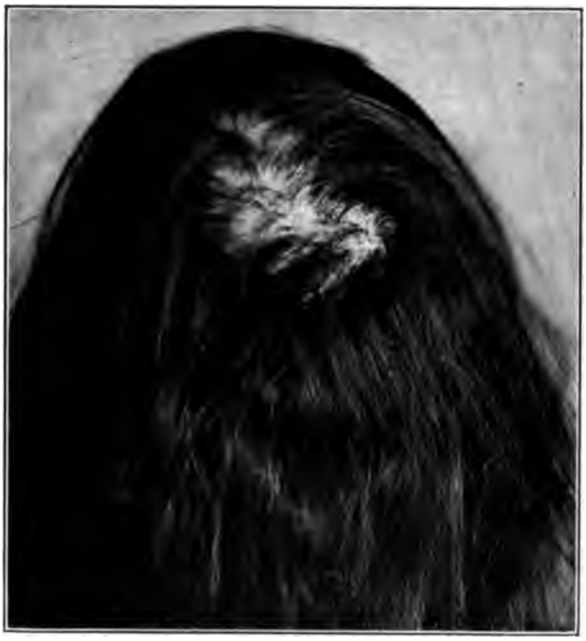
[1] Jour. Mal. Cut., 1903, xv, p. 644.

[2] For bibliography, see paper by Frank Hugh Montgomery, Jour. Cut. Dis., 1903, xxi, p. 529.

[3] Jour. Cut. Dis., 1910, xxviii, p. 18.

Alopecia Cicatrisata.[1]—**Synonyms**: Pseudo-pelade (Brocq), Alopecia circumscripta *seu* orbicularis (Neumann), Alopecie cicatriciélle (Besnier).

FIG. 425



Pseudo-pelade. (Fordyce.)

Definition.—This is a disease characterized by circular, oval or irregular, variously sized patches of alopecia, associated with atrophy of the skin in the areas, unaccompanied by subjective sensations, and terminating in permanent baldness.

Symptoms.—The areas of alopecia vary in size from that of a pinhead to a small coin and larger; are round, oval, or irregular in shape, white or slightly pinkish in color, and present a shining and atrophic appearance. The large plaques are usually formed by coalescence of smaller ones, and may be of the size of the palm of the hand. In these the contour is irregular or angular, and here and there areas still covered with hair extend into denuded patches.

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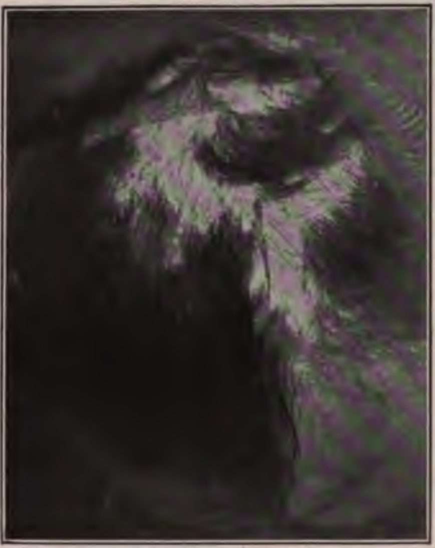
[1] Brocq, Lenglet et Ayrignac, Annales, 1905, pp. 1, 97, 209. An extensive exposition of the subject, with review of the literature and clinical and histological research with illustrations. This article was freely consulted in the preparation of this chapter in the text.

FIG. 426



Alopecia cicatrisata.

The disorder begins insidiously and on any part of the scalp, but usually over the vertex. Commonly, a few lesions are noted, new ones occurring around this point. Occasionally, large numbers appear suddenly. The most common sites are the vertex, the upper part of the occiput, the temporal regions, and the mastoid. The scalp in the areas presents usually a whitish appearance, rarely a rosy-pink at the border. A few reported cases have been erythematous. The skin is ordinarily thin and soft, atrophied, shining, and rather translucent, suggesting the term "onion skin." At times a genuine atrophy, with scar-formation, follows. In some areas epidermic cones are seen plugging the hair-follicles. These may cribble the whole surface, suggesting comedones. There are no "exclamation-point" hairs, which are characteristic of alopecia areata, but the loosened hairs when extracted show a somewhat transparent, more or less thickened, pulpy sheath. At the margins there may be furfuraceous scaling. There are no subjective symptoms, but there may be loss or diminution of the pain-sense.

The disease may complete its evolution in three or four months, or may be temporarily stopped by intercurrent disease. The usual course is one of activity, followed by rest. It may last for many years, but the alopecia never becomes complete. Recoveries have been reported, Brocq has never seen such an example. The disorder may be complicated by seborrhea and pityriasis, and nail-changes have been recorded.

Etiology.—The disease is essentially one of adult life, but has been seen in infancy. It is more common in men than in women. It occurs usually in persons with dark, vigorous, coarse and thick hair. Cultural experiments by Sabouraud and others have been negative. Associated disorders, such as keratosis pilaris, have occasionally been observed. The patients have been nervous and impressionable, suffering from attacks of depression. Dental complications have been frequently described. Metabolism determination in a few instances seems to indicate a reduced utilization of nitrogen. In searching for some etiological factor, every means has been exhausted by the author in the case of several patients without result.

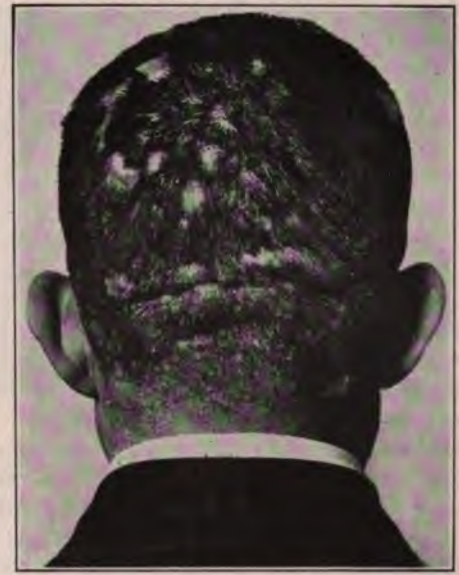
Pathology.—The following histological changes were described by Lenglet, in a study of material taken from three cases: The chief pathological change was noted about the bloodvessels in connection with the hair-follicles, but also in other situations. This consisted of an inflammatory, perivascular, cellular infiltration, involving the capillaries around and between the hair-follicles. In these areas enormous dilatation was present, and the vessels were engorged with erythrocytes and a few leukocytes. A perivascular cellular infiltration, composed of lymphocytes chiefly, with some plasma-cells and mast-cells, occurred. These collections of cells are described as commonly occupying half of the length of the sheath of the follicle, terminating below near the papilla. The cellular collections about the hair are greater in volume than those about the vessels at a distance, and their disposition is said to explain the fall of the hair. In the site of the cellular infiltration there is edema of the collagen and disappearance of the elastin. The epidermis shows chiefly secondary changes, being thinned, with the exception of the horny layer, which is slightly thickened. There is absence of the stratum granulosum, and also of the interpapillary prolongations. The sebaceous glands become partially involved in the infiltrate and atrophy. The sweat-glands are more resistant and only disappear after marked sclerosis. Pigment-cells are scattered throughout the corium.

Diagnosis.—The disease is to be differentiated from favus, lupus erythematosus, the late stages of syphilis, and the terminal stages of folliculitis decalvans. In lupus erythematosus the margins may show dilated sebaceous openings and the entire process may be more inflammatory. Outlying lesions behind the ears or in the ears would be of distinct value diagnostically if present. In certain cases a differentiation is almost impossible without the closest observation. The atrophy and scar-formation following favus may closely simulate alopecia cicatrisata, but the history of a previous inflammatory and crusting disorder, and, if any symptoms of that character are present, the demonstration of *Achorion Schönleinii,* make the differentiation. The scars induced by syphilis are preceded, as a rule, by ulcerative processes, and in addition there may be lesions in other parts. Moreover, the lesion of syphilis is made up of individual component nodules, evidence of which is usually impressed on the scar. Folliculitis decalvans is preceded, as the term indicates, by a suppurative inflammation of the hair-follicle, all evidence of which is absent in pseudo-pelade. In the terminal stages of the former disease the inflammatory process has cleared up and differentiation would be difficult; but the history and the location of the lesions, which are more likely to have irregular outlines, would be of value.

Treatment.—Treatment has very little if any effect on the disorder. In the nervous and debilitated patients the general treatment indicated for such conditions should be employed, and in addition the general management of the case in every respect is most important. Brocq suggests removal of the loose hairs and the alternate use of mercurial and sulphur preparations. Crocker employed an ointment of biniodid of hydrargyrum, 2 grains to the ounce. The local treatment, therefore, resolves itself into the use of preparations that are mildly stimulating, but great care should be exercised not to produce overstimulation.

Prognosis.—From what has preceded, it is evident that the prognosis is unfavorable so far as regrowth of hair already lost is concerned. The disease may progress for only a short period of time and be arrested, or continue for some years, with much loss of hair, before its activity ceases. In no case, however, is complete alopecia accomplished. The disfigurement in many cases is sufficient to produce much distress in the mind of the patient.

Fig. 427



Severe folliculitis of the scalp producing alopecia and folds which somewhat resemble cutis verticis gyrata.

Folliculitis Decalvans[1] (*Quinquaud's disease,*[2] *Folliculite épilante, Acné décalvante*[3]).—The disease described under these titles is a scar-leaving alopecia. The disorder begins with pustules or miliary abscesses, punctiform, pinhead-sized and larger, and involving the hair-follicles. In the early stages the lesions resemble those seen in coccogenous sycosis. The hairs piercing the suppurative lesions are loosened and fall, after which the follicles atrophy and the hairs are not reproduced.

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[1] Grünfeld, Archiv, 1909, xcv, p. 331; abstr. Jour. Cut. Dis., 1910, xxviii, p. 541 (a report of five cases, with review of the literature).

[2] Bull. de la Soc. med. des Hôp., 1888, s. iii, p. 395.

[3] Lailler and Robert, Thèse de Paris, 1889.

The lesions are irregularly disseminated about the scalp, producing areas of various sizes and configurations. The scalp is left dead-white, thin, depressed, atrophied, and cicatriform. The follicles remain distinct and are not fused into masses. In older cases only the peripheral portions or the spreading margins of the disorder show the active suppurative processes; and in certain cases, particularly in the variety described as *Acné décalvante,* the spread of the disease may continue after active evidences of inflammation have disappeared.

Etiology.—The disorder is probably of parasitic origin. Quinquaud isolated micrococci, which occurred in various forms (monococcus, diplococcus, and in groups of four), and with these he apparently reproduced the disease in animals. Brocq considers the disease due to *Staphylococcus aureus.* Schwartz,[1] after using various methods of cultivation, isolated only *Staphylococcus aureus.* The disease usually occurs in adults. Other predisposing factors are to be cited that may be operative in other follicular infections.

Diagnosis.—The differential diagnosis is that considered under Alopecia cicatrisata.

Treatment.—As the disorder is parasitic in nature, treatment must be directed along these lines. The internal treatment consists in correcting any systemic disorder or other departures from normal in the general health. The local treatment consists in the use of sulphur, resorcin, the mercurials, the tars, salicylic acid, and iodin. Bichlorid of mercury lotions in the strength of 1 to 400, boric-acid lotions, and others suggested in the treatment of alopecia steatoides may be useful. Jackson recommends an ointment composed as follows: Acid salicylic, 15 grains (1 gramme); sulphur colloidal, 1 drachm (4.); adeps lanæ and adeps anserini, of each p.e. add 1 ounce (30.) and ol. rosæ geran., 8 drops. It is suggested that the scalp be washed once or twice weekly and the ointment applied after the hair has dried. Ointments containing sulphur in the strength of 10 per cent., or ammoniated mercury in the same strength, have been of value. To such an ointment salicylic acid or acetic acid may be added to advantage. Epilation of the hairs may be practiced.

Prognosis.—The disease is chronic, and therefore may last over long periods of time. Even after the scalp appears to be entirely cleared up recurrences happen, and frequently large areas are invaded, with consequent deformity from the permanent hair-loss. With careful and continuous treatment much can be accomplished.

DERMATITIS PAPILLARIS CAPILLITII.[2]

Synonyms.—Keloid acne, Sycosis frambœsiformis (Hebra), Sycosis nuchæ necrotisans (Ehrmann), *Acné chéloïdique* (Bazin).

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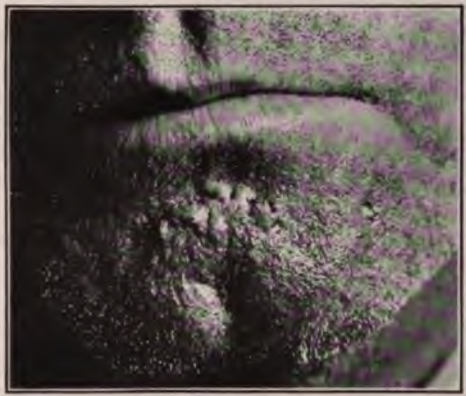
[1] Jour. Cut. Dis., 1913, xxxi, p. 419.

[2] For a survey of the literature, with a clinical and histological report, see Porges, Archiv, 1900, lii, p. 323; Adamson, Brit. Jour. Derm., 1914, xxvi, p. 69 (report of 4 cases, with modern histological study); and Tryb, Derm. Wochenschr., 1912, lv, p. 1491, abstr. Jour. Cut. Dis., 1913, xxxi, p. 184 (a careful histological study and review of the subject).

This disorder was described by Kaposi in 1869, as being characterized by papules as large as a pinhead occurring upon the back of the neck at the border of the hair; at first isolated, later closely aggregated. These coalesce into scar-like keloidal, projecting, very firm, pale or red plaques, upon which the hairs appear tangled and matted together, while other parts appear entirely bald. The hairs are pulled out with difficulty, break off in the act, and appear twisted and tortuous. The nodules creak when incised, and the cut surface bleeds from numerous points. Here and there a small pustule is seen. A few cases were subsequently reported by other observers, including Hyde;[1] in 1883, Dyer and Heitzmann[2] in America, and others in other parts of the world. The clinical description given by all agrees, all cases concluding with the production of a keloid-like, cicatriform, irregularly shaped, and circumscribed elevation of the surface. This feature distinguishes the disorder from follicular infections of the hairy region

FIG. 428



Dermatitis papillaris capillitii.

The disease is encountered chiefly upon the nucha, the occiput, and the vertex. Samberger[3] recorded an example of the disorder on the upper lip. The disease seems to be due fully as much to an inflammatory process in the subcutaneous tissue between the unyielding pericranium and the thick scalp, as in the cutis proper. Puncture of the pinhead-sized pustules commonly gives exit to the usual quantity of pus, but pressure upon the scalp in the periphery will at once be followed by the appearance of a still larger quantity of similar pus, which evidently is expressed from a circumscribed subcutaneous abscess. When by such pressure the abscess cavity is emptied, it slowly fills with venous blood and produces a firm, semisolid elevation of the surface, which subsequently undergoes sclerosis, and produces the starved hairs above described. Papillomatous vegetations, crust-covered, hemorrhagic, and with a foul-smelling secretion, sometimes form, and eventually retract into a sclerotic tissue. The disease is chronic in character, is particularly liable to relapse in crops of pustules and papules, and it extends from nucha to vertex, avoiding the frontal and temporal regions.

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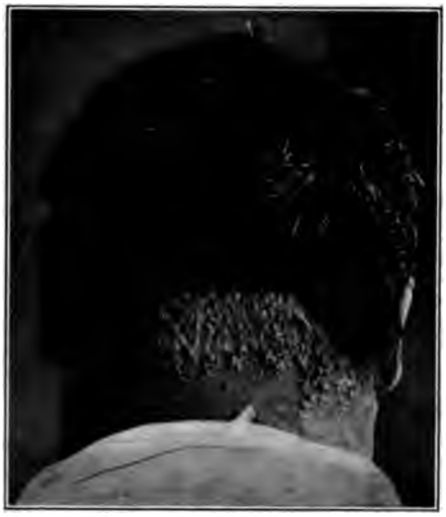
[1] Jour. Cut. Dis., 1883, i, pp. 33 and 78.

[2] Ibid., 1889, vii, p. 450.

[3] Archiv, 1907, lxxxiii, p. 163 (abstr. Brit. Jour. Derm., 1908, xx, p. 23).

FIG. 429



Dermatitis papillaris capillitii. (MacKee.)

Etiology and Pathology.—Irritation from friction by the collar is given as the probable cause. No organism has been isolated. Hebra, Ehrmann,[1] Sabouraud, and others believe the disease to be a sycosis of staphylococcic origin, with later chronic inflammatory changes. According to Besnier and Doyon, the disease is a papillomatous development, likely to occur in this region of the scalp as a sequel of epilation, cicatricial acne, eczema, or traumatism. The process is described by certain authors as beginning in and around the hair follicles, in the same manner that an ordinary follicular infection begins; while others describe the earliest process as being in the corium, about the bloodvessels, independent of hair-follicles. Cellular infiltrations are described by all, and later increased connective-tissue formation. In a study of sections taken from four cases, Adamson concludes that the sequence of events is as follows: first, dilatation of bloodvessels, with deposit of plasma-cells immediately around the vessels; transition of plasma-cells into connective-tissue cells, and young connective-tissue fibers; in other words, the reaction is mainly a fibrous-tissue-cell reaction, the plasma-cell infiltration representing, in the writer's opinion, a stage toward fibrous-tissue deposit. The lesions bear no resemblance to those of true keloid. Unna describes the transformation of the whole substance of the cutis into hypertrophic connective tissue, and believes the condition to be an infectious fibroma, which is not a keloid; nor is the initial folliculitis a sycosis or an acne. Adamson's findings support the view of Kaposi that the disease is a chronic inflammation and connective-tissue new-formation, leading by pressure to atrophy of the follicles, sebaceous and sweat-glands, and is possibly of microbic origin, but not a reaction to ordinary pus cocci.

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[1] Archiv, 1895, xxxii, p. 324.

Treatment.—All of the older methods of treatment prove ineffectual. Radiotherapy is the method of choice in this disorder. In several cases with this method we have succeeded in arresting the active process and causing a partial disappearance of the disfiguring scars. In addition to this method, surgical procedure may be necessary. The affected surfaces should be freed from subcutaneous abscesses by puncture and expression of the contents, and the usual wet dressings employed in surgery applied. When the abscesses have been cleared up, epilation may be practised and the area treated with a 3 to 10 per cent. ointment of sulphur or the same strength ointment of ammoniated mercury. Crusts may be removed by shampooings with green soap. Electrolysis, linear scarification, erasion, excision, and electrocauterization may be used in certain cases.

Prognosis.—In time, with radiotherapy, the disorder may be eradicated. With other measures recurrences are the rule.

COCCOGENOUS SYCOSIS.

Synonyms.—"Non-parasitic" sycosis; Sycosis vulgaris; Sycosis staphylogenes; Mentagra; Sycosis; Folliculitis barbae (Lat.); Bart-finne; Bartflechte (Ger.); Sycose (Fr.).

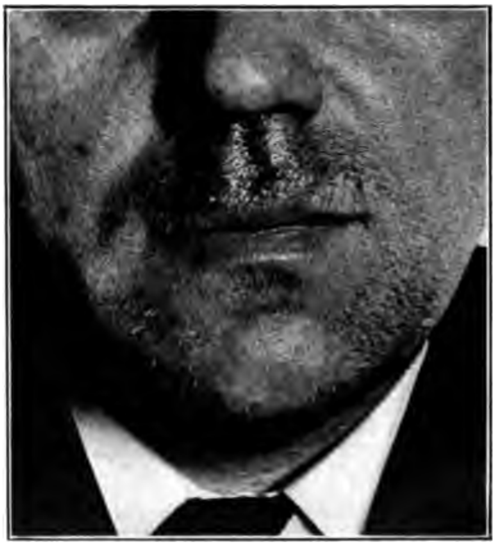
Definition.—Sycosis vulgaris is a chronic inflammatory disorder involving the hair-follicles, usually of the bearded region, and occasionally other hairy parts, characterized by inflammatory papules and pustules, commonly pierced by a hair.

Symptoms.—The lesions appear upon the face, occurring one or more at first, successively or simultaneously; the chin, the upper lip, and more rarely the eyebrows, the axillae and the pubes. The disease, however, is almost always limited to the region of the beard in men. It may be limited to one or more small or large areas for long periods of time, or it may involve the entire bearded region.

When seated upon the upper lip, which it is [illegible] [illegible] the [illegible] symptoms may be those of a [illegible] [illegible] [illegible] [illegible] attacks may precede the onset of the disease. [illegible] [illegible] with the acute symptoms exhibited in the early stage of [illegible] [illegible] [illegible] and with [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] the [illegible]

tion of each lesion by a filament of hair. These lesions may persist, and when typically discrete and visible at the point at which the hair makes its exit from the mouth of the follicle they suggest the appearance of the surface of the fig, whence the disease derives the name sycosis. They are apt to occasion a burning and at times a moderately itching sensation when, on being picked or torn open by the fingers, the pus concretes into a crust at the base of the hair. In severer cases these lesions, while not coalescing, are so closely set together as to form a patch of continuous infiltration. These patches may be weeping or be crusted; in the latter case the crusts are likely to be small and numerous, each crust being limited to the shaft of a single hair, and revealing when removed a minute, crateriform excavation at the mouth of the follicle.

FIG. 430

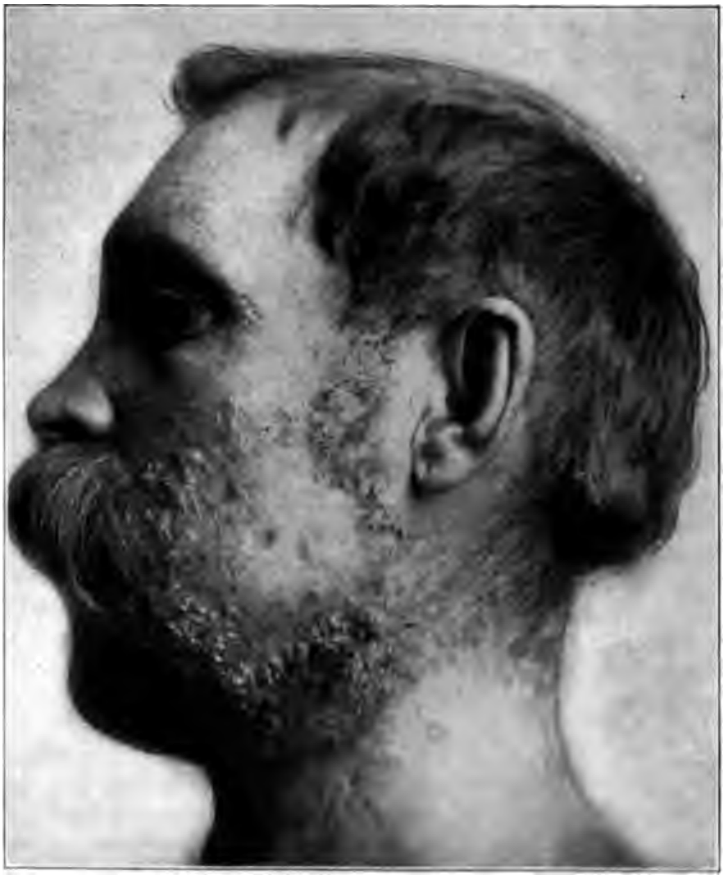


Sycosis vulgaris of the upper lip due to a chronic nasal discharge.

Involution of several lesions may be followed by fresh crops, and sooner or later distinct patches of disease are thus formed. When fully developed, the surface of the skin is reddened, swollen, infiltrated, and thickened; covered irregularly with papules, pustules, crusts, and scales, and frequently with excoriations. The disease often lapses into chronic conditions, and in these chronic cases the deformity is characteristic and totally unlike that produced by the vegetable parasites. Early in the process the hairs are usually fixed firmly in their follicles, while later, when suppuration is pronounced, they become loosened and may be readily removed. In cases of long standing the hairs are thinned and poorly nourished.

In typical cases of long standing, in which the region of the beard is involved, an important clinical feature is the symmetrical, general, and uniform involvement of the entire surface. The sparse hairs scarcely serve to disguise the reddened, tumid, painful surface beneath, which displays the severe lesions of the malady. Furuncles, abscesses, cicatrices, vegetations, and eczema of the ears may complicate the process. Sycosis is occasionally acute in its course, but is more often chronic and rebellious. A typically chronic and untreated case of the malady rarely terminates by spontaneous involution.

FIG. 431



Lupoid sycosis.

The thinning of the hairs, described above as a consequence of long persistence of the disease, is far more characteristic than any resulting alopecia; the latter, however, very rarely occurs, but when it does is remediless. The same may be said of resulting cicatrization, which is one of the rarest consequences.

Sycosis vulgaris is a disease of chronic course, which may endure for years and be characterized by relapses and aggravations, but is entirely curable; it is only in neglected cases that such persistence may be expected.

In certain rare instances the disease assumes a more destructive character. In these the usual pustules form and the disease spreads peripherally, causing destruction of the follicles, and leaving permanent scars. The active margin with an atrophic center resembles lupus, and to this form Milton gave the name *Lupoid sycosis*. Unna later described it as *Ulerythema sycosiforme*. According to Robinson, the inflammation in these cases spreads peripherally, upward or downward, with a narrow, infiltrated margin. The lesions outside of the follicles may be papular, vesicular, or pustular in type. The tendency to extension from a given center and to irregular scarring are the chief characteristic features of this phase of the malady.

Many of these cases strongly suggest in their features the symptoms of lupus erythematosus. The malady is exceedingly obstinate and lasts for years.

Etiology.—Sycosis vulgaris is unquestionably due to invasion of the pilo-sebaceous follicles by microörganisms. Obviously, in many cases there is a special reason for the accessibility of the germs to the crypts where they are lodged. Shaving, and the use in common of towels, brushes, or combs in public establishments (club-houses, barber-shops, hotels), and the employment of pillows, lounges, and reclining-chairs in public resorts, are often the origin of the disease.

The disorder is encountered chiefly among men after puberty, and in those of all social conditions and grades of health. It is not transmissible by heredity. The mere performance of shaving is not known to produce it. At times the immediate cause of the disease is recognized when the upper lip is constantly irritated by a discharge due to profuse nasal catarrh. In other cases, again, all the causes of eczema may be invoked in explanation of the result.

Pathology.—The essential process is an indolent type of folliculitis and perifolliculitis caused by microörganisms. Bockhardt was the first to identify the pyogenic organisms (*Staphylococcus aureus* and *albus*) as causative factors. According to Robinson, the process begins as a perifollicular inflammation, the follicle later becoming transformed into an abscess. The early changes as described about the follicles correspond with those ordinarily seen in the vascular connective tissue of any region: Serum and other products of the inflammatory process penetrate the hair-follicle and produce swelling and disintegration of its cellular elements. The process is most marked in the lower part and spreads upward, the pus breaking through the epidermis around the follicle. When the hair is removed the cavity is found to be lined with pus-cells. The sebaceous glands are involved after the follicle, while the sweat-coils are rarely attacked. The papillæ usually escape destruction, so that permanent alopecia seldom follows. Unna describes the process as occurring in four stages: the first superficial, involving the neck of the follicle; the second a superficial, perifollicular, inflammatory process; the third a limited perifollicular abscess occupying only one side of the follicle; and the fourth a widespread suppurative process, which involves the whole follicle, is destructive and induces scar-formation. In lupoid sycosis all the structures about the hair, including the hair-papilla, are destroyed, leaving a permanent scar. The microörganisms discovered by Tommasoli in what appeared to be a superficial sycosis were bacilli measuring 1 to 1½ microns in length by 0.25 to 0.3 microns in width. Cultures were made from these organisms and the disease reproduced.

Treatment.—The treatment of cases necessarily varies according to the condition. Where there are much tenderness, pain, swelling, pustulation, and crusting, these must be relieved by appropriate measures. Hot dressings of boric-acid solution applied several times daily are of value. At the same time a soothing ointment may be applied between the applications. In Vienna, the unguentum diachylon alba of Hebra is applied on strips of linen and firmly bandaged over the cheeks, chin, or lips and kept in place for twenty-four hours. After the marked inflammatory symptoms have been reduced, and in all other ordinary cases, the local treatment should be as follows: Shaving should be practised daily, or at least every other day. This may be painful, but should be insisted upon. In the evening hot applications are made, using a solution of the bichlorid of mercury in the strength of 1 to 2000, after which an ointment containing the ammoniated mercury in the strength of from 3 to 5 per cent. is applied. In the morning the area is bathed with the bichlorid solution and a dusting-powder, consisting of the mild chlorid of mercury 4 parts, boric acid 4 parts, and talcum powder 20 parts, is applied. In a short time great improvement is noted following this treatment. When the process becomes stationary, the same treatment should be employed, but in the place of the bichlorid of mercury the bathing should be done with boric-acid solution. The ointment following the bath should consist of sulphur in the strength of 3 to 5 per cent.; and in the morning, in the place of the mild chlorid of mercury, sulphur should be substituted in the dusting-powder. At a later date, in case it becomes necessary, other stimulating preparations, among which are the tars, resorcin, and salicylic acid, may be employed. In still other cases, ointments and lotions of ichthyol are of service.

Epilation is often essential for the relief of the disorder and is a valuable measure, a fact well demonstrated in the rapid relief of cases in which epilation follows the use of *x*-rays.

Van Harlingen advises for acute cases a wash composed of ½ pint (250.) of rosewater, to which 1 drachm (4.) each of precipitated zinc carbonate and zinc oxid in powder have been added, with 2 drachms (8.) of glycerin and dilute liquor plumbi subacetatis. Veiel recommends a solution of pyrogallol (1 part to 50) for painting over the region affected, followed in the day by emollient cataplasms and in the night by diachylon or weak tannin ointments. Sycosis of other portions of the body is to be treated as described for the region of the beard.

In view of the parasitic nature of the disease, vaccine therapy[1] has been given a thorough trial. The results in sycosis have been similar to those in other diseases, sometimes brilliant, sometimes a total failure. It is difficult, as Schamberg early remarked, to account for the fact that of two patients treated similarly one will respond perfectly and the other show no evidence whatever of the treatment. It is a valuable addition in most cases, entirely relieves some, and is therefore worthy of trial.

Radiotherapy[2] offers another valuable method of treatment. When used in moderation (merely enough to cause epilation of the hairs), the immediate results are good, and with this amount of treatment no remote ill effects follow. With the regrowth of the hairs, which occurs in about two months, recurrence may happen, but this, as a rule, occurs in more limited areas and can safely be treated a second time. This agent should always be employed with caution, and when so employed nothing but happy results occur.

Internal treatment should be employed according to the indications of the individual patient. Owing to the chronic character of the disease, numbers of these patients when first seen have been taking various sorts of internal treatment; a suspension of this is followed by improvement. Every sort of external irritation should be avoided during treatment. The diet should conform to the needs of the particular patient. A tonic treatment is suggested in the case of patients whose general condition is poor and whose nutrition is below par. It is also essential to promote the eliminative processes by the free use of water and by the use of proper medication when indicated.

Prognosis.—The disease is entirely curable, and will in a large majority of cases either entirely disappear or be greatly improved by judicious treatment. The latter requires the personal supervision of the physician and calls for attention to details. In exceptional cases the disorder is exceedingly chronic and obstinate and requires a great amount of time for its eradication, and it must be remembered that relapses are of frequent occurrence. In the few rare cases of lupoid sycosis, there is cicatricial tissue left after the disappearance of the disorder.

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[1] Colcott Fox, Brit. Jour. Derm., 1907, xix, p. 420; Gildersleeve, Jour. Cut. Dis., 1907, xxv, p. 320; Schamberg, Trans. VI Internat. Derm. Cong., p. 290; MacKee, Jour. Cut. Dis., 1913, xxxi, p. 1042.

[2] Howard Fox, Jour. Cut. Dis., 1908, xxvi, pp. 185 and 464; ibid., 1909, xxvii, p. 357; and Pfaler, ibid., 1913, xxxi, p. 116.

DISEASES OF THE NAILS.[1]

ONYCHAUXIS.

Synonyms.—Hypertrophy of nail, Onychogryphosis.

The term onychauxis signifies hypertrophy of the nail. This condition may involve one or more nails of both hands and feet, either as an idiopathic or symptomatic affection. The nails may be augmented in length, breadth, or thickness, and changed in shape, density, color, or texture. The term onychogryphosis is restricted by most English authors to the condition in which twisted, contorted, and other anomalies of the nails are present. In the latter condition the nail, particularly of the great toe, becomes claw-like or twisted like a horn. Occasionally, other nails of the toes, and rarely of the fingers, become involved. There is marked thickening, due to hyperkeratosis of the nail-bed beneath, the nail-plate not being particularly involved. In the cases not characterized by this peculiarity the nail-plate may be increased in all dimensions, uniformly or partially. The nails are dull-colored, opaque, yellowish-brown or dirty-black in hue, and are often ridged, rugous, or furrowed. The subungual space is choked with poorly-formed corneous material, which may be at times secondarily infected. Occasionally the subungual horny deposit raises the nail from its bed at the distal end. A remarkable series of this latter variety, occurring as an hereditary disorder, was recorded by Wilson.[2] In these cases the nail of the hand was normal in appearance, but at the free extremity became raised over its bed by a dark, friable, horny mass, which projected from the free edge. [illegible] described the case of a child of [illegible] years, whose nails, both of the fingers and toes, had [illegible] projected upward from one-half to three-fourths of an inch. [illegible] reported the case of an infant three months of age whose nails were similar to those described above, and when the nail was cut a [illegible] like substance escaped. Nicolle and Halipré,[4] [illegible] and [illegible] have all described groups of cases having an associated [illegible] process in the nails and atrophic processes of the [illegible] several generations. In the first instance there were 36 individuals in six generations; in the second, the disease occurred in four generations; and in the third there were several cases in five generations. In these there occurred both hypertrophic and atrophic changes, with subungual inflammation.

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[1] Heller: Die Krankheiten der Nägel, [illegible]

[2] Brit. Jour. Derm., 1895, vii, p. 2. [illegible]

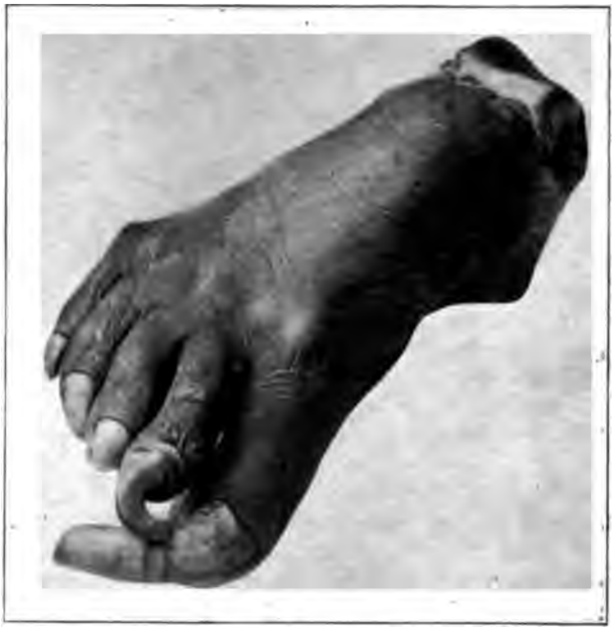
[3] Jour. Cut. Dis., 1903, [illegible] p. 11.

[4] Annales, 1898, [illegible] [illegible]

[5] Jour. Amer. Med. Assoc., [illegible]

The causes of this disorder are various. Local irritation from pressure or ill-fitting shoes, stockings or gloves, filth, neglect, and the exposures producing inflammation of the skin of the hands may all be responsible for the condition. Hypopituitarism was given as a cause in a case recorded by Hollander.[1]

FIG. 432



Onychogryphosis in a leper, a Hindoo afflicted with the neural form of leprosy. (Douglass W. Montgomery.)

Onychia (*Onychitis*) is the result of inflammation of the matrix or folds of the nail; and may be produced by any of the causes capable of exciting inflammation in other regions of the integument. Trauma, the pyogenic microörganisms, foreign bodies beneath or within the nail-plate, or parasites may excite inflammation of the soft parts about the nail sufficient to produce distortion, fall of the plate, ulceration, and even digital gangrene.

Onychia Maligna, whether occurring in children or adults, since the date of modern methods in diagnosis and therapy has become practically unknown. It may be due to struma, syphilis, tuberculosis, disease is a chronic inflammation and connective-tissue new-formation, leading by pressure to atrophy of the follicles, sebaceous and sweat-glands, and is possibly of microbic origin, but not a reaction to ordinary pus cocci.

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[1] Archiv. of Derm. and Syph., 1920, ii, p. 35; Onychauxis due to hypopituitarism.

Treatment.—All of the older methods of treatment prove ineffectual. Radiotherapy is the method of choice in this disorder. In several cases with this method we have succeeded in arresting the active process and causing a partial disappearance of the disfiguring scars. In addition to this method, surgical procedure may be necessary. The affected surfaces should be freed from subcutaneous abscesses by puncture and expression of the contents, and the usual wet dressings employed in surgery applied. When the abscesses have been cleared up, epilation may be practised and the area treated with a 3 to 10 per cent. ointment of sulphur or the same strength ointment of ammoniated mercury. Crusts may be removed by shampooings with green soap. Electrolysis, linear scarification, erasion, excision, and electrocauterization may be used in certain cases.

Prognosis.—In time, with radiotherapy, the disorder may be eradicated. With other measures recurrences are the rule.

COCCOGENOUS SYCOSIS.

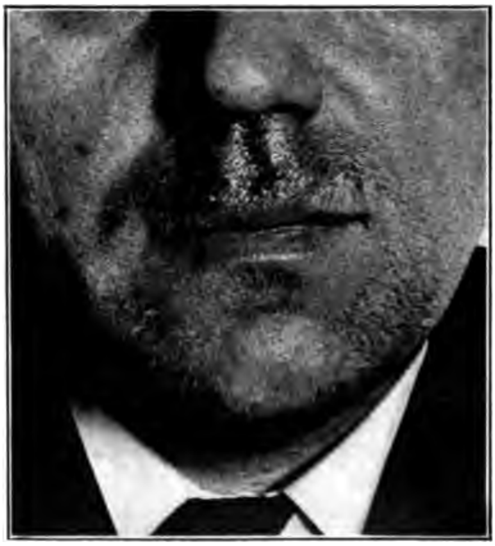
Synonyms. — "Non-parasitic" sycosis, Sycosis vulgaris, Sycosis staphylogenes, Mentagra, Ficosis, Folliculitis barbæ. Ger., *Bartfinne, Bartflechte;* Fr., *Sycose.*

Definition.—Sycosis vulgaris is a chronic inflammatory disorder involving the hair-follicles, usually of the bearded region and moustache, characterized by inflammatory papules and pustules, commonly pierced by a hair.

Symptoms.—The lesions appear upon the face, involving one or both cheeks, successively or simultaneously, the chin, the upper lip, and more rarely the eyebrows, the scalp, the axillæ, and the pubes. The disease, however, is almost always limited to the region of the beard in men. It may be limited to one or more small or large areas for long periods of time, or it may involve the entire bearded region.

When seated upon the upper lip, which is a favorite site, the first symptoms may be those of a nasal catarrh; seated elsewhere, an eczematous attack may precede the onset of the disease. It may be ushered in with the acute symptoms exhibited in the early stage of some forms of eczema, and with tumefaction, accompanied by a sensation of heat and burning; but often a few isolated and indolent lesions, the presence of which scarcely awakens attention, are the first symptoms of the disorder. Soon there may be recognized a larger or smaller number of discrete, pinpoint- to split-pea-sized, flattened or conical, reddish and painful papules, tubercles, or pustules, the anatomical seat of which is distinguished as the hair follicle by the penetration of each lesion by a filament of hair. These lesions may persist, and when typically discrete and visible at the point at which the hair makes its exit from the mouth of the follicle they suggest the appearance of the surface of the fig, whence the disease derives the name sycosis. They are apt to occasion a burning and at times a moderately itching sensation when, on being picked or torn open by the fingers, the pus concretes into a crust at the base of the hair. In severer cases these lesions, while not coalescing, are so closely set together as to form a patch of continuous infiltration. These patches may be weeping or be crusted; in the latter case the crusts are likely to be small and numerous, each crust being limited to the shaft of a single hair, and revealing when removed a minute, crateriform excavation at the mouth of the follicle.

Fig. 430

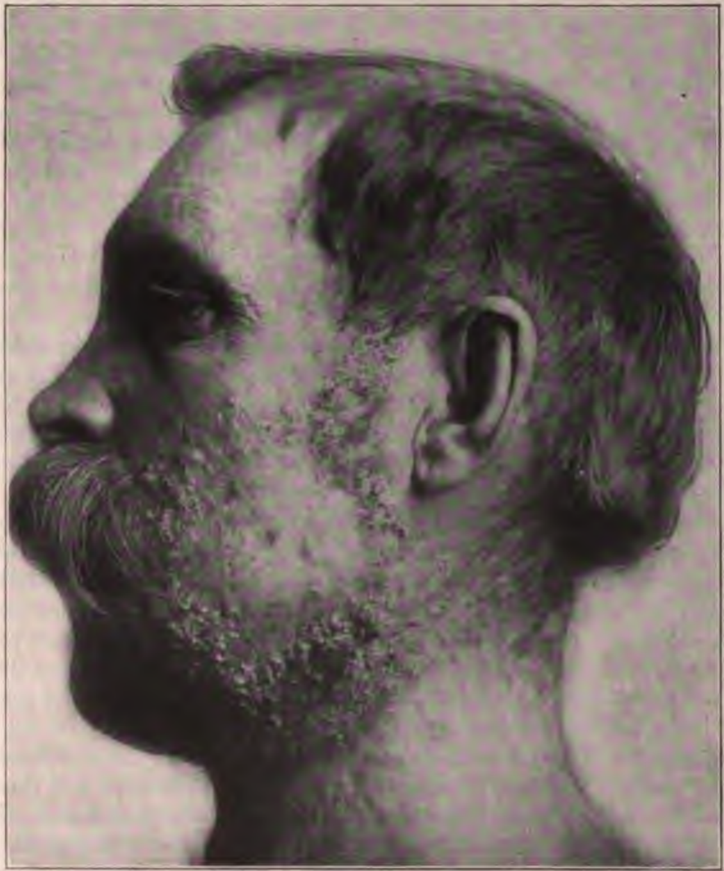


Sycosis vulgaris of the upper lip due to a chronic nasal discharge.

Involution of several lesions may be followed by fresh crops, and sooner or later distinct patches of disease are thus formed. When fully developed, the surface of the skin is reddened, swollen, infiltrated, and thickened; covered irregularly with papules, pustules, crusts, and scales, and frequently with excoriations. The disease often lapses into chronic conditions, and in these chronic cases the deformity is characteristic and totally unlike that produced by the vegetable parasites. Early in the process the hairs are usually fixed firmly in their follicles, while later, when suppuration is pronounced, they become loosened and may be readily removed. In cases of long standing the hairs are thinned and poorly nourished.

In typical cases of long standing, in which the region of the beard is involved, an important clinical feature is the symmetrical, general, and uniform involvement of the entire surface. The sparse hairs scarcely serve to disguise the reddened, tumid, painful surface beneath, which displays the severe lesions of the malady. Furuncles, abscesses, cicatrices, vegetations, and eczema of the ears may complicate the process. Sycosis is occasionally acute in its course, but is more often chronic and rebellious. A typically chronic and untreated case of the malady rarely terminates by spontaneous involution.

FIG. 431



Lupoid sycosis.

The thinning of the hairs, described above as a consequence of long persistence of the disease, is far more characteristic than any resulting alopecia; the latter, however, very rarely occurs, but when it does is remediless. The same may be said of resulting cicatrization, which is one of the rarest consequences.

Sycosis vulgaris is a disease of chronic course, which may endure for years and be characterized by relapses and aggravations, but is entirely curable; it is only in neglected cases that such persistence may be expected.

In certain rare instances the disease assumes a more destructive character. In these the usual pustules form and the disease spreads peripherally, causing destruction of the follicles, and leaving permanent scars. The active margin with an atrophic center resembles lupus, and to this form Milton gave the name *Lupoid sycosis*. Unna later described it as *Ulerythema sycosiforme*. According to Robinson, the inflammation in these cases spreads peripherally, upward or downward, with a narrow, infiltrated margin. The lesions outside of the follicles may be papular, vesicular, or pustular in type. The tendency to extension from a given center and to irregular scarring are the chief characteristic features of this phase of the malady.

Many of these cases strongly suggest in their features the symptoms of lupus erythematosus. The malady is exceedingly obstinate and lasts for years.

Etiology.—Sycosis vulgaris is unquestionably due to invasion of the pilo-sebaceous follicles by microörganisms. Obviously, in many cases there is a special reason for the accessibility of the germs to the crypts where they are lodged. Shaving, and the use in common of towels, brushes, or combs in public establishments (club-houses, barber-shops, hotels), and the employment of pillows, lounges, and reclining-chairs in public resorts, are often the origin of the disease.

The disorder is encountered chiefly among men after puberty, and in those of all social conditions and grades of health. It is not transmissible by heredity. The mere performance of shaving is not known to produce it. At times the immediate cause of the disease is recognized when the upper lip is constantly irritated by a discharge due to profuse nasal catarrh. In other cases, again, all the causes of eczema may be invoked in explanation of the result.

Pathology.—The essential process is an indolent type of folliculitis and perifolliculitis caused by microörganisms. Bockhardt was the first to identify the pyogenic organisms (*Staphylococcus aureus* and *albus*) as causative factors. According to Robinson, the process begins as a perifollicular inflammation, the follicle later becoming transformed into an abscess. The early changes as described about the follicles correspond with those ordinarily seen in the vascular connective tissue of any region: Serum and other products of the inflammatory process penetrate the hair-follicle and produce swelling and disintegration of its cellular elements. The process is most marked in the lower part and spreads upward, the pus breaking through the epidermis around the follicle. When the hair is removed the cavity is found to be lined with pus-cells. The sebaceous glands are involved after the follicle, while the sweat-coils are rarely attacked. The papillæ usually escape destruction, so that permanent alopecia seldom follows. Unna describes the process as occurring in four stages: the first superficial, involving the neck of the follicle; the second a superficial, perifollicular, inflammatory process; the third a limited perifollicular abscess occupying only one side of the follicle; and the fourth a widespread suppurative process, which involves the whole follicle, is destructive and induces scar-formation. In lupoid sycosis all the structures about the hair, including the hair-papilla, are destroyed, leaving a permanent scar. The microörganisms discovered by Tommasoli in what appeared to be a superficial sycosis were bacilli measuring 1 to 1½ microns in length by 0.25 to 0.3 microns in width. Cultures were made from these organisms and the disease reproduced.

Treatment.—The treatment of cases necessarily varies according to the condition. Where there are much tenderness, pain, swelling, pustulation, and crusting, these must be relieved by appropriate measures. Hot dressings of boric-acid solution applied several times daily are of value. At the same time a soothing ointment may be applied between the applications. In Vienna, the unguentum diachylon alba of Hebra is applied on strips of linen and firmly bandaged over the cheeks, chin, or lips and kept in place for twenty-four hours. After the marked inflammatory symptoms have been reduced, and in all other ordinary cases, the local treatment should be as follows: Shaving should be practised daily, or at least every other day. This may be painful, but should be insisted upon. In the evening hot applications are made, using a solution of the bichlorid of mercury in the strength of 1 to 2000, after which an ointment containing the ammoniated mercury in the strength of from 3 to 5 per cent. is applied. In the morning the area is bathed with the bichlorid solution and a dusting-powder, consisting of the mild chlorid of mercury 4 parts, boric acid 4 parts, and talcum powder 20 parts, is applied. In a short time great improvement is noted following this treatment. When the process becomes stationary, the same treatment should be employed, but in the place of the bichlorid of mercury the bathing should be done with boric-acid solution. The ointment following the bath should consist of sulphur in the strength of 3 to 5 per cent.; and in the morning, in the place of the mild chlorid of mercury, sulphur should be substituted in the dusting-powder. At a later date, in case it becomes necessary, other stimulating preparations, among which are the tars, resorcin, and salicylic acid, may be employed. In still other cases, ointments and lotions of ichthyol are of service.

Epilation is often essential for the relief of the disorder and is a valuable measure, a fact well demonstrated in the rapid relief of cases in which epilation follows the use of *x*-rays.

Van Harlingen advises for acute cases a wash composed of ½ pint (250.) of rosewater, to which 1 drachm (4.) each of precipitated zinc carbonate and zinc oxid in powder have been added, with 2 drachms (8.) of glycerin and dilute liquor plumbi subacetatis. Veiel recommends a solution of pyrogallol (1 part to 50) for painting over the region affected, followed in the day by emollient cataplasms and in the night by diachylon or weak tannin ointments. Sycosis of other portions of the body is to be treated as described for the region of the beard.

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Radiotherapy[2] offers another valuable method of treatment. When used in moderation (merely enough to cause epilation of the hairs), the immediate results are good, and with this amount of treatment no remote ill effects follow. With the regrowth of the hairs, which occurs in about two months, recurrence may happen, but this, as a rule, occurs in more limited areas and can safely be treated a second time. This agent should always be employed with caution, and when so employed nothing but happy results occur.

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[1] Colcott Fox, Brit. Jour. Derm., 1907, xix, p. 420; Gildersleeve, Jour. Cut. Dis., 1907, xxv, p. 320; Schamberg, Trans. VI Internat. Derm. Cong., p. 290; MacKee, Jour. Cut. Dis., 1913, xxxi, p. 1042.

[2] Howard Fox, Jour. Cut. Dis., 1908, xxvi, pp. 185 and 464; ibid., 1909, xxvii, p. 357; and Pfaler, ibid., 1913, xxxi, p. 116.

DISEASES OF THE NAILS.[1]

ONYCHAUXIS.

Synonyms.—Hypertrophy of nail, Onychogryphosis.

The term onychauxis signifies hypertrophy of the nail. This condition may involve one or more nails of both hands and feet, either as an idiopathic or symptomatic affection. The nails may be augmented in length, breadth, or thickness, and changed in shape, density, color, or texture. The term onychogryphosis is restricted by most English authors to the condition in which twisted, contorted, and other anomalies of the nails are present. In the latter condition the nail, particularly of the great toe, becomes claw-like or twisted like a horn. Occasionally, other nails of the toes, and rarely of the fingers, become involved. There is marked thickening, due to hyperkeratosis of the nail-bed beneath, the nail-plate not being particularly involved. In the cases not characterized by this peculiarity the nail-plate may be increased in all dimensions, uniformly or partially. The nails are dull-colored, opaque, yellowish-brown or dirty-black in hue, and are often ridged, rugous, or furrowed. The subungual space is choked with poorly-formed corneous material, which may be at times secondarily infected. Occasionally the subungual horny deposit raises the nail from its bed at the distal end. A remarkable series of this latter variety, occurring as an hereditary disorder, was recorded by Wilson.[2] In these cases the nail at the base was normal in appearance, but at the free extremity became raised over its bed by a dark, friable, horny mass, which projected from the free edge. Sympson[3] described the case of a child of eleven years whose nails, both of the fingers and toes, had since birth projected upward from one-half to three-fourths of an inch. Zeisler[4] recorded the case of an infant three months of age whose nails were similar to those described above, and when the nail was cut a thick, oily, serum-like substance escaped. Nicolle and Halipré,[5] White,[6] and Eisenstedt[7] have all described groups of cases having an associated dystrophic process in the nails and atrophic processes of the hair, occurring in several generations. In the first instance there were 36 individuals in six generations; in the second, the disease occurred in four generations; and in the third there were several cases in five generations. In these there occurred both hypertrophic and atrophic changes, with subungual inflammation.

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[1] Heller, Mraček's Handbuch der Hautkrankheiten, 1909, vol. iv, Part 2 (with references and thorough exposition of the subject). White, C. J., Boston Med. and Surg. Jour., cxlvii, 1902, No. 20, p. 537 (a clinical study of 485 cases of nail disease). Shoemaker, Jour. Cut. Dis., 1890, viii, pp. 334, 388, 419, 476 (with references and abstracts). Hutchinson's Arch. of Surgery, 1890–1891, vol. xi, p. 237. Montgomery, D. W., Twentieth Century Practice, vol. v. Trans. Amer. Derm. Assoc., 1901, p. 111 (symposium on Dis. of Nails: Grindon, Pollitzer, Zeisler, Hardaway). Pernet, Encycloped. Med., vol. viii, 1901 (with bibliography). Atlas of Ill. of Med., Surg., and Path., London, New Sydenham Society, 1906. Dubreuilh, La Pratique Derm., Paris, 1902, p. 607, art. Ongles (with illustrations). Jackson, Jour. Cut. Dis., 1905, xxiii, p. 153 (12 clinical cases). Hartzell, University of Pennsylvania Med. Bull., Oct., 1904.

[2] Brit. Jour. Derm., 1905, xvii, p. 13.

[3] Lancet, April 14, 1888, p. 772.

[4] Jour. Cut. Dis., 1901, xix, p. 511.

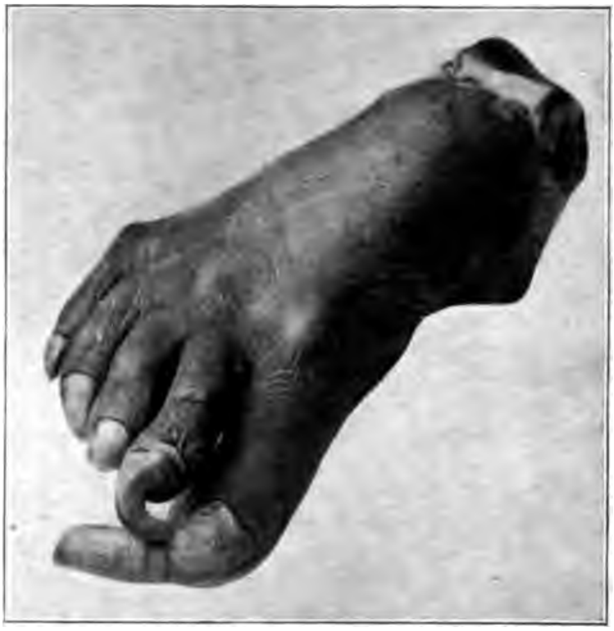
[5] Annales, 1895, s. iii, vi, p. 804.

[6] Jour. Cut. Dis., 1896, xiv, p. 220.

[7] Jour. Amer. Med. Assoc., 1913, lx, p. 27.

The causes of this disorder are various. Local irritation from pressure or ill-fitting shoes, stockings or gloves, filth, neglect, and the exposures producing inflammation of the skin of the hands may all be responsible for the condition. Hypopituitarism was given as a cause in a case recorded by Hollander.[1]

Fig. 432



Onychogryphosis in a leper, a Hindoo afflicted with the neural form of leprosy. (Douglass W. Montgomery.)

Onychia (*Onychitis*) is the result of inflammation of the matrix or folds of the nail; and may be produced by any of the causes capable of exciting inflammation in other regions of the integument. Trauma, the pyogenic microörganisms, foreign bodies beneath or within the nail-plate, or parasites may excite inflammation of the soft parts about the nail sufficient to produce distortion, fall of the plate, ulceration, and even digital gangrene.

Onychia Maligna, whether occurring in children or adults, since the date of modern methods in diagnosis and therapy has become practically unknown. It may be due to struma, syphilis, tuberculosis, septicemia, or to any infectious process. Commonly, an ulcer forms at the border of the nail, which gradually develops a necrotic floor and edge. A severe phlegmonous process complicates some cases.

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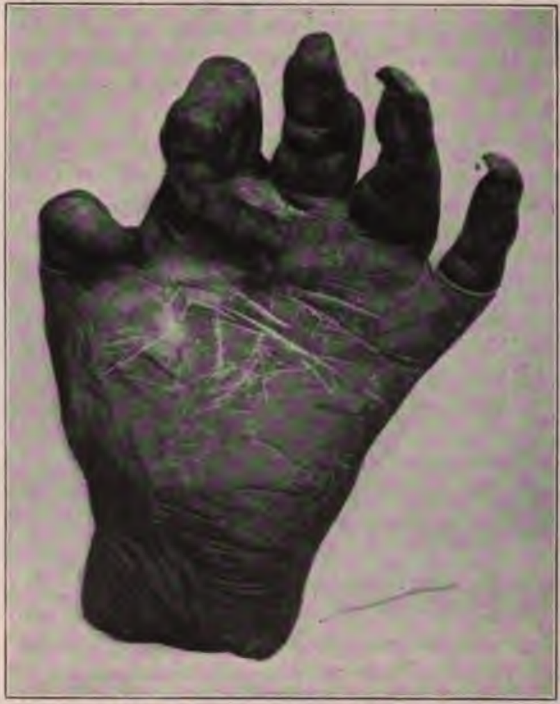
[1] Archiv. of Derm. and Syph., 1920, ii, p. 35; Onychauxis due to hypopituitarism.

Paronychia (*Panaritium, Whitlow*).—This condition may involve one or several nails, and the inflammatory process be of moderate or severe grade. The disorder is said by Sabouraud to be due to a streptococcic infection, but other organisms are commonly found. Morrow and Lee[1] in 16 cases found *Staphylococcus albus*. In the milder varieties, one, two or more nails are involved in a moderate inflammatory process, in which there occur redness and some swelling in the nail-fold, either in part or entirely surrounding the nail. As a rule, a moderate amount of tenderness and some pain are present. In a more severe grade the symptoms are intensified and a purulent inflammation, with accompanying discharge, is present. In these cases markedly painful sensations occur and the fingers are exquisitely tender to the touch. In a still more severe grade, which is practically a surgical affection, the process spreads to the surrounding tissues,

Fig. 433



Onychogryphosis. (Douglass W. Montgomery.)

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[1] Jour. Cut. Dis., 1915, xxxiii.

producing marked swelling of the entire digit, inducing eventually suppuration, necrosis, and at times exfoliation of the bone of the involved phalanx. A series of whitlows is a characteristic symptom of Morvan's disease.

Unguis Incarnatus (*Ingrowing nail*) occurs when an edge of the nail-plate impinges abnormally upon the soft parts in the vicinity and excites irritation. The ingrowth may occur to such an extent as to bury the edge of the plate deeply in a sulcus or ulcerated furrow on one side or the other of the soft parts, where it operates precisely like a foreign body. Often an exquisitely tender, granulating wound results, requiring surgical relief. The condition is one most often occurring in the feet, and particularly in the great toe, because of pressure effects from the coverings of the feet.

Pterygium.—The fold of the epidermal structure which in health furnishes the proximal border of the nail-plate may advance to a greater or less extent over the plate. In adult life, this advance may be due to radiotherapeutic treatment of the fingers when the nails are exposed to the rays. In some cases the condition is the result of neglect of hygiene of the nails, and occasionally produces a considerable disfigurement. Heller describes it as at times congenital, the nail of the big toe being set as if in a cap. The treatment is by hygiene of the nails and the use of the cuticle-knife.

Hangnails, Agnails (Ger., *Nietnagel;* Fr., *Envies*) originate from tags of the lateral nail-folds, detached mechanically and torn upward. At times the rift penetrates deeply into the sulcus by the side of the nail, leaving thus an ample atrium for infection with microörganisms. This may be the first step toward the production of a grave onychia terminating in exfoliation of the distal phalanx. In yet other cases chancres form in the part and syphilis follows. Biting and picking of the nails is a frequent cause of these apparently trivial affections. The treatment is by aseptic dressing, protection by sealing up the small wound, and, in severe cases, excision.

Subungual Hemorrhage is believed by Unna to be responsible for many otherwise unexplained cases of shedding of the nail. In some instances the hemorrhages are microscopic and appear only on section of nail-tissue. In other cases minute reddish or reddish-black specks become visible beneath the plate. In extreme cases the entire matrix becomes blackish from effused blood. The nail is shed when the process is sufficiently extensive to produce separation of the plate from the bed. Subungual hemorrhages occur after trauma; in scurvy; in hemophilia; and in certain disorders of the nervous centers (epilepsy). MacLeod[1] recorded the occurrence of subungual hemorrhage in a patient having a weak peripheral circulation. Pringle reported a similar case occurring in a Raynaud's subject. Removal of the plate may be required in surgical cases.

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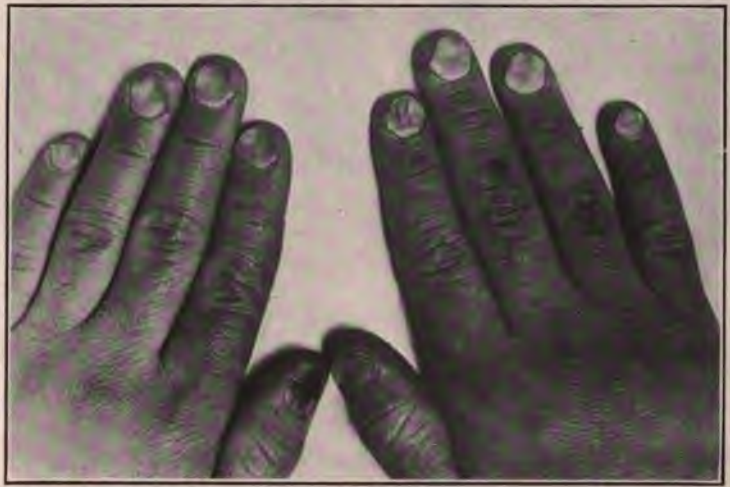
[1] Brit. Jour. Derm., 1911, xxiii, p. 364 (Hemorrhage into the nail-matrix and nail-bed of the finger-nails).

Subungual tumor-formation occurs rarely. Dr. Shepard[1] reports a subungual chondroma. Hutchinson, Jr.,[2] recognized an epithelioma in this situation; Kraske (cited by Shoemaker) a sarcoma. We have seen a number of corns growing beneath the plates. Heller gives details of other subungual tumors recognized by different authors, including papilloma, fibroma, leiomyoma, endothelioma, angioma, and angiosarcoma. Telangiectases have also been noted.

ATROPHIA UNGUIUM.

Onychatrophia (Fr., *Onychatrophie*).—Atrophy of the nails is always a symptomatic condition, due either to a local or systemic influence, whereby the formation of the horny material of the nail is rendered either wholly abortive or defective. In these cases the nail-plates may be changed in bulk, color, elasticity, firmness, shape, or position. They may be thinned and expanded, narrow and acuminate, friable, furrowed, laminated, ridged, or in other ways distorted. They may be striped, irregularly speckled, lusterless, or have a characteristic dull-yellowish color with "worm-eaten" aspect. In other conditions the nails are split or even crumbling, so that the relics only of the nail-substance are visible near the matrix, one-half or more of the distal flange having disappeared. In yet other cases sparse, horny spurs, "flakes," or pegs of a dull-greenish or dirty hue project from the proximal portions of the nail-bed.

FIG. 434



"Spoon" nails.

Hapalonychia.—Under this title Kaposi has described a condition of atrophy in which, through defective nail-production, the plates become softened and correspondingly weakened, being thus the more readily split and folded.

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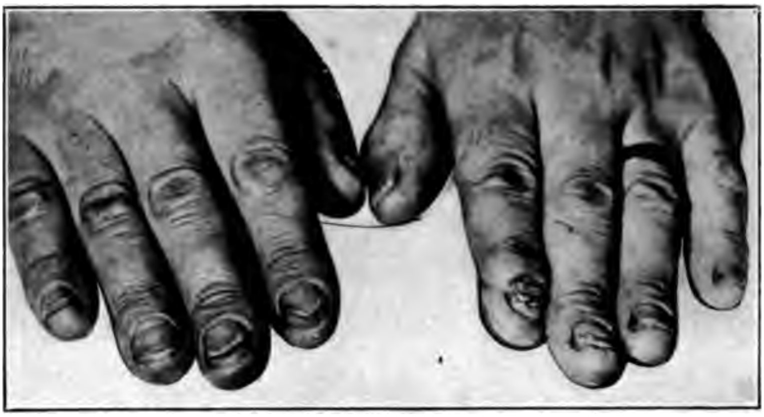
[1] Trans. Amer. Med. Assoc., 1901, p. 138.

[2] Trans. Path. Soc., London, xxxvi, p. 468.

Koilonychia (*Spoon nails;* Ger., *Aushöhlung der Nägel*).—In this condition, which has been described by Crocker,[1] Rille,[2] and others, the nail-plates are thinned and present a concavity more marked transversely than from before backward. It begins on one finger, others gradually becoming involved. In Crocker's case it occurred in association with lichen planus. It has accompanied acanthosis nigricans and certain wasting diseases, and has been associated with leukonychia. It has been described as occurring in several members of a family and for several generations. Among other causes mentioned is the immersion of the hands in strong alkalies, but the etiology in many cases is obscure.

FIG. 435



Onychoschizia; duration one year; female, aged thirty-seven years; toe nails similarly involved.

Onychoschizia (*Onycholysis;* Fr., *Décollement des ongles*).—In this condition the nail is loosened in varying degrees from its bed. It indicates a separation of the nail without shedding, and occurs as a symptomatic process in psoriasis, eczema, and syphilis, in which condition collections of cells beneath the nail mechanically raise it. It may also be induced in a similar manner by corns. Idiopathic neurotic cases are described by Dubreuilh and others.[3] In these the process begins at the free end or at one corner and gradually progresses until it has involved the entire nail as far as the lunule. Heller[4] describes a case of onycholysis partialis, associated with hyperidrosis, in which the nail-plates were loosened, the process beginning at the free border and extending until only a small portion of the nail remained attached.

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[1] Diseases of the Skin, p. 1257.

[2] Case cited by Heller, with illustration, Die Krankh. der Nägel, etc., p. 136.

[3] Weber, Brit. Jour. Derm., 1911, xxiii, p. 235 (Trophoneurotic separation of the nails, followed by alopecia areata); ibid., 1912, xxiv, p. 204.

[4] Derm. Zeitschr., July, 1912, xix, p. 609; abstr. Jour. Cut. Dis., 1913, xxxi, p. 279.

In the condition described by Hyde[1] under the title, "The egg-shell nail," the nail-plates are thinned and show a tendency to an upward thrust of the free border after leaving the nail-bed. The free portion is abnormally whitish in hue, suggesting the pinkish-white color of the inside of a hen's egg. The nails appear more translucent than normal, and their transverse curve may be exaggerated. In the cases he described there was an associated hyperidrosis.

Onychomadesis (*Onychoptosis; Alopecia ungualis; Defluvium unguium*).—Total and intermittent shedding of the nails occurs in many conditions, and is not infrequently connected with systemic affections of a severe grade. All of the nails may be shed or only certain ones of the fingers and toes. After a severe attack of scarlet fever the nails may all be shed with the skin of the palms and soles. They may also be shed in connection with generalized alopecia areata, syphilis and diabetes. Local inflammation involving the nail-bed and matrix, such as that occurring in onychia and paronychia, not infrequently produces the condition. Fox[2] describes exfoliation, with no change in the texture of the nails, in onychia without suppuration. Falcone[3] records recurrent shedding of the nails associated with inflammatory changes in the matrix, occurring in a neurotic patient. D. W. Montgomery[4] recorded a case of hereditary and continuous shedding of the finger-nails in a male patient, aged thirty-five years, who had suffered with the disorder since birth. The nails of the mother and two maternal uncles had been similarly affected. One or two of the nails were constantly falling after a yellowish-white change in the lunula.

Leukonychia (*Leukopathia unguium, Achromia unguium, Albugo, "White spots," Flores unguium, "Gift spots," Canities unguium*. Fr., *Décolorization des ongles*).—In this condition either small areas or the entire nail-plate may be involved. Usually white points, spots, streaks, or bands occur, beginning near the lunula, and with the growth of the nail gradually progress toward the free border. The condition is found chiefly in young subjects and upon the nails of the fingers. At times but a single or a few nails may exhibit one or several points of discoloration. In other instances every nail is the seat of numerous spots or bars, some occupying the larger portion of the nail-plate.

The causes of the condition are obscure. In some cases, as in those described by Longstreth,[5] Shoemaker, and Giovannini,[6] the condition is apparently related to nervous or systemic disorders. The pathogenesis of the disease is not understood. It is believed by some to be a trophoneurosis. Traumatism of the proximal portion of the plate, as in operations of the manicure, are given by Heidingsfeld[7] and others as the cause in a number of cases. It is usually stated that the white spots are due to air occurring between the epithelial cells. Heidingsfeld[1] found in the whitened areas imperfect keratinization, to which he attributed the clinical changes. Sibley[2] recorded the presence of nuclei in the cells in the situation, showing imperfect keratinization of the cell.

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[1] Jour. Cut. Dis., 1906, xxiv, p. 145.

[2] Brit. Jour. Derm., 1895, vii, p. 389.

[3] Gaz. d'Osped. Milano, 1887, viii, p. 156; ibid., Giorn., 1887, p. 206.

[4] Jour. Cut. Dis., 1897, xv, p. 374.

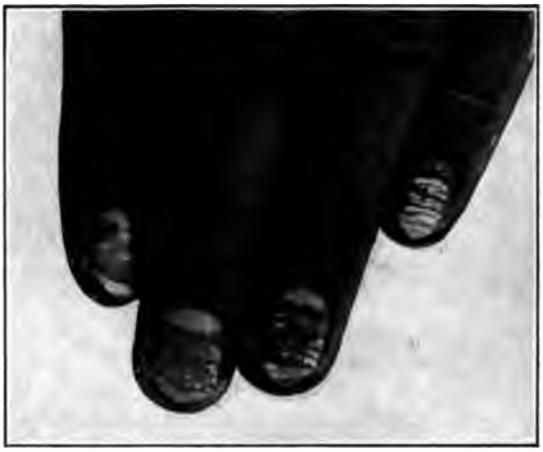
[5] Trans. College of Physicians, Philadelphia, s. iii, vol. viii, p. 113.

[6] Reform Med., Naples, 1891, vii, pl. 2, p. 865.

[7] Jour. Cut. Dis., 1900, xviii, p. 490.

Onychorrexis (*Reedy nails*).—Splitting of one or all of the nail-plates, usually in longitudinal lines, affects in inconspicuous degree many persons. When exaggerated, the nail-plate is thinned and presents longitudinal fracture, the free ends being broken, split, and thinned. Fluted, ridged, or reeded nails are described as being a manifestation of gout by Laycock, Duckworth[3] and others. They also occur in elderly people, and are then only one of many senile atrophic changes (Crocker).

Fig. 436



Leukonychia of the hand.

Transverse Furrows (*Beau's lines*).—Transversely directed lines or furrows in the nail-plates are the frequent, or perhaps invariable, consequence of impairment of nail nutrition, either systemic or local They are first seen over the lunule and gradually progress forward with the growth of the nail. They may be well marked or only indistinctly visible. They occur in various diseases; from nervous shocks, following accident or otherwise, attacks of sea-sickness[4] and other conditions. It is frequently possible to determine approximately the date of the illness, accident, or other condition that has caused the line by noting its relative position on the nail, recognizing the fact that it takes approximately six months for the nail to grow in its entirety. A number of lines may indicate a recurrence of the [illegible] exciting causes. Heisler in a study of [illegible] of the thigh, noted an arrest in growth of the nails [illegible] for some weeks. The ridge formed at the time gradually [illegible] forward, and divided the plate into a distal thin and atrophic portion and a proximal strong and normal area.

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[1] Loc. cit.

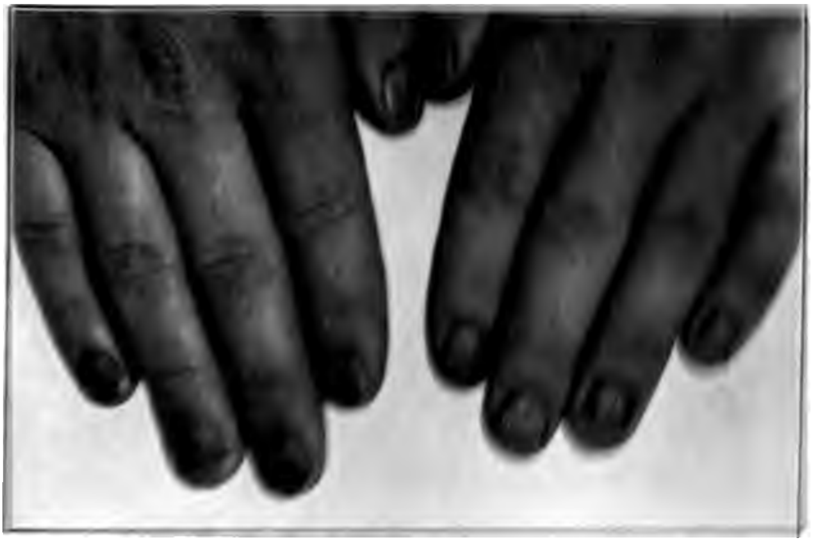
[2] Brit. Jour. Derm., 1911, xxiii, p. 281 (report of a case of leukonychia, with analysis of many recorded cases; also describing a case with yellowish discoloration, *Ungues flavi*).

[3] Quoted by Crocker, p. 1261.

[4] Hartzell, Trans. Amer. Derm. Assoc., 1901, p. 140.

Etiology and Pathology. Some etiological factors have been already mentioned. In onychauxis local irritation from traumatism, pressure, and occupations exposing the parts to the action of chemical and other agents, as well as to heat and cold, may all be factors. Dr. [illegible] lays stress upon the association of dysidrosis, with its frequent attendant circulatory disturbances. The condition occurs also through neglect, also in connection with constitutional diseases, such as lepra.

Fig. [illegible]



[illegible]

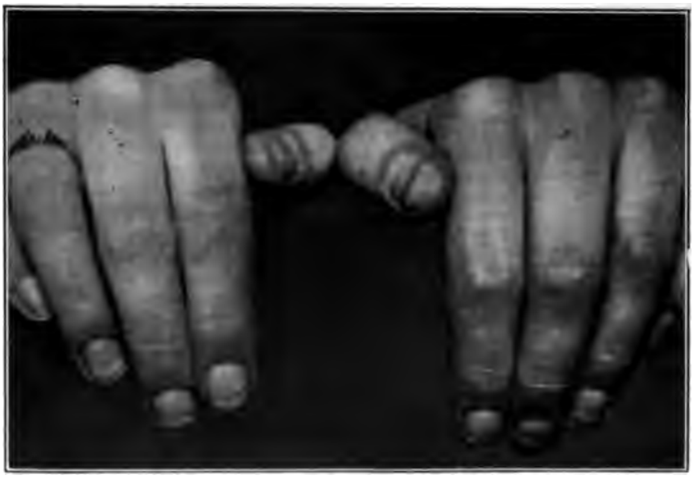
[illegible]

Treatment. [illegible]

In onychauxis any circulatory disturbances present should be corrected. The diet should be restricted, stimulants avoided, and sweets used in moderation. The systemic conditions found should be treated in accordance with the rules laid down in general medicine. In the anemic and asthenic, ferruginous preparations, cod-liver oil, and tonics in general are indicated.

Locally, the nails may be bathed at night with mild alkaline solutions containing bicarbonate of sodium or biborate of sodium, after which the irregularities may be removed to a degree by scraping with glass or other sharp instrument. Shampooing with the tincture of green soap during the process is of value, and the application of a soothing ointment for the night is indicated. The latter may contain zinc oxid and bismuth, lead oleate, or other similar preparations. Much time is required in some cases to accomplish results.

Fig. 438



Transverse furrows, following scarlet fever. (Foerster.)

The management of exaggerated cases of onychogryphosis is surgical.

In onychia local antiseptic dressings are employed. In cases with suppuration, the focal points are to be drained by incision, and in severe cases removal of the nail may be necessary. In most cases thorough local disinfection is required and the application of preparations such as ammoniated mercury, ichthyol, or salicylic acid in the form of a lotion or an ointment.

Paronychia, being chronic in many cases, requires great care for its relief. The local treatment suggested for onychia is indicated here. Radiotherapy has been of the greatest service to the author. In cases with ulcerations and granulations, silver nitrate may be used. Plaster-mulls containing ichthyol or mercury may be used in certain cases. Morrow and Lee[1] obtained uniformly good results by using a saturated solution of chrysarobin in chloroform, this preparation being throughly swabbed and packed into the area, occasionally preceding the packing by a slight incision, if necessary. In a group of 16 cases recovery took place in from one to three weeks. Finally, in these cases, the irritating cause of the disease should, if possible, be removed. Morrow and Lee discovered no significance in the vocation of their patients.

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[1] Loc. cit.

The treatment of the several varieties in which atrophy is a prominent feature resolves itself into the management of the general condition with which it is associated. A number of the disorders require no treatment, and in a number of others the treatment in the way of local applications is similar to that outlined above. Arsenic has been of value in some cases.

MORBID CONDITION OF THE NAILS INDUCED BY CUTANEOUS DISEASES.

Psoriasis.—The nails of the hands and the feet (one, several, or all) may be slightly or extensively changed in psoriasis. Most commonly, there is a concurrent psoriasis of the general integument; but in rare cases the nails only are involved. There are several types of this localization.

In the most common form the first symptom of a variation from the normal occurs in the distal portion, as distinguished from many of the eczematous changes in the nail which spring from the root. In this initial stage, the margin of one or more nails near the free border loses its natural hue; the edge of the plate is visibly loosened from its attachments; and a thin, granular mass interposes between the damaged portion of the nail and its bed. The plate at this point, being friable, may either remain in place by reason of its attachment to the sound portion, or it may break away. Patients usually pare off this portion before it is presented for examination. The process slowly advances upward to the root of the nail on one or both sides. As a rule, the nails attacked seem to be indiscriminately selected; in other cases, however, there is symmetrical involvement of all the nails of both hands and feet.

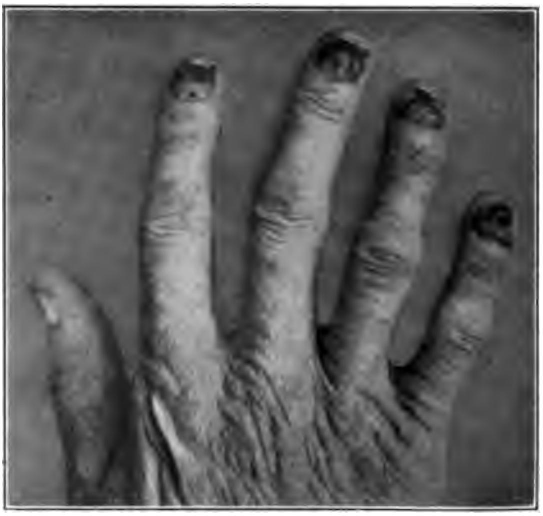
In what has been termed the "pure type" of psoriasis of the nails (*consomption dartreuse*, of Alibert; *psoriasis punctata unguium*) the process is less common and rather more conformable to that observed in tegumentary lesions. Multiple, pinhead-sized and smaller punctate lesions, often rather regularly disposed, represent points of softening of the nail-substance, where, after desquamation of the horny material, equally minute sunken depressions are left in the plate, a condition which has been likened to the exterior surface of a thimble. When the process is both exaggerated and diffused, a deep transverse furrow spreads across the nail-plate; the latter, both above and below this groove, may be normal.

In many cases of psoriasis the changes when well advanced are difficult to classify, the picture presented being that of numerous lesions due to malnutrition. "Worm-eaten," pitted, friable, and discolored nails, some split, some fractured, may leave a crumbling-edged, well-attached stump, the distal quarter or half the plate missing; and the exposed matrix covered with an imperfectly formed, horny epidermis.

In many cases where psoriasis affects the nails there is well-marked subungual keratoma, which may proceed to the point of partially detaching the nail from the bed, though the former is not often actually shed. In exceptionally severe cases, the nails are greatly thickened, distorted, dislocated, or destroyed.

Eczema.—Changes in the nails are frequently found in association with eczema of the fingers or other portions of the cutaneous surface. They are characterized by their multiformity and are largely due to nutritional disturbances.

Fig. 439



Eczema of the nails. (MacKee.)

When the parts adjacent to the nail-plates are the seat of an eczema, the latter are well-nigh invariably changed, losing their normal color and becoming discolored and dirty-yellowish in hue, furrowed, "worm-eaten," brittle, split in various directions; and the nail-bed may become the seat of a parakeratosis, at its lateral and distal portions inducing collections of scales under the plate, which increases its convexity. Later this plate may be thinned and finally exfoliated. The chief change is a marked interference with nutrition. Many of the changes noted are secondary (as in the eczematous skin), the sequels of traumatism, or friction, operating upon a weakened surface. Eczemas practically limited to the finger-tips, associated with nail-changes, occur often in workers in chemicals (*e. g.*, in physicians, chemists, photographers), in bartenders, laundry-workers, grocers, confectioners, and the like. In right-handed persons, the right hand is commonly most involved, and the most employed parts of the hand (thumb, index, and adjacent fingers) in proportionate measure show the character and grade of the local irritation.

Diagnosis.—Psoriasis and eczema of the nails are to be distinguished from ringworm, favus, syphilis, and onychia caused by pus-infection of the same region. The coincident involvement of the skin with the disorders mentioned, and a microscopic examination of nail-scrapings, are the chief aids in making the differentiation.

Treatment.—The local treatment of psoriasis of the nails is by the use of ointments containing salicylic acid, ammoniated mercury, or tar. Radiotherapy is an excellent method when used cautiously. In eczema soothing applications are required. An oily or other lotion, or the soothing ointments suggested in the treatment of eczema of the skin, should be kept applied constantly on dressings. In some cases more stimulating remedial agents may be necessary.

Internally, arsenic has occasionally been of value.

Other dermatoses in which the nails are secondarily affected, usually as complications only of the original process, are the several forms of pemphigus, dermatitis herpetiformis, epidermolysis bullosa hereditaria, pityriasis rubra pilaris, and pityriasis rubra. In most of these disorders the nail-changes (thickening, thinning, discoloration, subungual hemorrhage, loosening, and splitting) are resultants of the general disorder. In all type-cases of pityriasis rubra the nails are involved, being usually dislocated from the bed and often shed. In the early stages of the disease, the changes in color and nutrition of the nails are well marked.

Generalized alopecia areata is frequently accompanied by nail-changes, including leukonychia, onychorrexis, and other atrophic and dystrophic changes.[1]

SYPHILIS OF THE NAILS.[2]

The nail may be attacked by the initial lesion of syphilis, or be the seat of the later manifestations of the disease. Chancres of the finger are often seated in the nail-folds, partly in consequence of the frequency of hang-nails in that region, partly because of the exposures incidental to the use of the digits.

The chancre is usually single, and may occur as an indolent, indurated fissure, accompanied by the usual adenopathy in the epitrochlear region; or, beginning as a painless, indurated nodule, may develop into a circumscribed, dull-reddish, exuberant mass. The tumor-like projection usually ulcerates superficially. Occasionally, cases are observed where there is uninterrupted evolution of the lesion, as the surgeons, midwives, and others commonly infected have usually cauterized, excised, or otherwise treated the sore. Some of them suffer simultaneously from sepsis, and, aside from the accompanying epitrochlear and axillary adenopathy, develop febrile temperatures, have axillary abscesses, and suffer greatly in health before the syphilitic process is distinctly recognized.

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[1] Sequeira, Brit. Jour. Derm., 1912, xxiv, p. 122.

[2] White, C. J., Boston Med. and Surg. Jour., 1902, cxlvii, No. 20, p. 537; Adamson, Brit. Jour. Derm., 1910, xxii, p. 53; and Adamson and McDonagh, ibid., 1911, xxiii, p. 68 (a good description of the dry forms of onychia syphilitica).

In yet other cases a wide-margined, florid, and exuberantly granulating fungus mass springs from the nail-fold, capped with a sanguineous ulcer, the nature of which is long unsuspected. This induces parallel ridges in the nail-plate, or, by ulceration, may cause its exfoliation (White).

Syphilis of the Nail-plate (*Syphilonychia sicca, Friable onychia, Scabrities unguium syphilitica.* Fr., *Onyxis syphilitique*).—In this condition the nail may be attacked in whole or in part, and in the latter event with definite contour of the involved area. The distal portion is commonly first involved, the horny plate losing its polish, becoming dull-reddish or yellowish-white in color, friable, cracked, thickened, roughened, and fissured. The nail-folds may be secondarily infiltrated and scaling. In some cases the nails are considerably thickened; in others pinhead-sized, necrotic, sharply defined points open to the matrix; the condition strongly resembling the similar change seen in psoriasis. Longitudinal striations may occur, such cases having been recorded by Heller[1] and Adamson.[2] One or several of the plates may be shed in a painless process, though prompt amelioration may occur under treatment.

Pigmentation of the nails in secondary syphilis, occurring as black spots in the lunule, progressively increasing and developing coincidently with the generalized specific exanthem, though not even or uniform in deposit, is described by Vörner.[3]

Syphilis of the Nail-Bed and Matrix (*Paronychia syphilitica ulcerosa*).—The paronychia due to syphilis is the more frequent of the nail-symptoms of that disease, and may first attack the nail-wall or fold, which then becomes dull-reddish in hue, infiltrated, and scaling. After persistence the plate begins to show the changes seen when the latter is primarily attacked. The process is indolent, though under appropriate therapy it may terminate before ulceration sets in.

In other cases a papule, pustule, or an infiltrated and indurated nail-wall breaks down with ulcer-formation, attacking the border of the nail and extending beneath the plate, which undergoes the secondary changes already described. In severe cases the nail, after turning a greenish-black hue, is dislocated to one side or shed, and the entire matrix becomes the seat of an extensive and spreading ulcer. The phalanx becomes swollen, clubbed, and painful; and abortive attempts at new nail-formation may be recognized. Taylor describes a rapid necrosis, beginning with a brilliant, diffuse redness of the entire phalanx, in which the nails are destroyed "as if struck by a blight," resulting in grave ulceration, with lymphangitis and adenopathy. The entire forearm becomes reddened and swollen, due to a sequestrum of the embedded portion of the dead nail.

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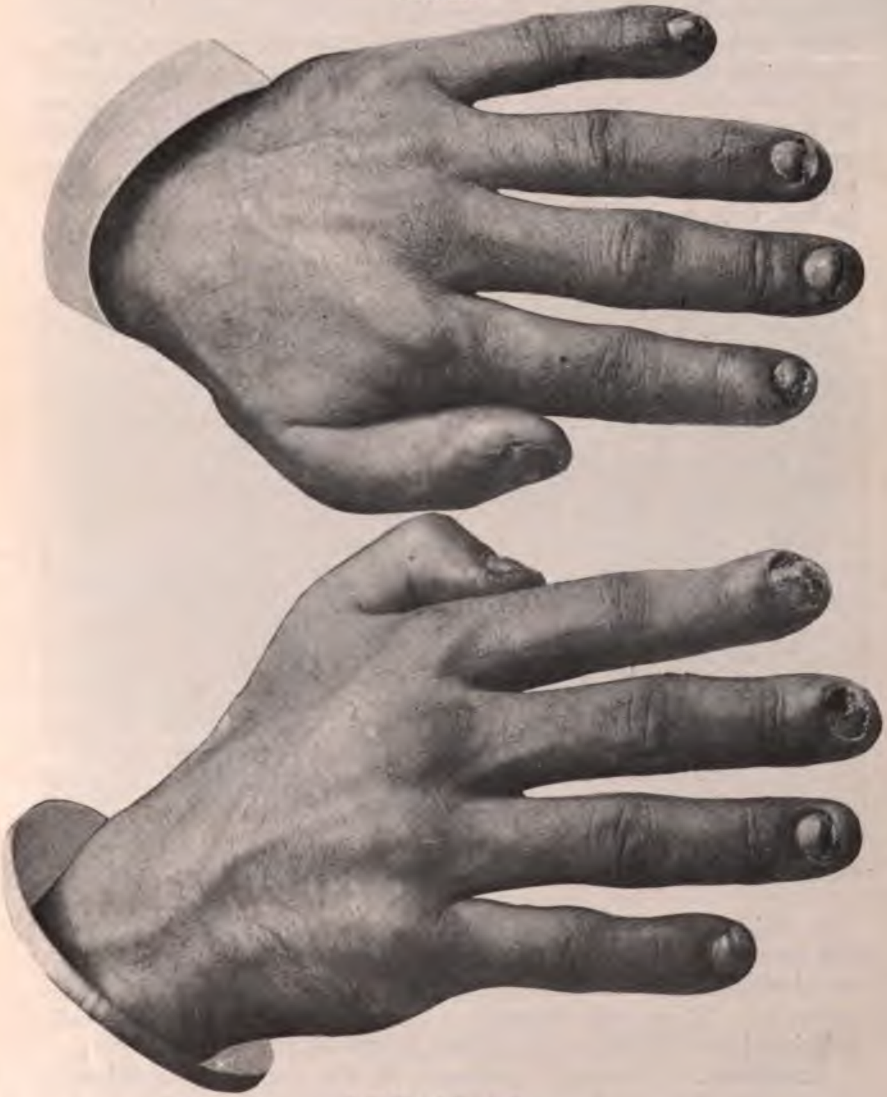
[1] Zeitschrift, 1909, xvi, Heft. 1, p. 31; abstr. Brit. Jour. Derm., 1909, xxi, p. 130 (Striæ longitudinales medianæ unguium syphilicæ).

[2] Loc. cit.

[3] Münch. med. Wochenschr., December 10, 1907; abstr. Jour. Cut. Dis., 1908, xxvi, p. 437.

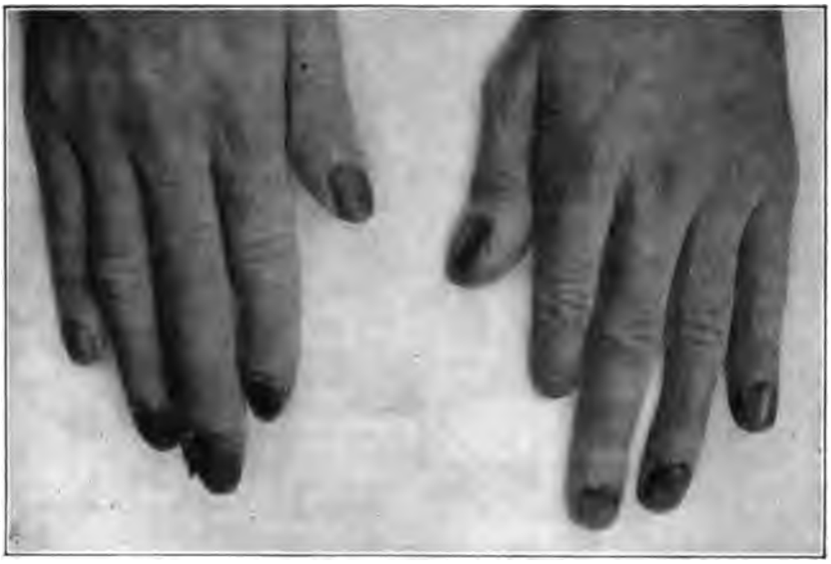
Fig. 440



Syphilis of the nails.

In congenital lues the nails are less often affected than in the acquired forms of the disease. In both the process may assume the onychia type. In the former the resulting onychia is often a part of a specific dactylitis, the earliest lesion being a papulo-pustule at the margin of the nail, which bursts and leaves an ulcer extending to the matrix and surrounding soft parts. The phalanx on which the nail is implanted is the seat of a painful osteitis, becomes club-shaped, and the ulcer, when fully formed, presents the characteristics of specific tissue-loss in general, with everted edges, sloughy floor, and indolent infiltration of the skin in the vicinity. In other cases a chronic inflammatory process affects the soft parts about the nail, and the plate undergoes consequent changes, losing its polish and becoming dirty-yellowish in hue, thickened, friable, and furrowed.

Fig. 441



Onychia and paronychia occurring in conjunction with a generalized pustular syphiloderm.

In Vajda's case, cited by Shoemaker, a speckled appearance of the nail first appeared, due to splitting of the young nail-substance into undulating lamellæ, with a corresponding "wavy arrangement of the nail-cells." There was enormous, massive thickening of the nail, associated with hypertrophy of the papillæ of the bed.

Treatment. — Energetic treatment of the systemic condition is required in all luetic diseases of the nails. The special mode of such treatment depends, as a rule, upon the time which has elapsed since infection; but the existence of a well-marked nail-lesion of undoubted syphilitic character always points to the urgent need of remedies directed to the correction of the toxic disorder. As a rule, most of the diseases of the nail due to syphilis are both chronic in course and rebellious under even energetic remedies.

In most of the dry and non-ulcerative affections, shampooing of the nails should be practised daily. When scraping or filing of the nails is required for the purpose of removing crumbling tissue, a previous soaking in a 50 per cent. aqueous solution of potassium hydroxid may be required. At night a pomade should be applied, containing mercurial ointment, 1 part to 2 or 3 of lanolin oil, kept in place by a cot. For this may be substituted ammoniated mercury, 1 part to 15 or 30 of cold-cream salve. The sulphur salves in 10 per cent. strength, with the red sulphid of mercury added in the strength of 10 decigrammes to 30 of salve basis, are often efficient.

In ulcerating nail-disorders the treatment of the attacked parts is very largely that of ulcerations elsewhere. The strong caustics once advocated are now much less frequently applied. Soaking in bichlorid of mercury solutions, 1 to 1000, is preferable; and when granulations are present pencillings with a 10 to 15 per cent. solution of argyrol or silver nitrate. Plaster-mulls of hydrargyrum serve a useful purpose, especially in the dry forms; but may be used also in the others by thoroughly cleansing the area each time before their application.

ONYCHOMYCOSIS.[1]

The diseases of the nails due to invasion by vegetable parasites are much less frequent than cutaneous affections of similar origin. It is, however, accepted that this group of diseases is much more common than is generally believed. A single nail of the hand or foot (occasionally several) has exhibited morbid symptoms year after year before the exact nature of the change was recognized. It is undetermined whether ringworm or favus of the nails is more common in this country. Of 101 cases observed by Foster[2] 84 were ringworm and 17 were favus.

Trichophytosis Unguium (*Tinea trichophytina unguis, Ringworm of the nails.* Fr., *Onychomycose;* Ger., *Schimmelpilzmykose der Nägel*). —Ringworm of the nails is an affection of great rarity. It was formerly thought that the trichophyton ectothrix was the most common cause of the affection, but recent cultural experiments reveal the endothrix most frequently, especially in England and Scotland.[3] Whitfield, reporting Low's findings, states that in three cases he obtained an endothrix and in one an ectothrix, and further states that an endothrix was demonstrated by Pernet[4] and Sequeira.[5]

There may be coincident ringworm of the body,[6] and the parasite may attack one nail only or several or all.[7] After invasion of the nail, the plate becomes changed in color, consistency, and shape, the process beginning commonly in the anterior border of the lateral fold. After infection of the nail-bed and matrix, the nail becomes friable and breaks away irregularly from its attachment; there is often subungual débris of cells; and the hyphomycetic invasion extends in longitudinal striæ toward the lunula. As usually seen, the nail

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[1] Grindon, Trans. Amer. Derm. Assoc., 1901, p. 111. Vignolo-Lutati Giorn. Ital. d. Mal. Ven. e. della Pelle, 1917, fasc. i, p. 54. Contribution to the study of Tinea unguium Familialis.

[2] Jour. Amer. Med. Assoc., August 22, 1917.

[3] Low, Edinborough Med. Jour., 1911; abstr. Brit. Jour. Derm., 1911, xxiii, p. 86 (in seven out of eight successful cultures the endothrix fungus was found).

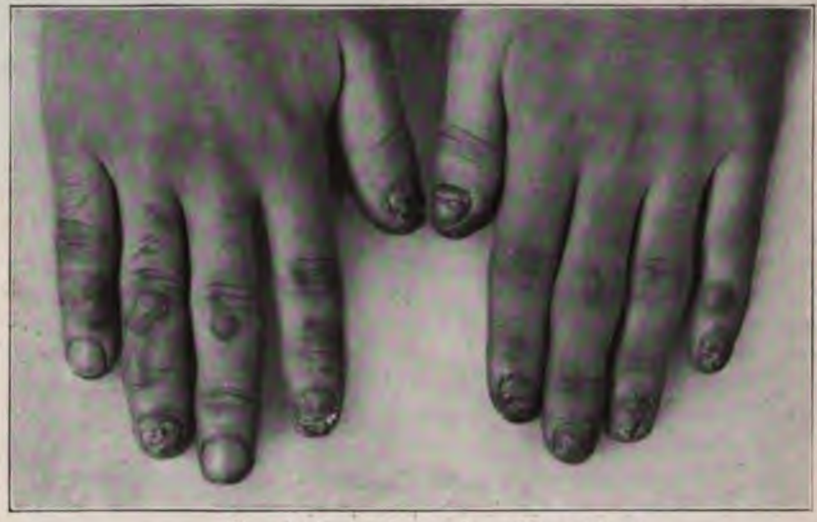
[4] Brit. Jour. Derm., 1901, xiii, p. 476.

[5] Ibid., 1906, xviii, p. 269.

[6] Ravogli, Jour. Amer. Med. Assoc., July 27, 1907, p. 308 (with four illustrations).

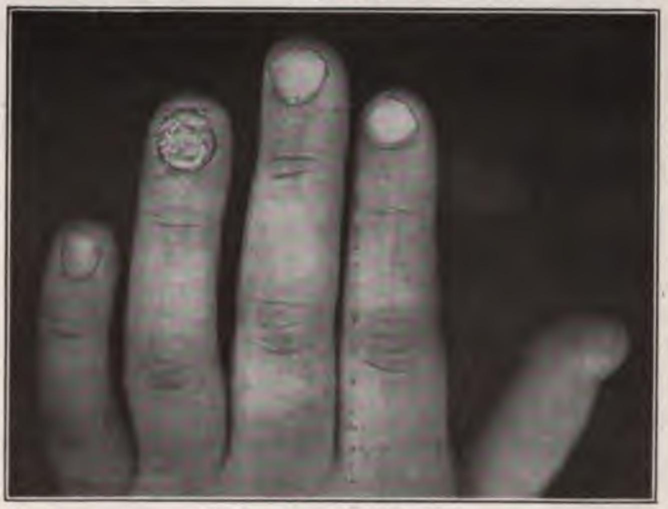
[7] Lancashire, Brit. Jour. Derm., 1912, xxiv, p. 286 (all nails of hands and feet involved; contracted from cattle).

Fig. 442



Tinea trichophytina unguis. (Howard Fox.)

Fig. 443



Trichophytosis unguium.

surface is irregular, showing ridges and depressions; is spongy, granular, and of a yellowish or brownish hue. The anterior portion of the plate is finally cast, leaving a stump near the lunula. Marked deformity of the plate is thus induced.

Onychomycosis Favosa (*Tinea favosa unguium, Favus of the nails.* Fr., *Onychomycose favique, Favus des ongles*).—In favus of the nails the effective parasite is the achorion of Schoenlein, usually implanted on the nails by autoinfection from scutella.

The nail-changes in favus closely resemble those described in ringworm. The nails become yellowish, brownish, or blackish in hue, and are traversed by ridges and furrows. Subungual masses of cells form, though no true scutella; a dirty-whitish, dry powder, containing scales, forming both beneath the free border and beneath the attached plate, raises the latter from its bed. The nail may present a honeycombed appearance, and later disintegrate and largely disappear.

Diagnosis.—The diagnosis of both ringworm and favus must be confirmed with the microscope. Greater difficulty is experienced in demonstrating the fungus in the nail than in scrapings from the skin, but by allowing more time for the potassium hydroxid to dissolve the nail-cells it can usually be successfully accomplished. When the disease is present on the skin or in the scalp, the diagnosis is more readily made.

Treatment.—In the treatment of both trichophytosis and favus of the nails, it is first necessary to remove as far as may be the horny substance, which interferes with the penetration of a parasiticide. The plate should be first scraped and then anointed with some substance having power of penetration, such as the mercuric oleates, 10 to 20 per cent. or stronger; bichlorid of mercury solutions; olive oil and pyrogallol, equal parts (Pellizzari, Dubreuilh);[1] solutions of iodin and iodid of potassium, 15 grains (1.) of the first to half a drachm (2.) of the second, dissolved in a liter (1000) of distilled water (Sabouraud).[2] Leistikow (quoted by Hardaway)[3] advises:

Pyrogallol,	gr. xxiv;	1.5	
Naphthol,	ʒss;	2	
Hydrarg. ammon.,	gr. xv;	1	
Tinct. guaiaci.,	ʒvijss;	30	M.

Harrison, after scraping the nail, applies on lint 1 part of the iodid of potassium to 4 each of liquor potassæ and distilled water, for fifteen minutes, after which a 1 per cent. solution of the bichlorid of mercury in equal parts of alcohol and water is kept in contact with the part for twenty-four hours. This method is advocated by Radcliffe-Crocker. Sulphurous acid from a freshly opened container, or an aqueous solution of the hyposulphite of sodium, 1 part to 6 or 10, mopped over the nail after application of dilute acetic acid, is often effective. Walker[4] advises dressing the nails for two days with Fehling's solution on lint, covered with a finger-stall, then removing the softened nail and dressing it subsequently with a 2 per cent. solution of copper sulphate.

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[1] Monatshefte, 1896, xxii, p. 212.

[2] Annales, 1896, vii, p. 33.

[3] Trans. Amer. Derm. Assoc., 1901, p. 133.

[4] Quoted by Low, Loc. cit.

The disease in either case is persistent and difficult to eradicate.

CONGENITAL ABNORMALITIES AND DISEASES OF THE NAILS.

Diseases of the nails in this group, aside from anonychia mentioned below, are occasionally exhibited in newborn infants. The most of these cases illustrate the changes described above under the titles Onychauxis, Onychogryphosis, and Onychatrophy. At times one or more, rarely all, the nails of both hands and feet are defective in production of the horny substance of the nail; or the extremity of a bulbous and deformed digit may be capped with greatly enlarged nails; or with nail-plates forming upward-projecting, horny, peg-like structures; or a well-marked subungual keratosis may have been determined. The congenital affections of the integument most often concurrent are: ichthyosis of the "hystrix" type, epidermolysis bullosa hereditaria, and, very rarely, syphilis. With these anomalies may exist partial or total absence of hair from the scalp, brows, and other regions of the body, as also failure of eruption of the teeth.

Anonychia.—Nail-plates may be wholly or partially absent in the newborn, even with normal development of phalanx, nail-bed, and nail-fold. Very rarely all the nails, more commonly one or several of those of the fingers or the toes, have been wanting. Cases of congenital anonychia are on record in which in after-life the nails did not develop. In other instances, nails existing at birth have soon afterward been shed, without further production of nail-growth. Again, the newborn suffering from intrauterine affections of the skin may exhibit a loss of nails due to such congenital disease, as in severe grades of ichthyosis (harlequin fetus), variola *in utero*. True congenital absence of the nails is extremely rare. Heidingsfeld,[1] in reporting an example, found few others in the literature.[2]

Polydactyly and Syndactyly.—Supernumerary fingers and toes commonly have properly adjusted nails. In some such anomalies, however, the nails have been wanting; in yet others double nails have existed on the supernumerary digit; and in still other cases claw-like nails have developed.

Onych-heteropia.—In the rare cases where rudimentary fingers and toes have been implanted elsewhere than upon the hands and feet, nails have been produced in anomalous situations.

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[1] Trans. XVII Internat. Congress of Med., London, 1913, Sect. 13, Part 2, p. 93.

[2] Bergé and Weissenbach, Annales, 1912, p. 244; abstr. Jour. Cut. Dis., 1913, xxxi, p. 603 (complete congenital absence of nails of all fingers).

DISEASES OF THE MUCOUS MEMBRANES IN PROXIMITY TO THE SKIN.

COINCIDENT involvement of the mucous membranes and the skin occurs in a number of diseases, such as lichen planus, erythema multiforme, pemphigus, and many others, their characteristics having been outlined in connection with the general description of the disease. In this chapter a few independent disorders not infrequently seen by the dermatologist are considered.

LEUCOKERATOSIS BUCCALIS.[1]

Synonyms.—Leucoplasia, Leucoma, Psoriasis linguæ, Smokers' patches of the mouth, Buccal psoriasis, Ichthyosis linguæ, Tylosis linguæ, Leucoplakia buccalis. Fr., *Leucoplasie, Plaques blanches de la bouche.*

In the year 1868 Bazin described with accuracy the several conditions indicated by the names given above; and since that date the subject has been enriched by a literature contributed to by Debove, Kaposi, Sigmund, Plumbe, Mauriac, Schwimmer, Ingals, and others.

Symptoms.—The disease as manifested in the mouth, is characterized at the outset for some weeks or months by special sensitiveness to irritation produced by ingesta; later, by the occurrence on the inner faces of the lips and cheeks, and on the dorsum and edges of the tongue, of sharply outlined, dull-whitish, slate-colored, or silver-white points, disks, streaks, bands, ribbons, or patches of an irregular shape, either flattened or slightly elevated above the general level of the mucous surface. The aspect of the lesions often suggests that they have been lightly penciled with the nitrate of silver. The disease may occur in isolated points or in pinhead-sized nodules, discrete or confluent, and in cases grouped, the grouping being often in linear arrangements following the lines indicated by the streaks or the striæ of similar composition. According to Butlin, the smoker's patch occurs most frequently near the center of the tongue and begins as a smooth, erythematous spot, or the early change may be a bluish-white or pearly area.

The sites of election of the lesions are: the inner face of the cheek in a line following that traced by the conjunction of the teeth of the upper and lower jaw when approximated; the gums above the upper canine teeth and lateral incisors; the sulcus beside the upper and lower gums in the roof and floor of the mouth; the dorsum and edges of the tongue, where the arrangement is usually in lines along the longitudinal axis.

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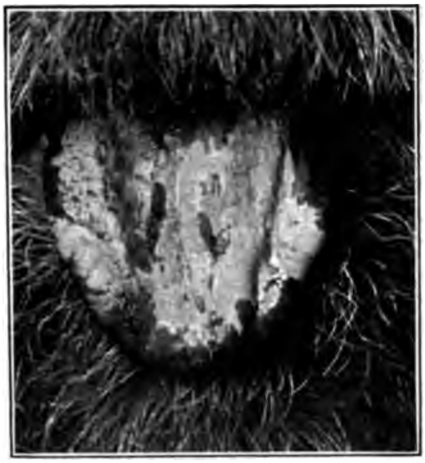
[1] For bibliography, see Bénard, La Pratique Derm., ii, p. 969; and Butlin and Spencer, Diseases of the Tongue, London, 1900.

When closely examined these lesions are found to be made up of a hyperkeratinized epithelium, being covered by an adherent and more or less dense pellicle, removable only by artificial measures and closely applied to the inferior stratum of the mucosa. The lesions are rough to the touch, both to the finger of the physician and to the tongue of the subject of the disease, but are, as a rule, not tender, though at times annoying by producing a certain degree of stiffness and immobility of the parts affected. At times the membrane in the vicinity is reddened and tender. In other cases projecting, thick, rough nodules develop, or a dense, well-defined, elevated plaque.

These lesions are extremely chronic of evolution, requiring months and often years for their full development, and resisting in a remarkable way the action of topical medicaments. They may be removed without recurrence; or may recur after complete and radical ablation. If unmolested and not undergoing resolution (a course somewhat doubtful of occurrence), they usually, by reason of increased density, crack or fissure at one or another point, the fissure extending to the derma and arousing a local inflammatory process, with the production of pain and distress. The surface is then prone to exfoliate and ulcerate, and epithelioma of the mouth may result.

Fig. 444



Leucoplakia buccalis.

The proportion of the benign cases to those which result in epithelioma is not determined. Every leucokeratosis, however, may prove the initial stage of epithelioma, and the treatment of the former is, therefore a matter of no little consequence. Leucokeratosis is prone to develop fissures, and in cases where the patches are thickened or verrucous epithelioma often results. The cases which develop in syphilis cannot be distinguished clinically from others. They are classified by Fournier as parasyphilitic lesions, and are rebellious to treatment.

More rarely, other parts, such as the vaginal and other mucous membranes, have been involved.[1]

Etiology.—The cause of these cases is suggested by some of the names given above. The disorder occurs almost exclusively in the mouths of men, and usually after middle life. A typical and rather extensive case in a woman has been under the observation of the author. Unquestionably, the irritation produced by tobacco, whether used in smoking or chewing, and the influence of carious teeth, or those with sharp edges after fracture, irritating the edge of the tongue, are all important. We have seen a large patch of verrucous leucoplakia occupying the exact region between the cheek and teeth in which a piece of chewing tobacco had rested nightly for twenty years. We have, however, observed typical lesions in the mouths of men who had never contracted syphilis nor used tobacco. The resemblance of these lesions to the mucous patches of syphilis is obvious; and it is believed that syphilis, when not actively efficient in the production of leucokeratosis buccalis, may be one of its indirect causes. It is, however, important to note that all symptoms here described occur in persons who have never suffered from syphilis; and such symptoms are in the latter class as intractable as in others.

Pathology.—Butlin states that the processes going on are a proliferation of the cells of the rete Malpighii, with cellular infiltration in the papillary layer of the corium; a gradual shrinking and disappearance of the papillæ; and the formation of some scar-like tissue immediately beneath the epidermis. It is not definitely known whether the primary change is a pure hyperkeratinization of the epithelium or an inflammatory process of the papillary layer. The horny layer is hypertrophied, the cells retaining their nuclei. Leloir insists that the epitheliomatous process always begins, not at the level of the hyperkeratosis of the mucous membrane, but below the fissure or other lesion induced by the induration of the plaque or streak; indicating, in other words, that the epitheliomatous change is rather an accident than an essential part of the process.

Diagnosis.—The diagnosis is chiefly from syphilitic lesions of the mouth, which should be recognized, as a rule, by their softness and tendency to ulcerate, as well as by their situation, which is far less distinctive than in the case of leucokeratosis of the mouth. A history of infection and of symptoms of the disease in other regions of the body will usually indicate the nature of the process.

Another malady likely to be confounded with leucokeratosis of the mouth is lichen planus; and it is important to note that some confusion exists on this point in several descriptions of the two diseases.

In lichen planus of the mouth there may be recognized over the tongue, the palate, and other parts, dense, smooth or fissured plaques, rings, festoons, linear striæ, or disks, covered by a silver-white pellicle. It is clear that the distinction between these and leucokeratotic lesions is in a high degree obscure. The presence of the lesions of lichen planus upon the cutaneous surfaces of the body would make the differentiation.

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[1] Pflanz, Zeitschrift, November, 1899, xvi, p. 710; abstr. Brit. Jour. Derm., 1910, xxii, p. 205: On leucoplakia of the mucous membranes, with special reference to leucoplakia of the genital region. Bohac, Archiv, Bd. cv, Heft. 1 and 2, p. 179; abstr. Brit. Jour. Derm., 1911, xxiii, p. 126: Leucoplakia and kraurosis of the mucous membrane and skin.

Treatment.—The treatment of leucokeratosis of the mouth is first by abstention from all local irritants (tobacco; highly spiced, heated, acetous, and iced articles of food and drink); by the care of the teeth; and by the employment of soothing sprays or lotions containing potassium chlorate, boric acid, balsam of Peru, iodized phenol, myrrh, or chlorid of iron.

Silver nitrate may be applied to any ulcerated or fissured points, both in solution and by sweeping the solid crayon over the surface. The French make use of the salicylates in the same way.

Destruction or removal of the lesions may be secured by the employment of caustics, chemical or galvano-cauteric; by erasion with a curette; or by surgical ablation. When practicable, the burr of the dental engine may be used, after injection of cocain hydrochlorid. Where the patches are not too dense and extensive, this has generally been productive of good results. Vidal employed a 20 per cent. solution of chromic acid.

Sherwell[1] reports complete removal of the patches by the use of undiluted liquor hydrargyri nitratis. The mouth is stuffed with cotton to protect adjacent parts and the solution is applied and allowed to remain from fifteen to twenty minutes, after which it is neutralized with sodium bicarbonate. If necessary, the application may be repeated two or three times, at intervals.

Pierce was successful in one case after rubbing into the patches pyoktanin blue, followed immediately with an aqueous solution of anilin oil. The applications were made daily for three months. In selected cases *x*-rays and carbon-dioxid snow may be employed.

For leucokeratosis of the vulvo-anal region complete excision has given the best results.

Prognosis.—The prognosis is fairly favorable in the case of all subjects of the disease who consent to deny themselves absolutely the luxury of tobacco in every form, and who can follow a prescribed hygienic and medicinal course. For all others there is more danger of epithelioma.

GROOVED TONGUE.[2]

Synonyms.—Furrowed tongue, Wrinkled tongue, Sulcated tongue, Scrotal tongue, Ribbed tongue, Fluted tongue.

In this disease the tongue may assume the characteristics suggested by the above titles as the result of a superficial glossitis, or occur as a congenital or family disease. In certain cases a furrowed tongue indicates a slight stage of macroglossia, the tongue being too large for the mouth. Fine furrows occur on the dorsal aspect of the tongue in many persons. The major portion of people have such a furrow extending along the median line of the tongue. This in inflammatory conditions more readily becomes the seat of excoriation and ulceration, and is obstinate to treatment, on account of the constant contact of the sides of the furrow. Natural furrows also occur on other parts of the tongue, usually running in a longitudinal direction; they may, however, be curved or forked. Following inflammatory and other conditions, numerous furrows may be seen running over the surface of the tongue in all directions, producing a lobulated and grooved condition, and occasionally becoming so numerous as to present the appearance of a network. Particles of food accumulate in these situations and by undergoing decomposition produce irritant effects.

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[1] Jour. Cut. Dis., 1899, xvii, p. 185.

[2] Butlin and Spencer, Diseases of the Tongue, 1900.

The disorder may be the result of syphilis, inherited or acquired, the latter producing a glossitis, which is followed by the furrowing. Smoking, drinking, and the friction of carious teeth are also predisposing causes of the condition. In older patients gout is given as a factor.

As a rule, there being no subjective symptoms and no sequels, treatment is not required. The tongue, however, should be kept clean, the individual depressions swabbed, and particles of food should not be allowed to accumulate. Mild alkaline and antiseptic mouthwashes may be employed. As the condition is a deformity, it cannot be overcome.

TRANSITORY BENIGN PLAQUES OF THE TONGUE.[1]

Synonyms.—Glossitis areata exfoliativa, Pityriasis linguæ, Annulus migrans, Exfoliatio areata linguæ, Erythema migrans, Wandering rash, Circinate eruption of the tongue. Fr., *Desquamation aberrante en aires de la langue, Erythème ambulant;* Ger., *Zungenfratt.*

Wandering rash is an uncommon disease, and, though essentially a children's disease, has been occasionally observed in adults. Both sexes are equally liable to its attack. The disease consists of one or more patches on the dorsum of the tongue, which in the early stages are pea-sized and smooth, red, and on the same level as the surrounding surface of the tongue, although they may appear slightly depressed or slightly elevated, according to the condition of the tongue and the thickness of the fur. The filiform papillæ have been shed, but the fungiform papillæ may remain and even appear more prominent. The patch soon spreads and becomes a ring, either circular or oval. Its center is smooth and redder than the normal mucous membrane, the redness increasing toward the border of the ring. The border is faintly or decidedly yellow in tint, and has been described as being a golden-yellow. It is slightly raised and sharply outlined. The patches almost invariably form on the dorsum or border of the tongue, and are more commonly observed toward the tip, though they may affect any portion of the dorsum in front of the circumvallate papillæ. They present no evidences of surrounding inflammation. Occasionally, they may extend over the dorsum to the under aspect of the tongue near the tip.

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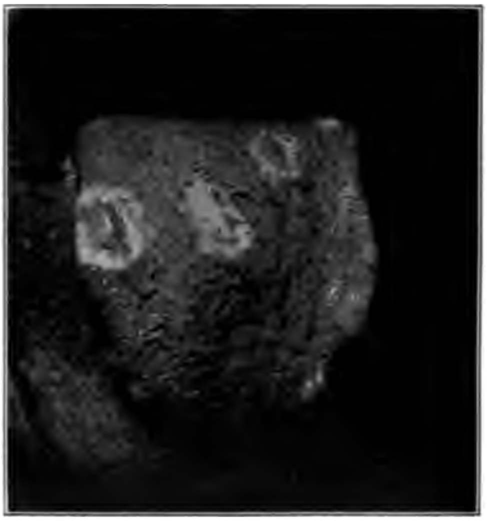
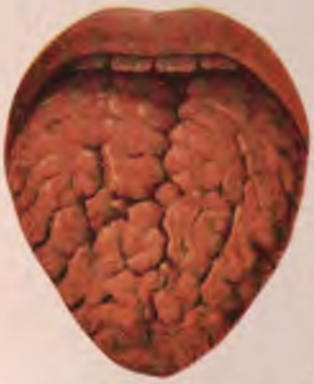
[1] LITERATURE: Moeller, Deutsche klinik, 1851, No. 26, p. 273. Parot, Progrès méd., 1881. Caspary, Vierteljahr., 1880, p. 183. Colcott Fox, Lancet, 1884, p. 842. Kinnier, Jour. Cut. Dis., 1887, v, p. 56. Böhm, Volkm. Sammlung klin. Vorträge, 1899, p. 249. Butlin and Spencer, Dis. of the Tongue, 1900, p. 94 (to this article the author is much indebted). Klausner, Archiv, ciii bd., 1 heft, p. 103 (extensive literature).

The evolution of the lesion is described by Butlin as follows: "If one of the patches or rings is watched, it is observed to grow larger, until, widening out, it reaches the border of the tongue; then, losing its circular or ring shape, it forms a segment of a circle, the other segment of which may be found on the under surface. Several rings on the same half of the dorsum, by peripheral extension, intersect each other, but the intersection is not complete, for where they come in contact the border of one of them ends abruptly, while the border of the other continues to advance unbroken. The meeting of the rings and the peculiar marking of the dorsum of the tongue which is produced by it led to the name 'geographical tongue.' As the circles widen out, so they may contract again, until each and every circle may disappear from the surface of the tongue. The disappearance may be so rapid that for a time the surface is redder and smoother than normal. Again, as the circles enlarge, fresh patches may form in their interior and by their peripheral extension form rings within

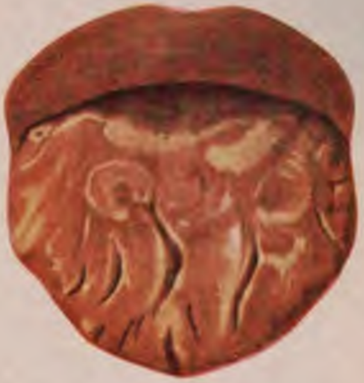
FIG. 445



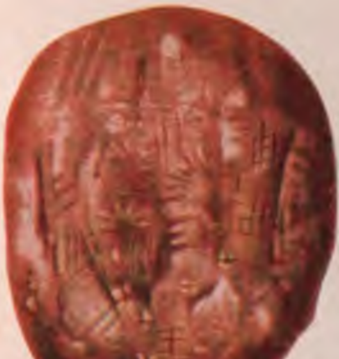
Glossitis exfoliativa.



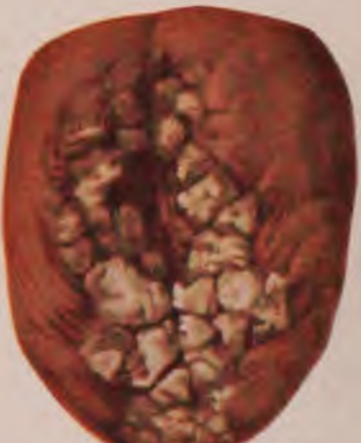
Congenital Hypertrophy of Tongue.



Leucoplakia of Tongue.



Leucoplasic Striæ of Tongue.



Epitheliomatous Transformation of Leucoplasic Lesions of Tongue.

rings, and fresh rings may form in areas from which the former rings have recently disappeared. In all these changes the circles preserve their original character of smooth, red centers and slightly raised, whiter or yellowish borders. The regularity of the outline of each ring is not always strictly maintained, but may be crenate and present projections here and there."

The disorder produces no subjective symptoms, as a rule. Occasionally, itching is complained of, which in some cases has been severe. Increase in the flow of saliva is also exceptional.

The disease has been believed to be parasitic in origin, but no parasite has been discovered. It has also been attributed to syphilis. Fournier classes some of the cases in the parasyphilitic category. The disorder does not respond to antisyphilitic treatment. Debility appears to be either a predisposing or exciting cause. Many cases have been seen in delicate children. The histology as described by Parrot showed tumefaction of the epithelium at the level of the patches, with thickening of the rete Malpighii, and enlargement of the cells of the stratum corneum. In the papillary and subpapillary layers of the corium a perivascular and cellular infiltration of lymphoid cells occurs. It is therefore seen to be a subacute inflammatory process of the mucous membrane of the tongue.

The disease is obstinate, and its course is marked by exacerbations and partial recovery. The life of each ring is short, only lasting six or seven days, but the entire process may be prolonged. Its differentiation from mucous patches of syphilis and leucoplakia is readily seen.

Treatment.—The treatment consists in the constitutional remedies indicated in the individual cases. Locally, astringent lotions containing tannin, alum, sulphate of zinc and chlorid of zinc, or soothing lotions containing borax, potassium chlorate or chromic acid, are recommended at various times. All local irritants should be interdicted. Radium has been successfully employed by the author in several instances.

THE HAIRY BLACK TONGUE.[1]

Synonyms.—Lingua nigra, Hyperkeratosis linguæ. Fr., *Langue noire, Langue noire pileuse, Negritie de la Langue;* Ger., *Schwarze Haarzunge.*

Hairy tongue is produced by an overgrowth of the epidermis of the filiform papillæ, which gives the clinical appearance of hairs. These epithelial filaments may become from one-fourth to one-half of an inch in length. The color is often black, but it may be a sepia-brown, yellow, or even blue. The black patch occurs most frequently in the middle of the dorsum of the tongue, in front of the "V" formed by the circumvallate papillæ; less frequently behind this area or near the end of the tongue. The lesion is darkest in the center and fades toward the edge of the patch, where it may be a light-brown. As a rule, the discolored area is at first of small size, but extends slowly until it covers a large portion of the dorsum of the tongue, and it may persist for a few weeks up to many months. Disappearance is by a gradual desquamation of the epithelium, the color fading to a brownish-yellow at the border. Recurrences may happen. In exceptional cases, the discoloration is all that has been noticed, but as a rule the papillæ are enlarged and elongated. There seems to be no connection between the discoloration and the overgrowth of the papillæ, the former being due, apparently, to the various microörganisms.

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[1] For review of the literature, see Heidingsfeld, Jour. Amer. Med. Assoc., 1910, lv, p. 2117 (description of two cases, with histological study); and Butlin and Spencer, Diseases of the Tongue, 1900, p. 145.

The appearance of the tongue has been compared in different cases to various objects. In one the surface area was described as "like a field of corn laid by the wind and rain." In another the elongation of the papillæ was likened to feathers with a central stem and lateral webs. The cause of the disease is not known. Heidingsfeld believes that there are two forms, a true and a false. The former he ascribes to some anomaly of development, probably of congenital origin.

As above stated, the question of color is distinct from the origin of the hyperkeratosis. As a rule, no subjective symptoms are present. Slight pain and a mawkish taste have been described after the attention of the patient has been attracted to the presence of the disorder.

The duration of the disease is variable, but it ultimately tends to spontaneous disappearance, and in no case is followed by sequels. Treatment has been unsatisfactory. After shaving or scraping, the patch recurs, but later disappears of its own accord. If necessary, a 2 per cent. solution of salicylic acid may be applied, or a weak solution of phenol rubbed in three or four times daily. Lactic acid has also been employed.

CHRONIC SUPERFICIAL EXCORIATIONS OF THE TONGUE.[1]

Synonyms.—Moeller's glossitis.

This disorder, although uncommon, has recently been recorded by several observers since attention was again attracted to it by a study and report of two cases by the late Dr. Frederick G. Harris.

Symptoms.—The disorder occurs principally on the tip and edges of the tongue and also on its dorsal surface, occasionally on the mucous membrane of the lips, cheeks, and hard and soft palate. The lesions occur as intensely red, well defined irregular patches in which the filliform papillæ are thinned or absent and the fungiform papillæ are swollen and from which the superficial layer of the epidermis has been exfoliated. Between these areas the surface of the tongue may be smooth and whitish or opalescent. It is accompanied by sensations of burning and pain and the areas are sensitive to irritants. The disorder is chronic and characterized by periods of exacerbation and lessened activity of the symptoms. The areas above described may remain in the same situation for long periods, or undergo resolution and new patches appear. The tongue is extremely sensitive to external irritants, thus interfering with the general nutrition because of the suffering induced by the taking of food. Hot or cold articles, spiced foods, acids, or salty foods are among the chief irritants. In some instances solid food of any kind cannot be tolerated on account of the pain induced. In addition to occurring in the area the pain may radiate toward the jaw.

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[1] Harris, Jour. Cut. Dis., 1915, xxxiii, p. 742; "Chronic superficial excoriations of the tongue or Moeller's glossitis, with report of two cases," a thorough exposition of the subject with review of previously recorded cases. Idem., ibid., 1919, xxxvii, p. 628; case demonstration (his third case). Heimann, Ibid., 1916, xxxiv, p. 560; case demonstration. Wise, ibid., 1918, xxxvi, p. 324, and 1919, xxxvii, p. 415; case demonstration. Rostenberg, Archiv of Derm. and Syphil., 1920 (Jour. Cut. Dis., xxxviii), n. s. i, p. 116; case demonstration. Engman and Weiss, Ibid., 1920, i, p. 119, "Monilia candida infection of the mouth." In this article two cases of Moeller's glossitis are described.

Etiology and Pathology.—The disorder occurs most frequently between the fortieth and sixtieth years of life and usually in women. The cause is unknown. Digestive disturbances may be coincidental. In some of the early cases tapeworm was associated, but Harris concluded this was also incidental. He suggested that the process might be an inflammation of the corium or submucosa, followed by edema and cellular infiltration of the epidermis, resulting in exfoliation of the major portion of the rete, the nerve terminals thereby losing their protection and becoming easily irritated.

Treatment.—The treatment thus far recommended has but little effect on the disorder. Cauterization, astringent and antiseptic mouth washes have been employed.

Prognosis.—The disorder is serious so far as recovery from the local disease is concerned. In one case (Harris) death resulted from starvation. In the group reviewed by Harris only three of more than twenty-were reported as having recovered. The persistence of the disease, the discomfort induced and the interference with the general health are important points in forecasting the future.

CHEILITIS GLANDULARIS.

Synonyms.—Cheilitis glandularis apostematosa (Volkmann), Myxadenitis labialis.

This disorder was described by Volkmann[1] in 1870, after observation of five cases. The disease occurs chiefly on the lower lip, which becomes swollen, tumid, and tense, and is studded with pinpoint- to hemp-seed-sized elevations representing muciparous glands with dilated follicular orifices, often admitting for some distance a fine probe, from which exudes a thin mucoid or muco-purulent fluid, and at times a thin, clear serum. Purdon[2] observed four examples presenting practically the same features. In his cases there appeared to be an active catarrhal condition of the lining membrane of the cheeks and gums. A rather severe catarrhal inflammation of the mouth and throat occurred in the cases described by Sutton,[1] Howard Fox,[2] Schamberg,[3] and Wise.[4] The enlarged mucous glands of the lip may be felt with the finger as nodules beneath the mucous membrane. Not infrequently the lips are glued together in the morning by the drying of the mucoid substance.

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[1] Virchow's Archives, 1870, i, p. 142.

[2] Brit. Jour. Derm., 1893, v, p. 23.

Etiology and Pathology.—The cause of the disorder is not known. Volkmann believed it to be due to a catarrhal inflammation of the glands of the lip. It has been suggested that the inflammation of the pharyngeal and buccal mucosa is responsible for the disorder occurring in the lip. Sutton,[5] in a histological study, found the greatest changes in the ducts of the mucous glands; the corium was altered but little. He concludes that there is an adenomatous condition of the mucous glands of the lip, characterized by enormous dilatation and hypertrophy of the ducts, usually accompanied by a great increase in the amount of glandular tissue (probably congenital in origin) and subsequent degenerative changes and increase of elastic fibers in the surrounding derma.

Prognosis and Treatment.—In three of Volkmann's cases relief was obtained with potassium iodid after one or two months' administration. Local applications of various sorts have been tried without much benefit. In one of Sutton's cases *x*-rays proved of value.

CHEILITIS EXFOLIATIVA.

Synonym.—Persistent exfoliation of the lips (Stelwagon).

Cheilitis exfoliativa is an inflammatory disease of the mucous membrane of the lips, which appears to be closely related to, if not identical with, dermatitis seborrhoica. Several of the earlier reported cases were recorded under different titles,[6] but have been collected by more recent observers. Under the title given above cases have been recorded by a number of observers.[7]

The disorder is characterized by the formation of scales and crusts limited to the vermilion of the lips. In Stelwagon's and in some other cases a slight seborrhea of the scalp was associated. Some cases exhibit a constant exfoliation of scales of varying thickness and size. In several cases quite marked crusting has been recorded, the crusts forming rapidly, exfoliating soon, and reforming again. Yellowish and darker crusts are described as occurring in heaped-up masses, projecting to the extent of a quarter of an inch or more, and covering the entire

[1] Jour. Cut. Dis., 1909, xxvii, p. 150 (a report of five cases and review of the literature).

[2] Ibid. p. 229 (case presentation); ibid., 1913, xxxi, p. 415 (case presentation).

[3] Ibid., 1911, xxix, p. 449 (case presentation). [4] Ibid., p. 504 (case presentation).

[5] Unna's Festschrift, 1910, i, p. 611.

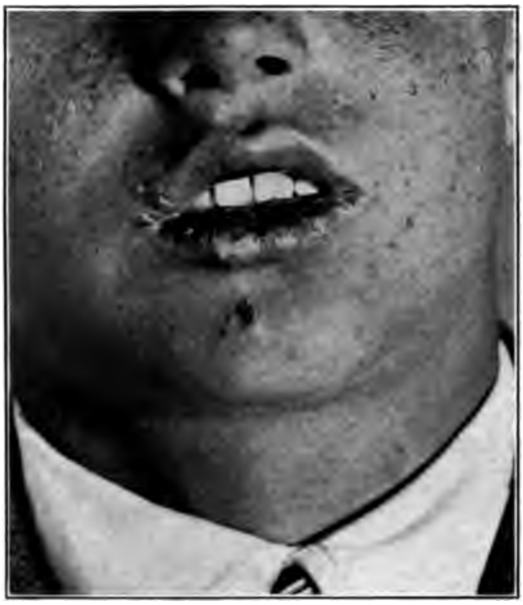
[6] Galloway, Brit. Jour. Derm., 1895, vii, p. 113; Jamieson, Brit. Med. Jour., December 7, 1895, p. 1410; Stelwagon, Jour. Cut. Dis., 1900, xviii, p. 268, and ibid., 1904, xxii, p. 351.

[7] MacLeod, Brit. Jour. Derm., 1907, xix, p. 388; Ravitch, Jour. Cut. Dis., 1908, xxvi, p. 359; Little, Brit. Jour. Derm., 1909, xxi, p. 252; Davis, ibid., 1911, xxiii, p. 149; Ocks, Jour. Cut. Dis., 1911, xxix, p. 244; Trimble, ibid., 1913, xxxi, p. 584; Gaskill, ibid., 1914, xxxii, p. 498.

red portion of both lips. On removal of the crusts a reddened and slightly fissured surface is left, upon which a new crust rapidly forms. The lesions are likely to be picked and scratched, and moistening of the lips has been thought to be a factor in some cases. Tenderness and a moderate amount of pain, with burning sensations, have been described. The course of the disease is chronic. A neurotic element is frequently present. The major portion of patients have been women.

Treatment.—The disease is rebellious to treatment. Stelwagon recommends a sulphur ointment and a resorcin lotion. *X*-rays have been used successfully in the condition by Morris,[1] ourselves and others. Montgomery[2] successfully treated a case with radium.

Fig. 446



Cheilitis exfoliativa. (Fordyce and MacKee.)

FORDYCE'S DISEASE.

Synonym.—Pseudo-colloid of the lips.

Definition.—This is a chronic disorder limited to the mucous membranes of the lips and oral cavity, characterized by discrete, yellowish or light-colored, milium-like lesions, unaccompanied by subjective sensations.

The first case was described by Fordyce[3] in 1896, at which time, and for a brief time after, it was thought to be rare, but subsequent ob-

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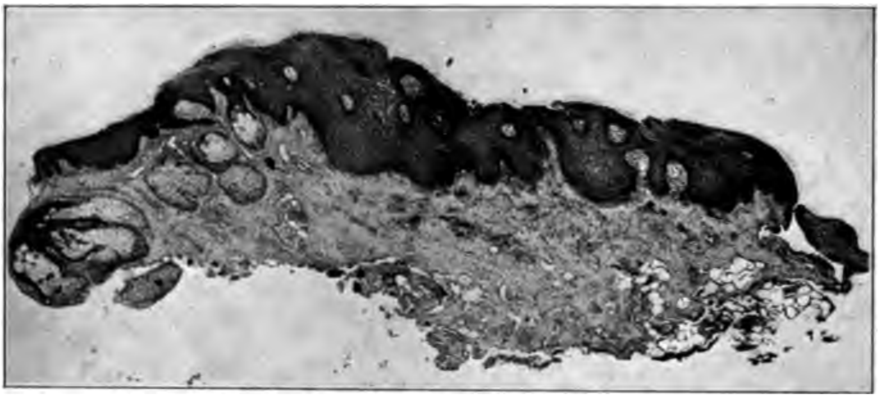
[1] Dis. of the Skin, 1911, p. 618.

[2] Jour. Cut. Dis., 1918, xxxvi, p. 363, cheilitis exfoliativa.

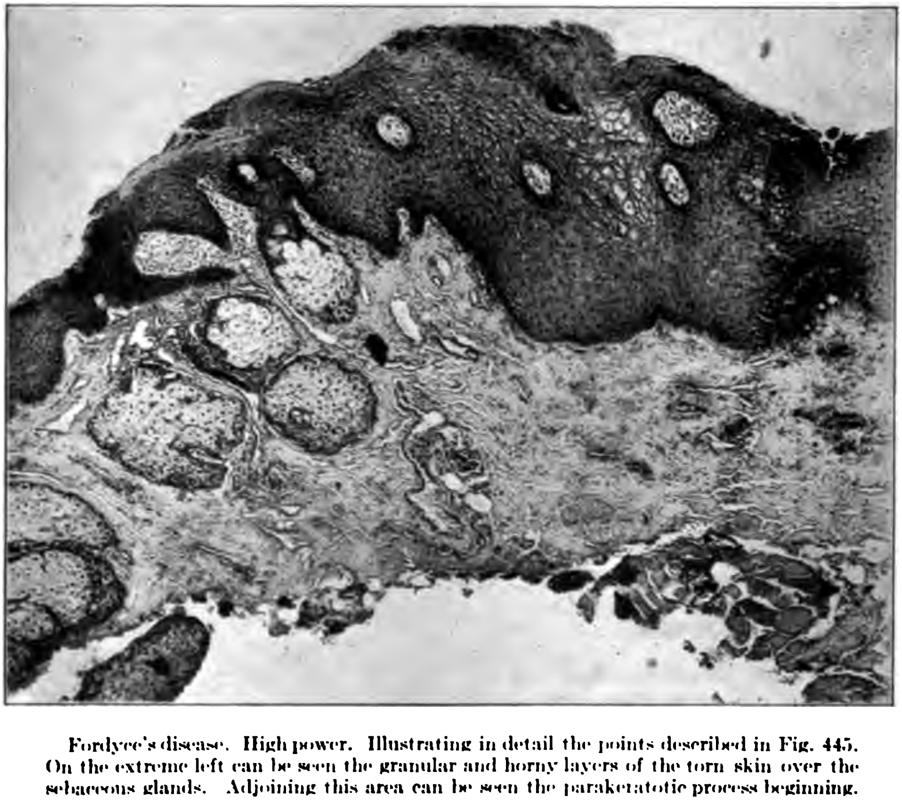
[3] Jour. Cut. Dis., 1896, xiv, p. 413 (A peculiar affection of the mucous membrane of the lips and oral cavity).

FIG. 447



Fordyce's disease. Low power. Showing on the left the sebaceous structures, lying for the most part under the epidermis, covered by torn skin. On the right appears the disease proper, consisting of a greatly hypertrophied epidermis. (C. J. White.)

FIG. 448



Fordyce's disease. High power. Illustrating in detail the points described in Fig. 445. On the extreme left can be seen the granular and horny layers of the torn skin over the sebaceous glands. Adjoining this area can be seen the parakeratotic process beginning. Farther toward the right can be seen the hypertrophied epidermis; the somewhat abnormal palisade layer with its infiltrating cells; the edematous, poorly staining rete-cells; the highly swollen reticulated cells; the superficial parakeratotic cells; and lastly, the widely dilated lymph-vessels and blood-vessels of the corium. (C. J. White.)

servation has demonstrated that it is relatively common. It is more frequently detected on examination for other lesions, as patients rarely apply for its relief, owing to the absence of any subjective sensations.

Symptoms.—The lesions are situated most frequently on the upper lip, lower lip, and on the oral mucous membrane extending along the line of the teeth as far as the last molar. They may be few or abundant, at times forming a plaque or band by aggregation of individual lesions. They vary in color from a yellowish hue on the lips to a whitish shade inside the mouth. They are primarily maculo-papules, and are best seen by putting the mucous membrane on the stretch.

Etiology and Pathology.—C. J. White,[1] in 70 per cent. of 65 cases, found these lesions in association with other disorders of the sebaceous glands, such as acne, rosacea, seborrhoic dermatitis, and alopecia furfuracea. The same proportion of these patients suffered from dyspepsia. The disease develops more frequently in males than in females, and most commonly between the ages of twenty and forty years, though it occurs both before and after these periods. Fordyce originally attributed the condition to a granular degeneration of the rete-cells. White confirmed this. Other observers found hypertrophy of the sebaceous glands a conspicuous feature.

Treatment is usually not required, and when instituted is of little avail.

Prognosis.—The disease is persistent, though benign in nature and productive of little discomfort or inconvenience. In neurotic individuals the lesions may be a source of anxiety until their benign nature is explained.[2]

PERLÈCHE.[3]

Synonyms.—Bridou, Labialitis, Poissonade, Niarde.

Definition.—A superficial, inflammatory, contagious disease of the lips, occurring usually in children.

The disease was first described in 1885 by Lemaistre and later by several French and other observers. Attention has been called to it recently in America through reports of many cases by J. E. Lane and W. L. Smith.

Symptoms.—The disorder begins at the labial commissure, is usually symmetrical and spreads toward the center of the lip. It frequently is limited to the commissure but may extend for a short distance over the skin and on the mucous membrane inside the lips. The disease is characterized by patches of whitened epithelium which appear macerated and are easily detached. The French describe a whitish pellicle which becomes wrinkled, producing a fissured appearance. According to Lane's observations, at the beginning of the disease the epithelium is smooth and whitish, with a mother of pearl tinge. It later becomes macerated, slightly thickened, loses this mother of pearl color, and small transverse fissures appear which show a reddened base when the lip is stretched. The fissures are superficial and do not bleed. Occasionally some of the pellicle can be removed, but more often there is some hypertrophy.

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[1] Jour. Cut. Dis., 1905, xxiii, p. 97: Fordyce's Disease. (A review of the literature, with discussion and report of a clinical study of sixty-five cases, with histopathology of one case.)

[2] Pringle, Brit. Jour. Derm., 1914, xxvi, p. 11.

[3] Lemaistre, Jour. Soc. med. et de la Haute-Vienne, Limoges, 1886, pp. 41–55–74. Raymond, Annales, 1893, p. 578. Planche, Thèse de Paris, 1897. Lane, J. E., Trans. Sec. on Derm., Amer. Med. Assoc., 1917, p. 137. Smith, W. L., Archiv of Pediat., 1917, xxxiv, p. 274; "Perlèche; its bacteriology, symptoms and treatment in 223 cases."

After the lesion heals a whitened appearance may remain for some time. The subjective symptoms are mild, indicated by slight burning sensations and a feeling of dryness which causes the child frequently to moisten the area with the tongue, hence the name. Deeper, painful fissures are described. In some instances a diphtheroid or impetiginous stomatitis with impetiginous lesions on the cutaneous surface also have been described in association with the disorder. As a rule, the disease runs its course in about four weeks, but relapses or recurrences through reinfection are common.

Etiology.—The disease occurs in children, often in epidemic form, is contagious and transmitted directly or by mediate objects, such as drinking cups, towels, pencils and other articles used in common. Epidemics in schools and asylums for infants and children are recorded. Lemaistre isolated an organism which he termed *Streptococcus plicatilis*, to which he attributed the disorder. Raymond, Planche, and others attributed the disorder to staphylococci, chiefly *Staphylococcus cereus albus*. Colcott Fox and Sabouraud classed the disease with impetigo and other local streptococcic infections. Lane constantly found streptococci and usually other bacteria. W. L. Smith isolated an anaërobic streptococcus in pure culture in 135 out of 223 cases. He believed the infection occurred secondary to abnormal conditions in the mouth increasing the salivary secretions.

Diagnosis.—The disease is to be chiefly differentiated from syphilis. The several French observers, including Fournier, say that the resemblance of the disease to syphilis is so close that only the absence of other signs of syphilis can determine the matter. The age of the patient, the limitation of the disease to the commissures, its contagious character and short course are all valuable aids. In addition, a microscopic examination for *Spirochetæ pallida* and the complement-fixation test may be employed.

Treatment.—To prevent the disease spreading to other people care should be exercised with the utensils, drinking cups, towels and other articles used by the patient. The local lesions readily respond to mild applications of silver nitrate, 2 to 10 per cent., used daily, or mild solutions of alum or sulphate of copper.

APHTHOUS STOMATITIS.

Definition.—This disorder is characterized by the occurrence of vesicles, discrete or grouped, on the mucous membrane of the mouth in children and usually runs a short course.

Symptoms.—Not infrequently moderate and indefinite general symptoms occur with the eruption, indicated by general indisposition, slight elevation of temperature and loss of appetite. The local lesions consist of vesicles which very soon rupture, leaving a shallow, superficial, circular erosion. The lesions usually occur in successive crops. They are commonly found at the border of the tongue and inside of the lips, although any part of the mucous membrane of the mouth and pharynx may be involved. They may be few and isolated or sufficiently numerous practically to cover the mucous membrane of the mouth. Large patches are not infrequently formed by the coalescence of individual lesions. The early lesions are yellowish in color and later are apt to become dirty gray, and occasionally resemble the diphtheritic membrane. An areola may or may not be visible. The subjective symptom is pain, which is often sufficient to interfere with the taking of food. The disease commonly lasts for one or two weeks and disappears spontaneously. Relapses are common.

Treatment.—The local treatment consists in employing mild alkaline mouth washes and the individual lesions may be treated with mild solutions of silver nitrate or with powdered alum.

VINCENT'S DISEASE.[1]

In 1919, Foerster[2] suggested that with the return of the troops from abroad Vincent's disease of the mouth would become of importance. The so-called "Trench mouth," a disorder with which a large number of troops were affected, was demonstrated to be a variety of Vincent's disease. In addition to the deep ulceration which occurs on the tonsil, ulcerations may occur on the ramus of the lower jaw posterior to the last molar tooth, or the disease may be exhibited as a general infection involving most of the mucosa of the pharynx and at times the tongue, or it may remain limited to the gums, where it strongly suggests pyorrhea alveolaris. Mild cases, which may become chronic, occur as tonsillitis or gingivitis, or shred-like patches may occur on the cheeks and lips. These mild manifestations may be succeeded by severe exacerbations, with the production of sloughing serpiginous ulcers covered with a heavy pultaceous and often adherent false membrane. The ulceration may be superficial or deep and destructive. It may spread with rapidity and involve large areas of the mucosa. In these instances the glands are swollen and a moderate or severe systemic reaction is present. The spirillum of Vincent and the associated fusiform bacillus are recognized as causative factors and are readily detected in smears. Examination with the dark field illumination is the best procedure. The resemblance of this disorder to syphilis is sometimes striking, and it occasionally is mistaken for diphtheria.

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[1] BIBLIOGRAPHY: Bowman, Prac. Soc. Med., 1916, ix (Med. Sect.), p. 51. Infectious stomatitis among troops, and Lancet, October 6, 1917, Vincent's infection involving mouth, eyes and penis.

[2] Jour. Amer. Med. Assoc., lxxiii, p. 653; "Dermatology and associated disorders of the mucous membranes."

Treatment.—Bowman found the following formula valuable as a mouth wash and for local application.

℞—Vin. ipecacuanhæ,	℥ss;	15
Glycerin,	ʒj;	4
Liquor arsenicalis,	℥ss;	15

Arsphenamin and hydrogen peroxid are useful local applications.

PERIADENITIS MUCOSA NECROTICA RECURRENS (SUTTON).

Synonym.—Ulcus neuroticum mucosæ oris (chronische apthen).—Löblowitz[1] and Sutton[2] independently described a similar disorder. The disorder begins in early childhood and may occur in several members of a family. It is characterized by necrotic or ulcerative lesions occurring on the buccal and laryngeal mucosa. In Sutton's patient the disorder had been present since infancy and was described by him as follows: The early lesion was a red nodule (occasionally two or three) varying in size from a pinhead to that of an ordinary lead pencil, which is smooth, hard, resistant to the touch, and painful. In the course of three or four days sloughing occurs, and a solid mummified-like plug separates leaving a crateriform depression which extends well down into the corium. There is no evidence of secondary infection. With the development of these lesions the regional lymphatic glands become enlarged and tender. The lesions heal within six or eight days, leaving smooth, pliable, grayish scars, with irregular outline, from 0.5 to 1 cm., or more, in diameter. The lesions are usually found on the mucous surface of the lips and cheeks. No etiological factor was determined, the disease, however, being exaggerated in the spring and autumn.

Löblowitz described the disorder as occurring in several members of one family. The early nodule in these was painless, but the subsequent ulcers, some of which became as large as a twenty-five cent piece, were extremely painful. In these the slough separated in two to five days, leaving a temporary scar the entire process extending over a period of from one to three weeks. He emphasized the neurotic element in the cases and its apparent familial tendency. According to Sutton, the histological findings consist in a superficial necrosis with marked inflammatory manifestations in the underlying structures. The connective tissue is densely infiltrated with cells of various types, chiefly plasma cells, fibroblastic cells, mononuclear eosinophiles and polyblasts. No bacteria have been discovered. The disease pathologically is characterized by an intense inflammatory process in the periglandular tissues with ensuing necrosis and suppuration of the central part of the area.

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[1] Archiv, 1910, cii, p. 191.

[2] Jour. Cut. Dis., 1911, xxix, p. 65.

ATROPHY OF THE MUCOUS MEMBRANES OF THE TONGUE AND MOUTH.[1]

This report is founded on the observation of two cases presenting atrophic areas on the mucous membrane of the mouth. The first occurred in a woman, aged thirty-four years, with a negative history. The lesion to be described had been present for three months. It began as a small, superficial white patch, accompanied by no subjective disturbances. The lesion was situated on the dorsal surface of the tongue opposite the second molar about 1.5 cm. from the left lateral edge, and was a roughly oval, sharply circumscribed patch 10 mm. in length by 4 in width. It was glistening, white, smooth, devoid of papillæ and resembled a scar. Later a similar lesion developed anteriorly to this, and at a still later date junction occurred, with the formation of a larger but similar patch.

In the second case a similar lesion was seen on the buccal mucous membrane opposite the second lower molar. No etiological factors could be determined.

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[1] Hazen, Jour. Cut. Dis., 1916, xxxiv, p. 801; "A hitherto undescribed superficial atrophy of the mucous membranes of the tongue and mouth."

INDEX.

A

- ABRASIONS (Excoriations), 57
- Absägen (Ainhum), 532
- Abscess, multiple skin, of infants, 222-223
- Absorption by skin, 51
- Acanthia lectularia (Cimex lectularius), 939
- Acanthoma adenoides cysticum (Multiple benign cystic epithelioma), 614
- Acanthosis, 69
  - bullosa, 390
  - nigricans, 463-466
    - diagnosis of, 465
    - etiology of, 464
    - histology of, 465
    - pathology of, 465
    - prognosis of, 466
    - symptoms of, 463
    - treatment of, 466
- Acare des follicules, 964
- Acarodermatitis urticarioides (Grain itch), 940
- Acarophobia, 856
- Acarus folliculorum, 964
  - scabiei, 59, 947, 952
- Acetanilid eruptions, 176
- Acetylsalicylic acid eruptions, 176
- Achorion gypseum, 861
  - quinckeanum, 859, 861
  - schönleinii, 857, 861, 1082
- Achroma, 544
  - congenital, 543
  - unguium, 1088
- Acid, boric, 103
  - carbolic. See Phenol.
  - chromic, 10[illegible]
  - nitric, 10[illegible]
  - pyrogallic, 10[illegible]
  - salicylic, 10[illegible]
  - trichloracetic, 10[illegible]
- Acne, 1019-1039
  - aggregata, 102[illegible]
  - agminata, 687
  - albida, 100[illegible]
  - artificialis, 10[illegible]
  - atrophica, 1022, 1037
  - bacteriology of, 1021
  - cachecticorum, 687, 691, 1021
  - cancroïdale, 610
  - conglobata, 1022
  - diagnosis of, 1026
  - etiology of, 1023
  - frontalis, 1037
  - indurata, 1020
  - keloid, 555, 1084
  - keratosa, 205, 1036
  - necrotica, 1037
  - papulosa, 1020
  - pathology of, 1025
  - prognosis of, 1030
  - punctata, 1019
  - pustulosa, 1020
  - rodens, 1037
  - rosacea, 1030-1036
    - diagnosis of, 234, 1033
    - etiology of, 1032
    - false, in children, 986
    - pathology of, 1033
    - prognosis of, 1036
    - symptoms of, 1030
    - treatment of, 1034
  - scrofulosorum, 687, 691, 1021
  - sebacea, 988
  - symptoms of, 1019
  - tar, 1028
  - treatment of, 1026
    - local, 1027
    - vaccines in, 1030
    - x-rays in, 1029
  - urticata, 144, 203, 1037
  - varioliformis, 687, 1037-1039
    - diagnosis of, 1039
    - etiology of, 1038
    - pathology of, 1038
    - symptoms of, 1037
    - treatment of, 1039
  - vulgaris, 1019
- Acné. Acne, 1019
  - chéloïdique, 10[illegible]
  - comédon, 101[illegible]
  - cornée, 142
    - en aire, 142
  - décalvante, 10[illegible]
  - miliaire, 100[illegible]
  - ponctuée, 101[illegible]
  - rosacée, 103[illegible]
  - sébacée fluente, 98[illegible]
  - varioliforme, 591, 1037

- Acneform eruptions, **1036–1039**
- Acnitis, 687, **689**
- Aconite eruptions, 176
- Acrochordon, 562
- Acrodermatitis chronica atrophicans, 522
  - perstans, **339**
    - treatment of, 340
  - pustulosa hiemalis, 690
  - vesiculosa tropica, **343**
- Acrodynia, **403–405**
  - symptoms of, 404
  - treatment of, 405
- Acrodynie, 403
- Acromegaly, **512**
  - symptoms of, 512
- Actinomycose, 912
- Actinomycosis, **912–916**
  - diagnosis of, 912
  - etiology of, 914
  - pathology of, 914
  - prognosis of, 916
  - symptoms of, 912
  - treatment of, 915
- Addison's disease, pigmentation in, 538
  - keloid, 498
- Adenoma sebaceum, **610–613**
  - diagnosis of, 613
  - etiology of, 613
  - pathology of, 613
  - symptoms of, 610
  - treatment of, 613
- Adenomes sébacés, 610
  - cancroïdaux, 610
- Adiposis dolorosa, **571**
- Afghan plague (Oriental sore), 827
- Agchylostomiasis, 962
- Age as etiologic factor, **65**
- Agnails, **1096**
- Ainhum, **532–534**
  - etiology of, 533
  - pathology of, 534
  - prognosis of, 534
  - symptoms of, 533
  - treatment of, 534
- Air as etiologic factor, **62**
  - liquid, therapeutic use of, **113**
- Akne (acne), 1019
- Aktinomykose, 912
- Albinismus, **543–544**
  - complete, 543
  - partial, 543
  - symptoms of, 543
- Albinos, 543
- Albugo, 1099
- Aleppo evil (Oriental sore), 827
- Alibert's keloid, 551
- Alkalies, 90
- Alopecia, **1057–1083**
  - adnata, 1058
  - areata, 1069–1080
    - congenital, 1058
    - diagnosis of, 1076
    - etiology of, 1073
    - malignant, 1072
    - pathology of, 1076
    - prognosis of, 1079
- Alopecia areata, symptoms of, 1069
    - treatment of, 1077
  - cicatrisata, 1080–1083
    - diagnosis of, 1082
    - etiology of, 1081
    - pathology of, 1082
    - prognosis of, 1083
    - symptoms of, 1080
    - treatment of, 1082
  - circumscripta, 1069
    - seu orbicularis, 1080
  - congenita, 1058–1060
    - etiology of, 1060
    - pathology of, 1060
    - prognosis of, 1060
    - treatment of, 1060
  - etiology of, 1064
  - furfuracea, 1063
  - orbicularis, 1080
  - pathology of, 1063
  - pityroides, 1063
  - prematura, 1060–1063
    - idiopathica, 1061
    - symptomatica, 1062
  - prognosis of, 1068
  - senilis, 1064
  - treatment of, 1065
  - ungualis, 1099
- Alopécie, 1057
  - cicatricielle, 1080
  - congénitale, 1058
- Alphos (Psoriasis), 249
- Alpine scurvy (Pellagra), 393
- Amblyomma americanus, 946
- Ambrine, 171
- Analgesics, 90
- Anaphylaxis, 63
- Anaplasma liberum, 377
- Anatomical tubercle (Verruca necrogenica), 665
- Anatomy of skin, **17–47**
- Anderson's powder, 148
- Anemia, 68
  - dirt-eater's, 962
  - mountain, 962
- Anesthesia, cutaneous, 855
- Angio-elephantiasis, 577
- Angiokeratoma, **460–462**
  - diagnosis of, 462
  - etiology of, 461
  - pathology of, 462
  - prognosis of, 462
  - symptoms of, 461
  - treatment of, 462
- Angiokératome, 460
- Angiolipoma, 577
- Angiolymphangioma, 577
- Angioma, **575–584**
  - cavernous, 578
  - elephantiasis, 575
  - infective, 584
  - pigmentosum et atrophicum, 619
  - serpiginosum, **584–587**
    - diagnosis of, 587
    - etiology of, 585
    - pathology of, 585

- Angioma serpiginosum, prognosis of, 587
    - treatment of, 587
  - simplex, 576
    - hyperplasticum, 576
- Angiome, 575
  - cystique, 591
- Angiomyoma, 573
- Angioneurotic edema, **153–156**
    - diagnosis of, 155
    - etiology of, 154
    - pathology of, 155
    - prognosis of, 156
    - symptoms of, 154
    - treatment of, 155
- Anhidrose, 973
- Anhidrosis, 973
- Anidrosis, **973–974**
  - symptoms of, 973
  - treatment of, 974
- Animal extracts, 90
  - parasites, **932–968**
  - poisons, 324
- Ankylostoma duodenale, 962
- Ankylostomiasis, 962
- Annulus migrans, 1117
- Anonychia, **1112**
- Anthemata, 59
- Anthrax, **325–328**
  - diagnosis of, 327
  - etiology of, 326
  - pathological anatomy of, 326
  - pathology of, 326
  - prognosis of, 328
  - simplex, 319
  - symptoms of, 325
  - treatment of, 327
    - serum therapy in, 327
- Antimony, 89
- Antipyrin eruptions, 176
- Anus, eczema of, 226
  - pruritus of, 842
- Apes mellificæ, 947
- Aphthen, chronische, 1128
- Aplasie moniliforme intermittente, 1049
- Appendages, disorders of, **969–1112**
- Area Celsi, 1069
  - Johnstoni, 1069
- Areola, 54
- Argyria, **541**
  - localized, 542
- Arnica dermatitis, 167
- Arrectores pilorum, 34
    - development of, 48
    - heat regulation by, 50
- Arsenic and compounds, 88, 100
    - chloasma from, 177, 538
- Arsphenamin, 88
  - eruptions, 178
  - reactions, 777
  - in syphilis, **772–779**
  - untoward effects of, 777
- Arzneiexantheme (Dermatitis medicamentosa), 174
- Asiatic pill, 88
- Asphyxia, local, 349
- Astéatose, 1008
- Asteatosis, **1008–1009**
  - prognosis of, 1009
  - symptoms of, 1008
  - treatment of, 1009
- Atheroma (Steatoma), 1012
- Athrepsie (Sclerema neonatorum), 504, 505
- Atophan eruption, 178
- Atoxyl, 88
- Atrichia, universal congenital, 1058
- Atrophia, **519–534**
  - congenital 525
  - diffuse idiopathic, **520–523**
    - diagnosis of, 522
    - etiology of, 522
    - pathology of, 522
    - prognosis of, 523
    - treatment of, 523
  - maculosa et striata, **523–525**
    - etiology of, 525
    - idiopathic, 524
    - pathology of, 525
    - prognosis of, 525
    - symptomatic, 523
    - treatment of, 525
  - neuritica, 527
  - pigmentosum, 619
  - pilorum propria, **1045–1050**
    - forms of, 1045
  - senilis, **519–520**
    - symptoms of, 519
    - treatment of, 520
  - unguium, **1097–1103**
- Atrophic spots, 523
- Atrophoderma. See Atrophia.
- Atrophy. See Atrophia.
- Aussatz (Lepra), 781
- Autoserum treatment, 94

B

- Bacillus, acnes, 1025
  - anthracis, 326
  - Demme's, 136
  - diphtheriæ, 350
  - lepræ, 782, 792
  - Lustgarten's, 756
  - mallei, 328, 330
  - multiformis, 1047
  - pellagræ, 400
  - prodigiosus, 1053
- Bacteria in skin, **68**
- Bacterium fetidum, 975
  - maydis, 400
- Bad disorder (Syphilis), 709
- Balanitis, erosive and gangrenous, 345
- Baldness (Alopecia), 1057
- Balggeschwulst (Steatoma), 1012
- Balloon cell, 70
- Banko-kerende (Ainhum), 532
- Barbadoes leg (Elephantiasis), 507
- Barber's itch, 311, 878
- Barcoo rot of Queensland (Veldt sore), 314
- Barkoo (Veldt sore), 314

Barley itch (Grain itch), 940
Bartfinne (Sycosis, coccogenous), 878, 1087
Bartflechte (Sycosis, coccogenous), 1087
Basal layer of epidermis, 26
Bassorin paste, 243
Bath pruritus, 844
Baths, **95**
  alkaline, 95
  antiseptic, 95
  cold, 95
  continuous, 95
  etiologic influence of, 62
  hot, 95
  spring water, 95
  steam, 95
  sulphur, 95
  tar, 95
  vapor, 95
Bearded region, coccogenous sycosis of, 1087
    eczema of, 225
    piedra nostras of, 1061
    ringworm of, 878
    trichophytosis of, 875
Beau's lines, 1100
Bed-bugs (Cimex lectularius), 939
Bee stings, 947
Beigel's disease, 1052
Belladonna eruptions, 178, 409
Bettwanze (Cimex lectularius), 939
Beulenkrankheit, endemische, 827
Biers' hyperemic treatment, 104
Birthmark (Nevus vasculosus), 576
Biskra button (Oriental sore), 827
Bites as etiologic factor, 63
Black fever, 432
  head, 1014
  tongue, 1119
Bläschen (Vesiculæ), 55
Bläschenflechte (Herpes simplex), 351
Blasen (Bullæ), 56
Blasenausschlag (Pemphigus), 370
Blastomycetic dermatitis (Blastomycosis), 916
Blastomycosis, **916–927**
  diagnosis of, 926
  etiology of, 921
  pathology of, 923
  prognosis of, 927
  symptoms of, 917
  treatment of, 927
Blattern (Variola), 415
Blebs (Bullæ), 56
Bleeding stigmata, 979
Blisters (Bullæ), 56
Bloodvessels of skin, 28
  of nail region, 43
Blue disease, 432
  pigmentation, **542–543**
    treatment of, 543
Blushing, 139
Blutgeschwür (Furunculus), 315
Blutschwär (Carbunculus), 319
Boba (Frambesia), 830
Bockhart's impetigo, 305
Boil (Furunculus), 315
  Delhi, 827
Boric acid, 103
Borken (Crustæ), 57
Bot-fly, 965
Botryomycose humaine, 587
Botryomycosis hominis (Granuloma pyogenicum), 587
Bouba (Frambesia), 830
Bouton d'Amboine (Frambesia), 830
  d'Orient (Oriental sore), 827
Bowditch-Island ringworm (Tinea imbricata), 898
Brandschwär, 319
Brandy nose (Acne rosacea), 1030
Breast, cancer of, 625
  eczema of, 226
  Paget's disease of, **650–653**
Bridou (Perlèche), 1125
Brocq's dermatitis exfoliativa, 187, **189**
Bromhidrosis, 974
Bromidrose, 974
Bromidrosis, **974–976**
  etiology of, 975
  pathology of, 975
  symptoms of, 974
  treatment of, 975
Bromin eruptions, 178
Bronze diabetes, 538
Brown-tail moth dermatitis, **943–945**
    etiology of, 944
    pathology of, 944
    symptoms of, 944
    treatment of, 945
Bucnemia tropica (Elephantiasis), 507
Bulb corpuscles, 33
Bullæ, **56**
  hæmorrhagicæ, 435
  pathology of, 70
Bulles (Bullæ), 56
Burmese ringworm (Tinea imbricata), 898
Burns, **170–172**

C

CACHEXIA strumipriva, 512
  thyroidea, 512
Cachexie pachydermique (Myxedema), 512
Cacotrophia folliculorum, 440
Calamine liniments, 96
  lotion, 170
Calcification of skin, **609–610**
Callositas, **466–467**
  pathology of, 466
  symptoms of, 466
  treatment of, 467
Callosity, 466
Calomel, 103
Calvities (Alopecia), 1057
  premature, 1063
Calx sulphurata, 89
Camphorated oil tumors, 566
Cancer. See Carcinoma.
  en cuirasse, 625

Cancroid ulcer (Rodent ulcer), 631
Cancroïde, 592, 665
Cancrum oris in measles, 411
Canities, **1055–1057**
  etiology of, 1056
  pathology of, 1057
  symptoms of, 1055
  treatment of, 1057
  unguium, 1099
Canker rash (Scarlatina), 405
Cannabis indica eruptions, 180
Cantharides eruptions, 180
Caraate (Pinta), 907
Caraté (Pinta), 907
Carateus (Pinta), 907
Carbolic acid. See Phenol.
Carbon-dioxid, 113
  monoxid poisoning, gangrene following, 346
Carbuncle (Carbunculus), 319
Carbunculus, **319–322**
  diagnosis of, 321
  etiology of, 320
  pathology of, 320
  prognosis of, 322
  splenic fever, 325
  symptoms of, 319
  treatment of, 321
Carcinoma of breast, 625
  en cuirasse, 625
  epitheliale. See Epithelioma.
  lenticulare, **625–627**
  melanotic, 627–629
  pigmented, 627
  precancerous dermatoses, **653**
  of skin, **525–650**
    etiology of, 638–644
    pathology of, 628, 644–646
    prognosis of, 629, 650
    treatment of, 629, 646–650
      cauterization in, 649
      radiotherapy in, 647
  tuberosum, 627
Carlsprudel salts, 90
Carrion's disease (Verruga peruana), 834
Cascadöe (Tinea imbricata), 898
Cataphoresis, electric, 104
Cathartics, 90
Cativi (Pinta), 907
Causalgia, **854–855**
Caustics, 100
Cellulitis, 323
  crab, 336
Cellulome épithélial éruptif (Syringocystoma), 617
Chalazion, 1013
Chalazodermia (Dermatolysis), 515
Chancre, 711
  diagnosis of, 714
  extragenital, 714
  Hunterian, 711
  indurated, 711
  redux, 714
  relapsing, 714
  du Sahara, 827
Charbon (Anthrax), 325
Chaulmoogra oil, 795
Cheilitis exfoliativa, **1122–1123**
    treatment of, 1123
  glandularis, **1121–1122**
    apostematosa, 1121
    etiology of, 1122
    pathology of, 1122
    prognosis of, 1122
    treatment of, 1122
Cheiropodalgia, 403
Cheiropompholyx (Pompholyx), 340
Cheloid (Keloid), 551
Chéloïde (Keloid), 551
Chicken-pox (Varicella), 426
Chigger (Pulex penetrans), 957
Chignon fungus, **1052**
Chigoe (Pulex penetrans), 957
Chilblains (Erythema pernio), 117
  necrotizing, 687
Chinese ringworm (Tinea imbricata), 898
Chironomidæ, 947
Chiropodalgia (Acrodynia), 403
Chloasma, **537–541**
  in Addison's disease, 538
  diagnosis of, 539
  etiology of, 539
  in Graves' disease, 538
  idiopathicum, 537
  pathology of, 539
  prognosis of, 541
  symptomaticum, 537
  treatment of, 539
  uterinum, 537
Chloral eruptions, 180
Chloralamid eruptions, 180
Chloroform eruptions, 180
Chlorosis, tropical, 962
Chondrodermatitis nodularis chronica helicis, **568–569**
Chorionitis (Scleroderma), 495
Chromatophores, 36
Chromic acid, 100
Chromidrose (Chromidrosis), 976
Chromidrosis, **976–978**
  etiology of, 977
  pathology of, 977
  symptoms of, 976
  treatment of, 978
Chromophytosis (Tinea versicolor), 900
Chrysarobin, 101
Cicatrice (Cicatrix), 58, 549
Cicatrix, 58, **549–551**
  diagnosis of, 551
  etiology of, 550
  hypertrophic, 553
  pathology of, 550
  symptoms of, 549
  treatment of, 551
Cimex lectularius, **939–940**
    diagnosis of, 940
    treatment of, 940
Cimicidæ, 939
Cinchona eruptions, 184
Cladothrix, 336
Classification of skin diseases, **114**
Clastothrix, 1046

- Clavus, **467–468**
  - histology of, 467
  - symptoms of, 467
  - treatment of, 468
- Climate, etiologic influence of, 64
- Climatobacterium granulomatis, 840
- Clothing, etiologic influence of, 63
- Clou (Furunculus), 315
  - de biskra, 827
- Coccidoidal infection, **928**
- Cochin leg (Elephantiasis), 507
- Cod-liver oil, 89
- Coil ducts, 45
    - cysts of, 983
  - glands, **45–47**
    - fetal development of, 48
    - secretory function of, 50
- Cold sores, 351
- Coley's fluid, 819
- Collagen, 21
- Collodion, 99
- Colloid degeneration of skin, 73, **608–609**
    - diagnosis of, 609
    - etiology of, 609
    - pathology of, 609
    - symptoms of, 608
    - treatment of, 609
- Colloïdome miliaire (Colloid degeneration), 608
- Color of skin, 18, 27, 35. See also Pigment.
- Columnæ adiposæ, 20
- Comedo, **1014–1019**
  - diagnosis of, 1018
  - etiology of, 1016
  - pathology of, 1016
  - prognosis of, 1019
  - symptoms of, 1014
  - treatment of, 1018
- Comedone, 55
- Condurango eruptions, 180
- Condyloma, 729
  - acuminatum, 475
  - diagnosis of, 386
- Condylomatosis (Pemphigus vegetans), 383
- Consomption dartreuse, 1103
- Copaiba, 89
  - eruptions, 180
- Copper-nose (Acne rosacea), 1030
- Copra itch, **943**
- Cor, 467
- Corium, anatomy of, **21–25**
  - degeneration of, 73
  - development of, 47
  - giant cells of, 72
  - histology of, 71
  - mast cells of, 72
  - pathological cells of, 71
  - plasma cells of, 71
- Corn (Clavus), 467
- Corne de la peau (Cornu cutaneum), 468
- Cornification of epidermis, 28
- Cornu cutaneum, **468–470**
    - etiology of, 469
    - pathology of, 470
    - prognosis of, 470
    - symptoms of, 469
    - treatment of, 470
  - humanum, 468
- Corpuscles, Krause's, 33
  - Meissner's, 32
  - Merkel's, 33
  - Pacinian, 18, 30
  - tactile, 32
  - Vater's, 30
  - Wagner's, 32
- Couperose (Acne rosacea), 1030
- Cow-pox (Vaccinia), 429
- Crab cellulitis (Erysipeloid), 336
- Crab-louse (Pediculosis pubis), 595
- Cracks (Rhagades), 58
- Crateriform ulcer, 636
- Craw-craw, **967–968**
  - etiology of, 968
  - prognosis of, 968
  - treatment of, 968
- Creeping eruption, **966–967**
    - diagnosis of, 966
    - etiology of, 967
    - symptoms of, 966
    - treatment of, 967
- Crenation degeneration, 73
- Creosote, 89
- Croûtes (Crustæ), 57
- Crusta lactea, 990
- Crustæ, 57
- Crusts (Crustæ), 57
- Cubebs eruption, 180
- Culicidæ, 947
- Cute (Pinta), 907
- Cuticle (Epidermis), 25
- Cutis anserina, 35
  - hyperelastica Unna, 517
  - pendula, 515
  - vera, 21
  - verticis gyrata, **517–518**
- Cyanidrosis, 976
- Cyclops quadricornis, 959
- Cystadénome épithelial bénin (Syringocystoma), 617
- Cysticercus cellulosæ cutis, **960–961**
- Cytoplasma oviforme, 376
- Cytorrhyctes luis, 756
  - vaccinæ, 422
  - variolæ, 422

D

- Dactylolysis spontanea (Ainhum), 532
- Dandruff (Seborrhea), 988
- Darier's disease (Keratosis follicularis), 446
- Dartre humide, 305
- Deciduous skin, 122
- Décollement des ongles, 1098
- Defluvium capillorum, 1057
  - unguium, 1099
- Degeneration, colloid, 73, **608**
  - of corium, 73
  - crenation, 73

- Degeneration, edematous, 73
  - epithelial, 70
  - fatty, 73
  - hyaline, 73
  - mucoid, 73
  - myxomatous, 73
- Delhi boil, 827
- Demme's bacillus, 136
- Demodex in comedo, 1016
  - folliculorum, **964–965**
- Depilatories, 1044
- Dercum's disease, 571
- Derma, 21
- Dermacentor occidentalis, 433
- Dermamyiasis linearis migrans œstrosa (Creeping eruption), 966
- Dermatagra (Pellagra), 393
- Dermatalgia, **853–855**
  - diagnosis of, 854
  - treatment of, 854
- Dermatite (Dermatitis), 161
  - exfoliatrice, 186
  - polymorphe douleureux, 362
- Dermatitis, **161–208**
  - artificial, 200–203
  - blastomycetic, 916
  - blastomycotica, 916
  - brown-tail moth, **943–945**
  - calorica, **170–172**, 331
    - symptoms of, 170
    - treatment of, 171
  - congelationis, **172**
    - treatment of, 173
  - contusiformis, 135
  - exfoliativa, **186–197**
    - classification of, 187
    - diagnosis of, 286, 383
    - epidemica, 188, **198–200**
      - etiology of, 199
      - pathology of, 199
      - prognosis of, 200
      - treatment of, 200
    - neonatorum, 188, **196**
      - diagnosis of, 197
      - etiology of, 197
      - pathology of, 197
      - prognosis of, 197
      - treatment of, 197
    - universal, of children, **197**
    - Wilson type of, 187, **189**
      - diagnosis of, 191
      - etiology of, 190
      - pathology of, 190
      - prognosis of, 191
      - treatment of, 191
  - factitia, 200–203
  - feigned, 200–203, 234
  - gangrænosa, **344–348**
    - infantum, 347–348
      - diagnosis of, 348
      - etiology of, 348
      - pathology of, 348
      - prognosis of, 346
      - treatment of, 346
  - herpetiformis, **361–368**
    - bullous type of, 364

- Dermatitis herpetiformis in children, 366
    - diagnosis of, 367, 378
    - erythematous type of, 364
    - etiology of, 366
    - nail changes in, 1109
    - papular type of, 365
    - pathology of, 366
    - prognosis of, 368
    - pustular type of, 365
    - symptoms of, 362
    - treatment of, 637
    - vesicular type of, 364
  - infectious eczematoid, 218, 222, **247–248**
    - diagnosis of, 248
    - etiology of, 248
    - symptoms of, 247
    - treatment of, 248
  - lycopersicum, 166
  - malignant papillary, 650
  - match-box, 167
  - medicamentosa, **174–186**
    - diagnosis of, 175
    - etiology of, 174
    - list of drugs producing, 176–186
    - symptoms of, 174
    - treatment of, 176
  - micropapulosa erythematosa hyperidrotica nasi, 986
  - nodosa, 968
  - papillaris capillitii, **1084–1087**
    - etiology of, 1086
    - pathology of, 1086
    - prognosis of, 1087
    - treatment of, 1087
  - peculiar inflammatory, of the nose of the young, with sweating, 986
  - poison ivy, 166–170
    - diagnosis of, 168
    - treatment of, 170
  - primrose, 165
    - treatment of, 264
  - primula, 261
  - psoriasiformis nodularis, 270
  - repens, **337–339**
  - rhus, 166–170
  - scarlatiniformis recidivans, 120
  - seborrhoica, 257, **997–1008**
    - diagnosis of, 264, 278, 1007
    - etiology of, 1006
    - pathology of, 1006
    - symptoms of, 999
    - treatment of, 1008
  - traumatica, 163
  - uncinarial, 962
  - universal, of children at breast, 197
  - vegetans, **314–315**
  - venenata, **163–169**
    - course of, 165
    - diagnosis of, 168
    - etiology of, 166
    - prognosis of, 169
    - symptoms of, 163, 166
    - treatment of, 169
  - *x*-ray, 205

Dermatolysis, **515–517**
Dermatomycosis furfuracea, 900
Dermatomyoma (Myoma), 523
Dermatoneuroses, hysterical (Dermatitis factitia), 200
Dermatophilus penetrans, 957
Dermatosclerosis (Scleroderma), 495
Dermatoses affecting nails, 1105
  occupational, 63
  precancerous, **653–654**
Dermatosis Kaposi (Xeroderma pigmentosum), 619
Dermite (Dermatitis), 161
Dermographism, 143
Desmoids, 563
Desquamation aberrante en aires de la langue, 1117
  estivale en aire des mains, 344
Development of skin, **47–49**
Dhobie itch, 870
Diabetes, bronze, 538
Diabetic gangrene, 346
  xanthoma, **605**
Diagnosis of skin diseases, **74–85**
Diet, etiologic influence of, 63
Digitalis eruptions, 181
Dinitrochlorbenzol dermatitis, 167
Diphtheria antitoxin eruptions, 184
  of skin, **350**
    treatment of, 351
Diplodia zeæ, 400
Dirt eater's anemia, 962
Dissection tubercle, 665
  wounds, 324
Distoma hepaticum, **961**
Districhiasis, **1044**
Diuretics, 90
Dochmiasis, 962
Dondos, 543
Donovan's solution, 88
Dracontiasis, **958–960**
  diagnosis of, 959
  pathology of, 959
  prognosis of, 960
  symptoms of, 958
  treatment of, 959
Dracunculus medinensis, 958, 959
Dragonneau (Dracontiasis), 958
Dressings, protective, 98
Drugs, eruptions from, 167–168, **174–186**
  list of, causing dermatitis, 176–186
Duhring's disease, 361
  paste, 98
Dupuytren's lines of cleavage, 25
Dysidrose (Pompholyx), 340
Dysidrosis, **340–343**
  diagnosis of, 342
  etiology of, 341
  pathology of, 342
  symptoms of, 340
  treatment of, 343
Dystrophie édémateuse héréditaire, 507
  papillaire et pigmentaire, 463

E

Ears, eczema of, 224
  painful nodular growth of, 568
Ecchymomata, 435
Ecchymoses, 54, 435
Echinococcus, 961
Ecthyma, **311–314**
  diagnosis of, 313
  etiology of, 312
  gangrenous infantile, 347
  pathology of, 312
  symptoms of, 311
  térébrant, 347
  treatment of, 313
Ectothrix, 885
Eczem (Eczema), 212
Eczema, **212–249**
  acute, 222
  ani, 226
  of anus and anal region, 226
  aurium, 224
  barbæ, 225
  of beard, 225
  of breast, 226
  capilliti, 223
  capitis, 223
  in children, 226
  chronic, 223
  crurate, 226
  crurum, 226
  diabeticorum, 221
  diagnosis of, 234–237
  of ears, 224
  erythematosum, 214
  etiology of, 228–232
  exfoliativum, 220
  external causes of, 230
  of extremities. See Eczema of feet, hands, legs.
  of eyelids, 225
  of face, 223
  faciei, 223
  of feet, 226
  fissum, 220
  folliculorum, 215
  of genitalia, 225
  genitalium, 225
  of hands, 226
  impetiginoides, 217
  in infants and children, 226
    treatment of, 246
  intertrigo, 123, 222
  labiorum, 224
  of legs, 226
  lichenoides, 215
  of lips, 224
  madidans, 219
  mammæ, 226
  manuum, 226
  marginatum, 222, **868–873**
  of nails, 1104
  narium, 224
  of nipple, 226
  of nostrils, 224
  nummularis, 221

- Eczema orbiculare, 214
  - palmar, 227
  - palpebrarum, 225
  - papulosum, 215
  - parasiticum, 222
  - pathology of, 232–234
  - pedum, 226
  - plantar, 227
  - prognosis of, 246
  - pustulosum, 217
  - rhagadiforme, 220
  - rubrum, 219
  - of scalp, 223
  - sclerosum, 221
  - seborrheicum, 989, 997
  - squamosum, 220, 989
  - sycosiforme, 218
  - symptoms of, 212
  - topical varieties of, 223–228
  - treatment of, 237–246
    - antipruritics in, 239
    - diet in, 238
    - glycogelatins in, 243
    - internal medication in, 238
    - liniments in, 240
    - local, 239
    - lotions in, 239–241
    - ointments in, 239, 241–242
    - pastes in, 242
    - radiotherapy in, 245
    - tar preparations in, 244
  - tuberculous, of nurslings, 221
  - umbilici, 226
  - of umbilicus, 226
  - verrucosum, 221
  - vesiculosum, 215
- Eczème (Eczema), 212
  - craquelé, 221
- Edema, acute circumscribed, 150
    - essential, 153
  - angioneurotic, **153–156**
    - diagnosis of, 155
    - etiology of, 154
    - pathology of, 155
    - prognosis of, 156
    - symptoms of, 154
    - treatment of, 155
  - arthritic, 155
  - cretinoid, 512
  - of legs, hereditary, **507**
    - diagnosis of, 507
    - symptoms of, 507
    - treatment of, 507
  - neonatorum, **505–506**
    - diagnosis of, 506
    - etiology of, 506
    - pathology of, 506
    - prognosis of, 506
    - symptoms of, 505
    - treatment of, 506
  - pathology of, 70
  - peliotic, 155
  - Quincke's, 153
  - solid, of face, **337**
    - diagnosis of, 337
    - etiology of, 337
- Edema, solid, of face, treatment of, 337
  - wandering, 153
- Edematous degeneration, 73
- Egg foot (Mycetoma), 909
- Eiterbeule (Furunculus), 315
- Eiterblase (Ecthyma), 311
- Eiterflechte (Impetigo), 305
- Eitergeschwür (Furunculus), 315
- Ekthyma (Ecthyma), 311
- Elastin of corium, 21
- Electrolysis, **105–106**
- Electrotherapy, **104**
  - high-frequency, 104
- Eleidin, 27, 28
- Elephant foot, 909
  - leg, 507
- Elephantiasis, **507–511**
  - arabum, 507
  - congenital, 511
  - diagnosis of, 511
  - endemic, 510
  - etiology of, 510
  - græcorum, 781
  - lymphangiectatica, 590
  - nevoid, 510
  - nostras, 510
  - pathology of, 511
  - sporadic lymphatic, 510
  - symptoms of, 508
  - telangiectodes, 510, 576
  - treatment of, 511
  - tuberculosa cutis, **667–668**
- Elephantoid fever, 508
- Elliott's bassorin paste, 99
- Embryology of skin, **47–49**
- Endodermophyton, 899
- Endothelioma capitis, **654–655**
    - etiology of, 654
    - pathology of, 654
    - symptoms of, 654
    - treatment of, 655
  - cutis, **654**
- Endothrix, 881, 884
  - trichophyta, 881
  - violaceum, 881
- Engelure (Erythema pernio), 117
- Envies, 1096
- Environment, etiologic influence of, 62
- Ephélide (Lentigo), 535
- Ephelis (Lentigo), 535
- Ephidrosis, 969
- Epidermis, anatomy of, **25–28**
  - atrophy of, 70
  - degeneration of, 70
  - development of, 47
  - histology of, 69
  - hypertrophy of, 69
  - pathologic changes of, 69–71
- Epidermolysis bullosa, **390–393**
    - etiology of, 393
    - hereditaria, 390
      - nail changes in, 1105
    - pathology of, 392
    - prognosis of, 393
    - symptoms of, 391
    - treatment of, 393

Epidermophyton inguinale, 222, 869
  Perneti, 870
  rubrum, 870
Epithelial cancer, 629
  degeneration, 70
Epithelialkrebs (Epithelioma), 629
Epithelioma, **629–650**
  adenoides cysticum, 614
  contagiosum, 594
  deep, 633–635
  etiology of, 638–644
  of lip, 636
  morphea-like, 632
  multiple, 632
    benign cystic, **614–617**
      diagnosis of, 617
      etiology of, 616
      pathology of, 616
      prognosis of, 617
      treatment of, 617
  papillary, 635–636
  pathology of, 644–646
  prognosis of, 650
  superficial, 629–631
  treatment of, 646–650
Epithéliomatose pigmentaire (Xeroderma pigmentosum), 620
Epithéliome kystique bénin (Syringocystoma), 617
Epitrichial layer, 47
Eponychium, 40
Equinia, **328–330**
  chronic, 329
  diagnosis of, 330
  etiology of, 330
  pathology of, 330
  prognosis of, 330
  symptoms of, 328
  treatment of, 330
Erbgrind (Favus), 857
Erectores pilorum, 34
Ergot eruptions, 181
Ergotism, 403
Erntemilbe (Leptus), 956
Erosions (Excoriations), 57
Eruption, acneform, **1036–1039**
  chronique circinée de la main, 138
  creeping, 966–967
  diagnostic importance of, 75
    color, 76
    configuration, 76
    distribution, 75
  drug, **174–186**
  feigned, **200–203**
    diagnosis of, 203
    etiology of, 202
    prognosis of, 203
    symptoms of, 200
    treatment of, 203
  medicamenteuse, 174
  recurrent summer, 387
  ringed, 138
  vaccinial, 430
Erysipel (Erysipelas), 331
Erysipelas, **331–336**
  ambulans, 332
Erysipelas, chronic, 333, 336
  diagnosis of, 334
  etiology of, 333
  Lombardy, 393
  pathology of, 334
  perstans faciei, 137, 698
  prognosis of, 335
  symptoms of, 331
  treatment of, 334
Erysipelatoid attacks on the face, recurrent, 337
Erysipèle (Erysipelas), 331
Erysipeloid, **336–337**
  diagnosis of, 337
  pathology of, 336
  symptoms of, 336
  treatment of, 337
Erythanthemata, 59
Erythema, **115–138**
  ab igne, 116
  annulare, 119, 127
  bullosum, 128
    vegetans, 383
  caloricum, 116
  circinatum, 127
  diagnosis of, 119, 234
  elevatum diutinum, **141**
      pathology of, 141
      treatment of, 141
  epidemicum, 403
  exudativum multiforme, 125
  figuratum, 128
    perstans, 137
  fugax, 119
  gangrænosum, 200
  hyperemicum (seu simplex), 116
  idiopathic, 140
  induratum, 137, **683–685**, 688
    diagnosis of, 685
    etiology of, 684
    pathology of, 684
    scrofulosorum, 687
    symptoms of, 683
    treatment of, 685
  infectiosum, 119
  intertrigo, **123–124**
    diagnosis of, 124
    etiology of, 123
    symptoms of, 123
    treatment of, 124
  iris, 128
  Jacquet's, **118**
    diagnosis of, 118
    pathology of, 118
  læve, 118
  marginatum, 128
  migrans, 117, 336
  multiforme, **125–135**
    diagnosis of, 134, 367, 378
    etiology of, 133
    pathology of, 133
    prognosis of, 134
    symptoms of, 127
    treatment of, 134
  nodosum, **135–137**
    diagnosis of, 137

Erythema nodosum, etiology of, 136
        pathology of, 136
        prognosis of, 137
        symptoms of, 136
        treatment of, 137
    nuchæ, 116
    papulatum, 128
    papulosum, 128
    paralyticum, 522
    paratrimma, 116
    pernio, **117**
        treatment of, 117
    perstans, **137–138**
        etiology of, 138
        faciei, 137
        pathology of, 138
        treatment of, 138
    punctatum, 120
    scarlatiniforme, **119–122**
        diagnosis of, 121
        etiology of, 121
        prognosis of, 122
        symptoms of, 120
        treatment of, 122
    scarlatinoid, 119
    serpens, 336
    simplex, 116
    solare, 116
    symptomatic, 118
    syphilitic circinate, 720
    tertiary circinate, 720
    toxic, 116
    traumaticum, 116
    treatment of, 119
    tuberculatum, 128
    urticans, 119
    urticatum, 128
    variolous, 415
    venenatum, 116
    vesiculosum, 128
Erythémato-sclérose circinée du dos des mains (Granuloma annulare), 138
Erythème ambulant, 1117
    centrifuge, 693
    induré des scrofuleux, 683
    infectueux, 120
    intertrigo, 123
    noueux, 135
    polymorphe, 125
    scarlatiniforme desquamatif, 120
    scarlatinoïde, 120
Erythrasma, **905–907**
    diagnosis of, 907
    etiology of, 906
    pathology of, 906
    prognosis of, 907
    symptoms of, 905
    treatment of, 907
Erythroderma desquamativa, **197–198**
Erythrodermia, resistant maculopapular scaly, 270
Erythrodermie congénitale ichtyosiforme, **494–495**
            diagnosis of, 495
            pathology of, 494
            treatment of, 494
Erythrodermie exfoliante, 186
        mycosique, 814
        pityriasique en plaques disséminées, 270, 272
Erythromelalgia, **851–853**
        diagnosis of, 853
        etiology of, 852
        pathology of, 852
        prognosis of, 853
        symptoms of, 852
        treatment of, 853
Erythromelie, 522
Etiology of skin diseases, **62–67**
Eucalyptus eruptions, 181
Eucerin, 97
Euproctus cysorhœa, 943
Examination, method of, 83–85
Exanthemata, 59, **405–432**
        drug. See Dermatitis medicamentosa.
        lichenoid psoriasiform, 270
Excoriations, 57
        neurotic, **203–205**, 979
            diagnosis of, 205
            etiology of, 204
            prognosis of, 205
            symptoms of, 203
            treatment of, 205
Exfoliatio areata linguæ, 1117
Eyelid, eczema of, 225
        lupus of, diagnosis of, 242
        Meibomian glands of, 44
        Moll's glands of, 45
        seborrhea of, 990
        xanthoma of, 603–607

F

FACE, eczema of, 223
        psoriasis of, 253
        recurrent erysipelatoid attacks of, 337
        seborrhea of, 990
        solid edema of, 337
Farcin (Equinia), 328
Farcy (Equinia), 328
Fat absorption by skin, 51
Fatty degeneration, 73
        layer of skin, atrophy of, **525–527**
Favus, **857–865**
        diagnosis of, 862
        etiology of, 861
        of nails, 1111
        pathology of, 861
        prognosis of, 865
        squamosus, 859
        symptoms of, 857
        treatment of, 863
Feet, eczema of, 226
        malum perforans of, 528
        psoriasis of, 253
Fehleisen's streptococcus, 333, 510
Feigwarze, 475, 729
Fettsklerem (Sclerema neonatorum), 504
Fetus, Harlequin, 490

Feuermal, 577
Fever blisters (Herpes simplex), 351
  miliary, **983**
Fibroids of skin, recurrent, 817
Fibrolysin eruptions, 181
Fibroma, **557–564**
  diagnosis of, 564
  etiology of, 563
  hard, 561
  molluscum, 557
   gravidarum, 557
   lentigo in, 535
  pathology of, 563
  pendulum, 559
  prognosis of, 564
  recurrent fibroid, 817
  symptoms of, 557
  treatment of, 564
Fibromatosis tuberculosa cutis, 667
Fibromyoma, 573
Fibrosarcoma, 817
Ficosis (Sycosis, coccognus), 1087
Fig-wart (Verruca acuminata), 475
Filaria bancrofti, 510
  dracunculus, 958
  medinensis, 958
  perstans, 968
  sanguinis hominis, 510
Filarial fever, 508
Finsen's light treatment, 110
Fischenschuppenausschlag (Ichthyosis), 487
Fish-skin disease (Ichthyosis), 487
Fissures (Rhagades), 58
Flannel-rash, 1004
Flea, 945
Flechte (Herpes simplex), 865, 874
Flecke (Maculæ), 54
Flecken (Rubeola), 410
Fleckenmal (Nevus pigmentosus), 478
Floh (Pulex irritans), 945
Flores unguium, 1099
Fluted tongue (Grooved tongue), 1116
Focal infections as etiologic factor, 66
Folliclis, 687, **689**, 986
Folliculite cicatricielle nécrotique, 1037
  épilante, 1083
Folliculitis, agglomerated, 687
  agminate, 867
  barbæ, 1087
  conglomerate, 867
  decalvans, **1083–1084**
   diagnosis of, 1084
   etiology of, 1084
   prognosis of, 1084
   treatment of, 1084
  disseminated, 687
  ulerythematosa reticulata, **486**
  varioliformis, 1037
Food, etiologic influence of, 63
  urticaria from, 145
Foot, elephant, 909
  endemic degeneration of bones of, 909
  fungus, of India, 909
  madura, 909
Foot, perforating ulcer of, 528
Fordyce's disease, **1123–1125**
   etiology of, 1125
   pathology of, 1125
   prognosis of, 1125
   symptoms of, 1125
   treatment of, 1125
Fowler's solution, 88
Fragilitas crinium, **1045–1046**
   treatment of, 1046
Frambesia, **830–833**
  diagnosis of, 833
  etiology of, 832
  histopathology of, 832
  pathology of, 832
  prognosis of, 833
  symptoms of, 830
  treatment of, 833
Franzosenkrankheit, 709
Freckle (Lentigo), 535
French measles, 413
Friesel (Sudamen), 980
Frostbeule (Erythema pernio), 117
Frostbite, 172
  itch, 843
Fulguration, **104**
Fungus, chignon, 1052
  disease of India, 909
  foot of India, 909
Furoncle (Furunculus), 315
Furrowed tongue (Grooved tongue), 1116
Furrows of corium, 18
Furs, dermatitis from, 167
Furunculus, **315–319**
  diagnosis of, 317
  etiology of, 316
  prognosis of, 317
  symptoms of, 319
  pathology of, 315
  treatment of, 317
Furunkel (Furunculus), 315

G

GAD-BREEZE, 965
Gad-fly, 965
Gafsa button (Oriental sore), 827
Galactidrosis, 979
Gale (Scabies), 947
Galvanocautery, 104
Gangosa, **835–838**
  diagnosis of, 838
  symptoms of, 836
  treatment of, 837
Gangrene, diabetic, **346–347**
   etiology of, 347
   pathology of, 347
   prognosis of, 347
   treatment of, 347
  hysterical, 200
  multiple, 344
   disseminated, in infants, 347
  neurotic, 200
  spontaneous, 200
  symmetrical, of extremities, **349–350**

- Gangrene, symmetrical, of extremities,
    - diagnosis of, 350
    - etiology of, 349
    - pathology of, 350
    - prognosis of, 350
    - symptoms of, 349
    - treatment of, 350
- Gastrophilus, 967
- Gayle, 324
- Gefässmal (Nevus vasculosus), 576
- Gelatins, 98
- Generalized pigmentation, 543
- Genital region, eczema of, 225
    - seborrhea of, 990
    - serpiginous ulceration of, 838
- German measles (Rötheln), 413
- Geschwülste (Tumor), 55
- Geschwüre (Ulcer), 58
- Giant cells, 72
- Glanders (Equinia), 328
- Glands, 43–47
    - ceruminous, 45
    - coil, 45–47
        - development of, 48
        - function of, 50
    - Meibomian, 44
    - Moll's, 45
    - sebaceous, 43–44
        - development of, 49
        - function of, 50
    - sudoriparous, 45
    - sweat, 45
    - Tysonian, 44
- Glandulæ ceruminosæ, 45
    - glomiformes, 45
    - odoriferæ, 44
- Glossina palpalis, 963
- Glossitis areata exfoliativa, 1117
    - Moeller's, 1120
- Glossy skin, **527**
    - etiology of, 527
    - pathology of, 527
    - prognosis of, 527
    - treatment of, 527
- Glycogelatins, 98
- Gnat stings, 947
- Gogo (Tinea imbricata), 898
- Goose-flesh, 35
- Gottheil's formula, 767
- Grain itch, **940–943**
    - diagnosis of, 942
    - etiology of, 941
    - pathology of, 941
    - prognosis of, 943
    - symptoms of, 941
    - treatment of, 943
- Granular layer of epidermis, 26
- Granuloma annulare, **138–140**
    - etiology of, 139
    - pathology of, 139
    - prognosis of, 140
    - symptoms of, 139
    - treatment of, 140
    - fungoides, **798–810**
        - cachectic stage of, 801
        - diagnosis of, 808
- Granuloma fungoides, etiology of, 801
        - fungoid stage of, 800
        - infiltration period, 799
        - pathology of, 802
        - prefungoid stage of, 799
        - prognosis of, 810
        - symptoms of, 798
        - treatment of, 809
        - tumor stage of, 800
    - inguinale tropicum, 838
    - innominatum, 687
    - necrotic, 688, 1037
    - perforating, of thigh, 838
    - pyogenicum, **587–589**
        - diagnosis of, 589
        - etiology of, 588
        - pathology of, 589
        - prognosis of, 589
        - symptoms of, 588
        - treatment of, 589
    - sclerotizing, of pudenda, 838
    - telangiectatic, 587
    - trichophyticum, 868
    - ulcerating, of pudenda, **838–840**
        - diagnosis of, 840
        - etiology of, 839
        - symptoms of, 839
        - treatment of, 840
- Granulomata, 72
- Granulomes à pédicule bénin (Granuloma pyogenicum), 587
- Granulosis rubra nasi, **986–987**
    - diagnosis of, 987
    - etiology of, 987
    - pathology of, 987
    - symptoms of, 987
    - treatment of, 987
- Graves' disease, pigmentation in, 538
- Grayness of hair (Canities), 1055
- Groin ulceration (Granuloma, ulcerating, of pudenda), 838
- Grooved tongue, 1116
- Ground itch (Uncinarial dermatitis), 962
- Grutum (Milium), 1009
- Grützbeutel (Steatoma), 1012
- Guaiacol, 89
- Guaiacum eruptions, 181
- Guinea worm (Dracontiasis), 958
- Guineesche Draake (Dracontiasis), 958
- Gumma, scrofulous, 669, 692
    - syphilitic, 746–750
    - tuberculous, 692
- Gummatous infiltration of skin and hypoderm, 748
- Gune (Tinea imbricata), 898
- Gürtelausschlag (Herpes zoster), 355
- Gürtelflechte (Herpes zoster), 355
- Gutta rosea (Acne rosacea), 1030

H

- HAARSACKMILBE (Demodex folliculorum), 964
- Haarzunge, schwarze, 1119
- Hair, **36–40**

Hair, atrophy of, 1045
  beaded, 1049
  bulb of, 36
  cells of, 36
  changes of, 40
  cortex of, 37
  cuticle of, 36
  deficiency of, 1057
  development of, 48
  diseases of, **1039–1092**
  dyes, dermatiti froms, 167
  follicle of, **38–40**
    cells of, 39
    cuticle of, 39
    diseases of, **1039–1092**
    layers of, 39
    root sheath of, 39
    trichostasis spinulosa of, 1054
  grayness of, 1055
  hoariness of, 1055
  hypertrophy of, 1039
  knotting of, 1050
  medulla of, 38
  microscopic examination of, 878
  papilla of, 36, 42
  point, 36
  ringed, 1056
  root of, 36
  shaft of, 36
  sudden blanching of, 1056
  superfluous, 1039
  whiteness of, 1055
Hairiness (Hypertrichosis), 1039
Hairy tongue, 1119
Hands, eczema of, 226
  keratosis of, 451
  psoriasis of, 253
Hang-nails, 1096
Hapalonychia, 1097
Harlequin fetus (Ichthyosis congenita), 490
Harnschweiss (Uridrosis), 978
Harvest bug (Leptus), 958
Hautabschürfungen (Excoriations), 57
Hautentzündung (Dermatitis), 161
Hautfinne (Acne), 1019
Hauthorn (Cornu cutaneum), 468
Hautschruden (Rhagades), 58
Hautsclerem (Scleroderma), 495
Heat rash, 981
  regulating function of skin, **50**
Hebra's acne cachecticorum, 687
  prurigo, 156
Hefenmykose (Blastomycosis), 916
Heitzmann's lines of cleavage, 25
Helcosoma tropicum, 829
Hemangio-endothelioma tuberosum multiplex (Syringocystoma), 617
Hematidrosis, **979–980**
Hematolymphangioma, 594
Hemidrosis, 979
Hemizona (Herpes zoster), 355
Hemochromatosis, **538**
Hemorrhages, **435–438**
  subungual, **1096**
Hemorrhoidalis, 967
Henle's sheath, 39
Henoch's purpura, 477
Heredity as etiologic factor, **64**
Herpes circinatus bullosus, 361
  desquamans, 898
  facialis, 351
  febrilis, 351
  generalized, 352
  genitalis, 352
  gestationis, 361, **365**
  iris, 128
    diagnosis of, 311, 378
  labialis, 351
  phlyctenoides, 361
  preputialis, 352
  progenitalis, 352, 353
  simplex, **351–355**
    diagnosis of, 354
    etiology of 353
    pathology of, 353
    prognosis of, 355
    symptoms of, 351
    treatment of, 354
  tonsurans, 865, 874
    desquamatif, 898
    maculosus, 274
  vegetans, 383
  zoster, **355–361**
    abdominalis, 358
    brachialis, 358
    capillitii, 357
    collaris, 358
    diagnosis of, 360
    etiology of, 358
    facialis, 358
    femoralis, 358
    frontalis, 357
    gangrenosus, 356
    hemorrhagicus, 356
    nuchæ, 358
    ophthalmicus, 357
    pathology of, 359
    pectoralis, 358
    prognosis of, 361
    symptoms of, 355
    treatment of, 360
      local, 361
    variations of, 356
Herpés circiné trichophytique, 865
Herpetic fever, 352
Herxheimer's reaction, 778
  spiral fibers, 28
Hexanitrodiphenylamin dermatitis, 167
Hide-bound skin (Scleroderma), 495
Hidradénomes éruptifs (Syringocystoma), 617
Hidrocystoma (Hydrocystoma), 983
High frequency current, **104**
Hirsuties (Hypertrichosis), 1039
History, diagnostic importance of, 74
  outline of, 83
Hives (Urticaria), 141
Hoariness (Canities), 1055
Holzbock (Ixodes), 946
Hookworm disease, 962
Horn, cutaneous, 468

Hornauswuchs (Cornu cutaneum), 468
Horny layer of epidermis, 27
Hühnerauge (Clavus), 467
Hunyadi János salts, 90
Hutchinson's malignant lentigo, 819
  melanotic whitlow, 821
Huxley's sheath, 39
Hyalin degeneration, 73
Hyalom der Haut (Colloid degeneration), 608
Hyaloma (Colloid degeneration), 608
Hybrid measles, 413
Hydradenitis suppurativa, **986**
    etiology of, 986
    pathology of, 986
    symptoms of, 986
    treatment of, 986
Hydroa æstivale, 387
  herpetiforme, 361
  puerorum, 389
  vacciniforme, **387–390**
    diagnosis of, 390
    etiology of, 389
    pathology of, 389
    prognosis of, 390
    symptoms of, 387
    treatment of, 390
  vésiculeux, 128
Hydrocystoma, **983–985**
  diagnosis of, 985
  etiology of, 985
  pathology of, 985
  symptoms of, 984
  treatment of, 985
Hydrosadenitis destruens, 687
  suppurativa, 687
Hydrosis (Hyperidrosis), 969
Hyperalgesia, 855
Hyperemia, 68, **115–433**
Hyperemic treatment, **104**
Hyperesthesia, 855
Hyperhidrosis, 969
Hyperidrose, 969
Hyperidrosis, **969–973**
  etiology of, 970
  oleosa, 992
  pathology of, 971
  prognosis of, 973
  symptoms of, 969
  treatment of, 971
Hyperkératose figurée centtriuge atrophiante (Porokeratosis), 458
Hyperkeratosis, 69
  excentrica, 458
  linguæ, 1119
Hypertrichiasis (Hypertrichosis), 1039
Hypertrichosis, **1039–1045**
  etiology of, 1041
  symptoms of, 1039
  treatment of, 1042–1044
Hypertrophies, **439–518**
Hypoderm, 18
  nodular tuberculosis of, 670
Hypohidrosis, 973
Hyponomoderma (Creeping eruption), 966
Hyponychium, 40
Hypotrichiasis (Alopecia congenita), 1058
Hysterical dermato-neuroses, 200
  gangrene, 200

I

Ichthyol, 89, 102
Ichthyosis, **487–495**
  congenita, 490
  cornea, 482
  diagnosis of, 493
  etiology of, 491
  follicularis, 491
  hystrix, 482, 489
  linearis neuropathica, 482
  linguæ, 1113
  nacree, 488
  nigricans, 488
  nitida, 488
  pathology of, 492
  prognosis of, 493
  serpentine, 488
  simplex, 487
  symptoms of, 487
  treatment of, 493
Ichtyose (Ichthyosis), 487
Idiosyncrasy, 62
Idrosis (Hyperidrosis), 969
Ignis sacer (Herpes zoster), 355
Impetigo, **305–314**
  Bockhart's, 305
  contagiosa, **305–311**
    bullosa, 307
      diagnosis of, 310
      etiology of, 309
      pathology of, 310
      symptoms of, 305
      treatment of, 311
    gyrata, 306
  eczematodes, 217
  herpetiformis, 305, **368–370**
    diagnosis of, 367, 370
    etiology of, 369
    pathology of, 370
    prognosis of, 370
    symptoms of, 369
    treatment of, 370
  rodens, 1037
  varicellosa, 427
  variolosa, 420
India fungus foot, 909
  ringworm, 898
Infections, transmission of, **65**
Inflammation, 68
Inflammations, **115–433**
Insanity caused by dermatitis exfoliativa, 190
Insect wounds, etiologic influence of, 63, 66
  urticaria from, 146
Instruments, **106–108**
Internal secretions, etiologic influence of, 65

- Intertrigo. See Eczema intertrigo, and Erythema intertrigo.
- Iodids in syphilis, 769
- Iodin, 88
  - dermatitis, 167, 181–183
- Iodipin, 769
- Iodoform eruptions, 183
- Iron, 90
- Itch, 947. See also Pruritus, Scabies.
  - barber's, 878
  - barley, 940
  - copra, 943
  - dhobie, 870
  - frost, 843
  - grain, 940
  - ground, 962
  - lumberman's, 844
  - Norwegian, 951
  - prairie, 844
  - straw, 940
  - swamp, 844
  - washerwoman's, 872
- Itching sensation, 52
- Ivy poisoning, 166
- Ixodes, **946–947**
  - treatment of, 947

J

- Jaborandi, 89
  - eruptions, 183
- Jacob's ulcer (Rodent ulcer), 631
- Jacquet's erythema, **118**
- Jigger (Pulex penetrans), 957

K

- Kahlkeit (Alopecia), 1057
- Kakerlaken, 543
- Karbunkel (Carbunculus), 319
- Kelis (Keloid), 551
- Keloid, **551–557**
  - acne, 1084
  - Addison's, 498
  - Alibert's, 551
  - cicatricial, 553
  - diagnosis of, 556
  - etiology of, 555
  - false, 551
  - idiopathic, 551
  - pathology of, 555
  - prognosis of, 557
  - spontaneous, 551
  - spurious, 551
  - symptoms of, 551
  - treatment of, 556
  - true, 551
- Keratin, 27, 28
- Keratoangioma (Angiokeratoma), 460
- Keratodermia blennorrhagica, **456–458**
    - diagnosis of, 458
    - etiology of, 457
    - pathology of, 457
    - symptoms of, 457
- Keratodermia blennorrhagica, treatment of, 458
  - excentrica, 458
  - palmaris et plantaris, **451–456**
    - diagnosis of, 455
    - etiology of, 455
    - pathology of, 455
    - prognosis of, 455
    - symptoms of, 451
    - treatment of, 455
  - symmetrical, of the extremities, 451
  - syphilitic, 734
- Keratodermic sandal, 284
- Keratodermie blennorrhagique, 456
  - palmaire et plantaire, 451
- Keratohyalin, 27, 28
- Keratolysis, 122
  - exfoliativa, 344
    - etiology of, 344
    - pathology of, 344
    - treatment of, 344
  - neonatorum, 196
- Keratoma, 466
  - senile, 444
- Keratosis follicularis, **446–450**
    - contagiosa, 450
    - diagnosis of, 449
    - etiology of, 448
    - pathology of, 448
    - prognosis of, 450
    - spinulosa, 443
    - symptoms of, 446
    - treatment of, 450
  - nigricans, 463
  - pigmentosa, 472
  - pilaris, **439–442**, 487
    - diagnosis of, 286, 441
    - etiology of, 440
    - pathology of, 441
    - symptoms of, 439
    - treatment of, 441
  - preëpitheliomatous, 444
  - senilis, **444–446**
    - diagnosis of, 445
    - pathology of, 444
    - prognosis of, 446
    - treatment of, 446
  - suprafollicularis, 439
  - universalis congenita, 490
  - vegetans, 446
- Kerion celsi, 876
- Kleienflechte (Tinea versicolor), 900
- Knollenkrebs (Keloid), 551
- Knötchen (Papulæ), 54
- Knoten (Tubercula), 55
- Knotting hair, 1050
- Koilonychia, **1098**
- Kolbenkörperchen, 33
- Koplik's spots, 410
- Krätze (Scabies), 947
- Kraurosis vulvæ, **530–532**
    - diagnosis of, 532
    - etiology of, 531
    - pathology of, 532
    - prognosis of, 532
    - symptoms of, 531

- Kraurosis vulvæ, treatment of, 532
- Krause's corpuscles, 33
- Kromayer lamp, 111
- Krusten (Crustæ), 57
- Kuhpocken (Vaccinia), 429
- Kupferfinne (Acne rosacea), 1030
- Kupferrose (Acne rosacea), 1030
- Kyste sébacé (Steatoma), 1012

L

- LABIALITIS (Perlèche), 1125
- Ladrerie (Lepra), 781
- Lafa Tokelau (Tinea imbricata), 898
- Lambkin's metallic mercury, 767
- Land scurvy, 437
- Langer's lines of cleavage, 25
- Langhans' cells, 26
- Langue noire, 1119
  - pileuse, 1119
- Lanugo, 36
  - development of, 48
- La rosa (Pellagra), 393
- La rose (Erysipelas), 331
- Larva migrans, 966
- Lassar's paste, 98, 242
- Läusesucht (Pediculosis), 932
- Lax skin (Dermatolysis), 515
- Leg, Barbadoes, 507
  - eczema of, 226
  - edema of, 507
  - elephant, 507
- Leichdorn (Clavus), 467
- Leiomyoma (Myoma), 573
- Leishmania tropica, 827
- Lentigo, **535–536**
  - etiology of, 536
  - Hutchinson's malignant, 819
  - maligna, 620
  - pathology of, 536
  - symptoms of, 535
  - treatment of, 536
- Lentile (Lentigo), 535
- Leontiasis (Lepra), 781, 786
- Lepidophyton, 899
- Lepothrix, 978, **1053–1054**
  - etiology of, 1054
  - pathology of, 1054
  - symptoms of, 1053
  - treatment of, 1054
- Lepra, 249, **781–798**
  - acute, 788
  - anesthetica, 784, 788–791
  - Arabum, 781
  - bacillus of, 792
  - diagnosis of, 794
  - etiology of, 791
  - geographical distribution of, 782
  - Italica, 393
  - Lombardy, 393
  - maculo-anesthetica, 784, 788–791
  - mutilans, 791
  - nodular, 785
  - pathology of, 792
  - prognosis of, 798
- Lepra, prophylaxis of, 795
  - symptoms of, 783
  - tegumentary, 785
  - treatment of, 795–798
  - tubercular, 785
  - tuberosa, 784, 785–788
- Lèpre (Lepra), 781
- Lepromes en nappe, 787
- Leprosy. See Lepra.
- Leptus, **956–957**
  - Americanus, 956
  - autumnalis, 956
  - irritans, 956
- Lesions, classification and description of, **53–61**
  - diagnostic significance of, **76**
- Leucasmus, 543, 544
- Leucémide, 812
- Leucoderma, acquired, 544
  - congenital, complete, 543
  - syphiliticum, 720
- Leucokeratosis buccalis, **1113–1116**
  - diagnosis of, 1115
  - etiology of, 1115
  - pathology of, 1115
  - prognosis of, 1116
  - symptoms of, 1113
  - treatment of, 1116
- Leucoma (Leucokeratosis buccalis), 1113
- Leucoplakia buccalis (Leucokeratosis buccalis), 732, 1113
- Leucoplasie (Leucokeratosis buccalis), 732, 1113
- Leukasmus, 544
  - congenital, 543
- Leukemia cutis, **810–814**
  - etiology of, 813
  - pathology of, 813
  - symptoms of, 810
  - treatment of, 814
- Leukoderma, acquired, 544
  - congenital, complete, 543
  - syphiliticum, 720
- Leukonychia, 1099
- Leukopathia, 544
  - congenital, 543
  - unguium, 1099
- Lewin's soluble salts of mercury, 766
- Lichen annularis, 138
  - annulatus et serpiginosus, 1004
  - circumscriptus, 1004
  - eczematodes, 215
  - nitidus, **304**
    - diagnosis of, 304
    - etiology of, 304
    - pathology of, 304
    - symptoms of, 304
    - treatment of, 305
  - obtusus, 293
    - corneus, 160
  - pilaris, 439
  - pilaris seu spinulosus, 291, **442–444**
    - diagnosis of, 444
    - etiology of, 443
    - pathology of, 443
    - prognosis of, 444

- Lichen pilaris seu spinulosus, symptoms of, 442
    - treatment of, 444
  - planopilaris, 294
  - planus, 299, **287–301**
    - annularis, 293
    - diagnosis of, 235, 263, 286, 299, 303
    - erythematosus, 293
    - etiology of, 297
    - forms of, 292–297
    - hypertrophicus, 292
    - linearis, 293
    - morphœicus, 296
    - of mucous surfaces, 294
    - obtusus, 293
    - pathology of, 298–299
    - prognosis of, 301
    - sclerosus et atrophicus, 296
    - symptoms of, 287–292
    - treatment of, 299–301
    - verrucosus, 292
  - psoriasis, 280, 287
  - ruber, 279, 280
    - acuminatus, 263, 279, 280, 285
    - moniliformis, 293
    - planus, 279, 287
  - scrofulosorum, **685–687**
    - diagnosis of, 687
    - etiology of, 686
    - pathology of, 686
    - prognosis of, 687
    - treatment of, 687
  - scrofulosus, 685
  - simplex chronica, 301
  - spinulosus, 291, 440, **442**
  - syphilitic, 723
  - tropicus, 981
  - urticatus, 144
  - variegatus, 272
- Lichenification, 59, **301–303**
  - diagnosis of, 303
  - histopathology of, 303
  - symptoms of, 301
  - treatment of, 303
- Lineæ albicantes, 22, 523
  - gravidarum, 523
- Lines of cleavage, 25
- Lingua nigra, 1119
- Liniments, 96
- Linsenmal (Nevus pigmentosus), 478
- Lioderma essentialis cum melanosi et telangiectasia (Xeroderma pigmentosum), 620
- Lipiodol, 769
- Lipoma, **569–571**
  - diagnosis of, 571
  - etiology of, 571
  - pathology of, 571
  - prognosis of, 571
  - symptoms of, 569
  - treatment of, 571
- Lips, cancer of, 636
  - cheilitis glandularis of, 1121
  - eczema of, 224
  - exfoliation of, 1122
- Lips, perlèche of, 1125
  - pseudo-colloid of, 1123
- Liquid air, 113
- Liquor potassæ, 90
- Lithium carbonate, 90
- Lombardy erysipelas, 393
  - leprosy, 393
- Lotio alba, 704
- Lotions, 96
- Lousiness (Pediculosis), 932
- Lues venerea (Syphilis), 709
- Luetin test, **81–83**, 760
- Lumberman's itch, 844
- Lumpy jaw (Actinomycosis), 912
- Lupoid, benign miliary, 706
- Lupoïdes miliaires disseminées, 706
- Lupus crustosus, 661
  - edematosus, 658
  - elephantiaticus, 658
  - elevatus, 658
  - endemicus, 827
  - erythematodes, 693
  - erythematosus, **693–706**
    - diagnosis of, 235, 702
    - discoid, 693
    - disseminatus, 687, 697
    - etiology of, 698
    - pathology of, 700
    - prognosis of, 706
    - symptoms of, 693
    - telangiectatic, 698
    - treatment of, 703
  - exedens, 660
  - exfoliativus, 658
  - exuberans, 658
  - exulcerans, 661
  - fibrosus, 659
  - fungoides, 661
  - fungosus, 661
  - gangrenosus, 661
  - hypertrophicus, 658
  - keloides, 661
  - lymphaticus, 591
  - maculosus, 658
  - mutilans, 663
  - nevus, 584
  - nodosus, 658
  - non-exedens, 658
  - non-ulcerosus, 658
  - papillosus, 658
  - pernio, 698
  - planus, 658
  - profundus, 661
  - psoriasiformis, 658
  - psoriasis, 658
  - rodens, 661
  - rupioides, 661
  - scléreux, 659
  - sclerosus, 659
  - sebaceus, 693
  - serpiginosus, 661
  - superficialis, 661, 693
  - telangiectatic, 698
  - tuberculatus, 658
  - tumidus, 658
  - ulcerating, 660

Lupus vegetans, 661
  vulgaris, **658–664**
    complications of, 664
    diagnosis of, 677, 702
    erythematoid, 664, 678, 702
    etiology of, 673
    hycopersicum dermatitis, 166
    multiple disseminated, 664
    pathology of, 676
    prognosis of, 682
    symptoms of, 658–664
    treatment of, 679
Lustgarten's bacillus, 756
Lustseuche (Syphilis), 709
Lymphadenectasia, 591
Lymphadenides, 812
Lymphadenoma cutis, 814
Lymphadenosis, 613
Lymphangiectasis, 460, **590**
Lymphangiectodes (Lymphangioma circumscriptum), 591
Lymphangioma, **589–594**
  capillare varicosum, 591
  cavernosum, 591
  circumscriptum, **591–594**
    diagnosis of, 594
    etiology of, 593
    pathology of, 593
    prognosis of, 594
    symptoms of, 591
    treatment of, 594
  congenital, 591
  cystic, 591
  simple, 590
  tuberosum multiplex, 617
Lymphangiomyoma, 573
Lymphangitis, telangiectatic, 510
  tuberculous, 670
Lymphatic vessels, 33
Lymphodermia perniciosa, **814**
Lymphogranulomatosis, 814
Lymph-scrotum, **510**

M

Machrochilia, 590
Macroglossia, 590
Maculæ, 54
  ceruleæ, 938
Maculo-papules, 54
Madura disease, 909
  foot, 909
Madurafuss (Mycetoma), 909
Magnesium sulphate, 90
Majocchi's telangiectasia, 208
Mal de Meleda, 456
  perforant du pied, 528
  pintado, 907
  de los pintos, 907
  rosso, 393
Maladie pédiculaire, 932
Maliasmus (Equinia), 328
Malleus (Equinia), 328
Malum perforans pedis, 528
Mamanpian, 831
Mange, Texas, 844
Marsden's paste, 649
Masern (Rubeola), 410
Mast cells, 72
Match-box dermatitis, 167
Mazamorra (Uncinarial dermatitis), 962
Measles (Rubeola), 445
  bastard, 413
  French, 413
  German, 413
  hybrid, 413
Medina worm (Dracontiasis), 958
Medinawurm, 958
Megalosporon, 884
  ectothrix, 884
  endo-ectothrix, 884
  endothrix, 875, 884
Meibomian glands, 44
Meissner's corpuscles, 32
Melanidrosis, 976
Melanin, 35
Melanoblasts, 36
Melanoderma, 537
Melanoepithelioma, 628
Melanoma (Sarcoma, melanotic), 819
Melanosis lenticularis progressiva (Xeroderma pigmentosum), 619
Melanotic carcinoma, 627
  sarcoma, 819
  whitlow, 821
Meleda, mal de, 456
Menopause, 65
Menstruation, 65
Mentagra (Sycosis, coccogenous), 1087
  parasitica, 878
Meralgia paresthetica, **855–856**
Mercury, 88, 103
  acid nitrate, 100
  eruptions from, 168, 183
  in syphilis, 762–769
    fumigation of, 765
    ingestion of, 762
    injection of, 765
    inunction of, 764
Merkel's corpuscles, 33
Metol dermatitis, 168
Micrococcus pemphigi neonatorum, 309
Microsporon, 883
  Audouini, 875, 883, 884, 885, 886
  canis, 884
  felineum, 884
  fulvum, 884
  furfur, 871, 902
  lanosum, 868, 884, 885, 887
  minutissimun, 871, 906
  pubiscens, 884
  tardum, 884, 885
  tomentosum, 884
  umbonatum, 884
  velveticum, 884
  villosum, 884
Midal eruptions, 183
Midge bites, 947
Miliaire crystalline, 980
  scrofuleuse, 1037
Miliaria crystallina, 980
  rubra, **981–983**

- Miliaria rubra, diagnosis of, 982
    - etiology of, 982
    - pathology of, 982
    - symptoms of, 981
    - treatment of, 982
- Miliary fever, 983
- Milium, **1009–1011**
    - colloid, 608
    - congénitale, 1011
    - diagnosis of, 1011
    - etiology of, 1010
    - pathology of, 1010
    - prognosis of, 1011
    - symptoms of, 1009
    - treatment of, 1011
- Milroy's disease, **507**
- Milzbrand (Anthrax), 325
- Mineral waters, 90
- Mistura ferri acida, 90
- Mitesser (Comedo), 1014
- Moeller's glossitis, 1120
- Mole, pigmentary, 478
- Moll's glands, 45
- Molluscum contagiosum, **594–598**
    - diagnosis of, 597
    - etiology of, 595
    - pathology of, 596
    - symptoms of, 594
    - treatment of, 598
    - corpuscles, 596
    - epitheliale, 594, 1037
    - fibrosum, 557
    - sebaceum, 594
    - simplex pendulum, 557
    - verrucosum, 1037
- Mongolian pigment spots, **538**
- Monilethrix, **1049–1050**
    - etiology of, 1050
    - pathology of, 1050
    - prognosis of, 1050
    - symptoms of, 1049
    - treatment of, 1050
- Morbilli (Rubeola), 410
- Morbus gallicus, 709
    - maculosus Werlhofii, 437
    - pediculosis, 932
    - pedis entophyticus, 909
    - tuberculosis pedis, 909
- Moriglione (Varicella), 426
- Morococcus, 232, 1006
- Morphea, 498
    - guttata, 499
    - nigra, 787
- Morphea-like epithelioma, 632
- Morpion (Pediculosis pubis), 937
- Morvan's disease, **529–530**
    - diagnosis of, 530
    - etiology of, 530
    - pathology of, 530
    - symptoms of, 529
    - treatment of, 530
- Morve (Equinia), 328
- Mosquito bites, 947
- Mountain anemia, 962
- Mouth, atrophy of mucous membrane of, **1129**
- Mouth, leucokeratosis of, 1113
    - necrotic ulcer of, 1128
    - smoker's patches of, 1113
    - trench, 1127
- Mower's mite (Leptus), 956
- Mucoid degeneration, 73
- Mucous layer of epidermis, 25
    - membrane, atrophy of, 1129
        - disease of, **1113–1129**
    - patches, 729
- Multiple abscesses of skin of infants, **322–323**
    - benign cystic epithelioma, **614–617**
        - epithelioma of scalp, 654
        - sarcoid, **706–709**
        - tumor-like new-growths, **529**
    - epitheliomata, 632
- Muscidæ, 965
- Muscles, 34
- Mycetoma, **909–912**
    - diagnosis of, 912
    - etiology of, 911
    - pathology of, 911
    - prognosis of, 912
    - symptoms of, 910
    - treatment of, 912
- Mycétome (Mycetoma), 909
- Mycosis cutis chronica, 827
    - fungoides, 798
    - microsporina, 900
- Myiasis cutanea, **965–966**
        - treatment of, 966
    - dermatosa, 965
        - mucosa, 965
        - œstrosa, 965
    - externa, 965
    - interna, 965
    - intestinalis, 965
- Myoma, **573–574**
    - dartoic, 573
    - diagnosis of, 574
    - simple, 573
    - telangiectodes, 573
    - treatment of, 574
- Myxadenitis labialis (Cheilitis glandularis), 1121
- Myxedema, **512–514**
    - diagnosis of, 514
    - etiology of, 513
    - pathology of, 514
    - symptoms of, 513
    - treatment of, 514
- Myxomatous degeneration, 73

N

- NÆVUS. *See* Nevus.
- Naftalan, **102**
- Nagelaushöhlung, 1098
- Nail, absence of, 1112
    - atrophy of, **1097–1103**
    - Beau's lines of, 1100
    - bed, 41, 42
        - syphilis of, **1106**
    - body of, 40

- Nail, cells of, 41
  - chancre of, 1105
  - congenital abnormalities and diseases of, **1112**
  - cornification of, 42
  - diseases of, **1093–1112**
    - etiology of, 1101
    - pathology of, 1101
    - treatment of, 1101
  - eczema of, **1104**
    - diagnosis of, 1105
    - treatment of, 1105
  - favus of, 1111
  - fold of, 40
  - furrows of, 1100
  - gift-spots of, 1099
  - groove of, 40
  - growth and reproduction of, 43
  - hang-nails, 1096
  - hypertrophy of, 1093
  - ingrowing, 1096
  - lunula of, 40
  - matrix of, 40
    - syphilis of, 1106
  - morbid conditions of, induced by cutaneous diseases, **1103–1105**
  - mycosis of, 1109
  - parasitic diseases of, 1109
  - plate, 41–43
    - syphilis of, 1106
  - psoriasis of, 254, **1103**
  - pterygium of, 1096
  - reedy, 1100
  - ringworm of, 1109
  - root of, 40
  - splitting of, 1100
  - spoon, 1098
  - subungual hemorrhage of, **1096**
    - tumor formation of, 1097
  - supernumerary, 1112
  - syphilis of, **1105–1109**
  - trichophytosis of, 1109
  - ulcerating, 1109
  - white spots of, 1099
- Narben (Cicatrix), 58, 549
- Nares, eczema of, 224
  - pruritus of, 843
- Nastin, 797
- Natal sore, 314, 827
- Necator americanus, 962
- Negroes, piebald, 543
- Neoarsphenamin, 775
- Neoplasie circinée et nodulaire (Granuloma annulare), 138
- Neosalvarsan, 775
- Nerve fibers, 29
    - medullated, 30
    - non-medullated, 30
    - tumor of, 571
- Nesselsucht (Urticaria), 141
- Nettle-rash (Urticaria), 141
- Neuralgia cutis, 853
- Neurofibroma (Fibroma), 557
- Neurom (Neuroma), 571
- Neuroma, **571–572**
  - pathology of, 572
- Neuroma, symptoms of, 571
- Neuro-recurrences in syphilis, 779
- Neuroses, **841–856**
- Neuro-syphilide, 720
- Neurotic excoriations, 203, 979
- Nevocarcinoma (Sarcoma, melanotic), 819
- Nevomelanoma (Sarcoma, melanotic), 819
- Nevoxantho-endothelioma, 654
- Névrodermite (Lichenification), 309
- Névrome (Neuroma), 571
- Nevus anemicus, **583–584**
  - araneus, 581, 1030
  - capillary, 576
  - cutaneous, 576
  - flammeus, 577
    - treatment of, 581
  - giant, 479
  - linear, **479**
  - lipomatodes, 479
  - lupus, 584
  - nervosus, 482
  - papillomatosus, 479
  - pigmentosus, **478–486**
    - diagnosis of, 485
    - etiology of, 484
    - pathology of, 484
    - prognosis of, 486
    - treatment of, 485
  - pilosus, 479
  - sanguineus, 576
  - spilus, 478, 479
  - unius lateralis, 482
  - vasculaire, 482
  - vascularis, 482, 576
  - vasculosus, 482, **576–581**
    - diagnosis of, 579
    - etiology of, 579
    - pathology of, 579
    - prognosis of, 581
    - treatment of, 579
  - verrucosus, 479
- New growths, **549–840**
  - multiple benign tumor-like, 529
- Niarde (Perlèche), 1125
- Nietnagel, 1096
- Nigritie de la langue, 1119
- Nigua (Pulex penetrans), 957
- Nipple, eczema of, 226
  - eczematous epitheliomatosis of, 650
  - Paget's disease of, 650
- Nitric acid, 100
- Nocardiosis cutis, **932**
- Nodositas crinium, 1046
- Nodules (Tubercula), 55
- Noguchi's luetin test, **81–83**
- Noli me tangere (Rodent ulcer), 631
- Noma, 411
- Norwegian itch, 951

O

- OCCUPATION, etiologic influence of, **63**
- Œdema. See Edema.
- Oehl's layer, 27
- Œil de perdrix (Clavus), 467

Œstridæ, 965
Ohio scratches, 844
Oils, **96**
Ointments, **97**
Oligidrosis, 973
Oligotrichia congenita (Alopecia congenita), 1058
Onychatrophia, **1097**
Onychatrophie, 1097
Onychauxis, **1093-1097**
Onych-heteropia, **1112**
Onychia, **1094**
  friable, 1106
  maligna, 1094
Onychitis, 1094
Onychogryphosis (Onychauxis), 1093
Onycholysis, 1098
Onychomadesis, 1099
Onychomycose, 1109
  favique, 1111
Onychomycosis, **1109-1112**
  diagnosis of, 1111
  favosa, 1111
  treatment of, 1111
  trichophyosis, 1111
Onychoptosis, 1099
Onychorrhexis, 1100
Onychoschizia, 1098
Onyxis syphilitique, 1106
Ophiasis, 1073
Opium eruptions, 183
Opsonic index, **92**
Opsonins, 91
Oriental button, 827
  sore, **827-830**
    diagnosis of, 829
    etiology of, 828
    geographic distribution of, 827
    pathology of, 829
    prognosis of, 830
    symptoms of, 828
    treatment of, 829
  ulcer, 827
Oroya fever (Verruga peruana), 834
Orthoform dermatitis, 168
Osmidrosis (Bromidrosis), 974
Osteoma cutis, **574-575**
Osteosis cutis (Osteoma cutis), 574
Oxyuris vermicularis, 961

P

Pachydermatocele (Dermatolysis), 515
Pachydermia, 507
  lymphangiectatica, 590
Pacinian corpuscles, 18, 30
Paget's disease of nipple, **650-653**
    prognosis of, 653
    symptoms of, 650
    treatment of, 653
Pain sense, **52**
Panaris analgésique (Morvan's disease), 529
Panaritium, 1095
Panniculus adiposus, 18
Pannus carateus (Pinta), 967
Papillæ, nervous, 24
  vascular, 24
Papilloma neuropathicum unilaterale, 482
Papulæ, 54
Papules, 54
Papulo-pustules, 56
Papulo-squamous lesions, 55
Papulo-tubercles, 57
Papulo-vesicular lesions, 55
Paraffin dressings, 171-172
Paraffinoma, **564-568**
  diagnosis of, 567
  histopathology of, 567
  prognosis of, 568
  symptoms of, 565
  treatment of, 568
Parakeratosis, 70
  ostracea, 269
  scutularis, **269**
  variegata, 270, 272
Paralysis, analgesic, with whitlow, 529
Parangi (Frambesia), 830
Paraphenylendiamin dermatitis, 167
Parapsoriasis, **270-274**
  diagnosis of, 273
  en gouttes, 270
  en plaques, 270, 272
  etiology of, 273
  guttate, 270
  lichénoïde, 270, 272
  pathology of, 273
  prognosis of, 274
  retiform, 272
  symptoms of, 270
  treatment of, 274
Parasites, 66
  animal, diseases due to, **932-968**
  vegetable, diseases due to, **857-932**
Parasitic diseases, 66, **857-968**
Paratuberculoses (Tuberculides), 687
Parchment skin, 621
Paresthesia, 855
Paronychia, **1095**
  syphilitica ulcerosa, 1106
Pars papillaris, 22-25
  reticularis, 22
Paste-pencils, 98
Pastes, 98
Pathology of skin diseases, **68-74**
Pediculidæ, 932
Pediculoides venticosus, 845, 941
Pediculosis, **932-939**
  capitis, **934-936**
    diagnosis of, 935
    treatment of, 936
  corporis, **936-937**
    diagnosis of, 937
    treatment of, 937
  pubis, **937-939**
    diagnosis of, 938
    treatment of, 939
Pediculus pubis, 595
  vestimenti, 936
Peitschenwurm (Dracontiasis), 958

Pelade (Alopecia areata), 1069
Peliosis rheumatica, 436
Pellagra, **393–403**
  diagnosis of, 402
  etiology of, 399
  pathology of, 401
  prognosis of, 403
  symptoms of, 395–399
  treatment of, 403
  typhosis, 395
Pellarella (Pellagra), 393
Pemphigoides maligna (Pemphigus vegetans), 383
Pemphigus, **370–387**
  acute febrile grave, 371
  acutus, **370–373**
    etiology of, 372
    pathology of, 373
    prognosis of, 373
    symptoms of, 372
    treatment of, 373
  aigu, grave, fèbrile, à forme infectieuse, 371
    prurigineux, 362
  benignus, 375
  circinatus, 361, 375
  composé, 362
  contagiosus, 307
  diagnosis of, 311, 367
  disseminatus, 375
  foliaceus, 191, 196, **379–383**
    diagnosis of, 236, 383
    etiology of, 381
    pathology of, 382
    prognosis of, 383
    symptoms of; 379
    treatment of, 383
  gangrænosa, 347
  hemorrhagicus, 375
  malignus, 375
  neonatorum, 308
  prurigineux, 362
  solitarius, 375
  syphiliticus, 741
  vegetans, **383–387**
    diagnosis of, 315, 386
    etiology of, 385
    pathology of, 385
    prognosis of, 387
    symptoms of, 384
    treatment of, 386
  vulgaris, **373–379**
    diagnosis of, 377
    etiology of, 376
    pathology of, 377
    prognosis of, 379
    symptoms of, 374
    treatment of, 378
Penicillium glaucum, 400
Penis, Tysonian glands of, 44
Perforating granuloma of thigh, 853
  ulcer of foot, **528–529**
    diagnosis of, 528
    etiology of, 528
    pathology of, 528
    prognosis of, 529
Perforating ulcer of foot, symptoms of, 528
    treatment of, 529
Periadenitis mucosa necrotica recurrens, **1128**
Perical (Mycetoma), 909
Perionychium, 40
Perisyringitis chronica nasi, 986
Perlèche, **1125–1126**
  diagnosis of, 1126
  etiology of, 1126
  symptoms of, 1125
  treatment of, 1126
Pernio (Erythema pernio), 117
Perspiration. See Sweat.
Peruvian wart (Verruga peruana), 834
Petechiæ, 54, 435
Petite vérole (Variola), 415
Pfannenstiel's treatment of intranasal lupus, 711
Phenacetin eruptions, 183
Phenol, 89, **103**
  dermatitis from, 167
Phenolphthalein eruptions, 183
Phialophora verrucosa, 927
Phlegmona diffusa, **323**
    symptoms of, 323
    treatment of, 323
  gangrenosa, 323
  progressiva, 336
Phlyctenæ (Vesiculæ), 55
Phlyctenulæ (Vesiculæ), 55
Phosphoridrosis, 979
Phosphorus, 89
  eruptions, 184
Phototherapy, **110–111**
Phthiriasis (Pediculosis), 932
  palpebrarum, 938
Phtiriase (Pediculosis), 932
Phymata, 55
Physiologic state, etiologic influence of, **65**
Physiology of the skin, **49–52**
Pian (Frambesia), 830
  fongoïde, 798
Pick's liniment, 243
  varnish, 99
Piebald skin (Vitiligo), 544
  negroes, 543
Pied de madure, 909
Piedra, **1051–1053**
  diagnosis of, 1052
  etiology of, 1052
  nostras, **1053**
    treatment of, 1053
  pathology of, 1052
  treatment of, 1052
Pigment, 18, 27, **35**
  anomalies of, **535–548**
  development of, 48
  protective function of, 49
Pigmentary carcinoma, 627
  disease, peculiar progressive, **210–212**
  mole, 478
  sarcoma, multiple, 822
  spots, Mongolian, **538**
  syphiloderm, 744

- Pigmentary urticaria, 175
- Pigmentation, blue, **542**
  - generalized, **543**
- Pili annulati, 1049
- Pilocarpin, 89
  - eruptions, 183
- Pinta, **907–909**
  - diagnosis of, 908
  - etiology of, 908
  - pathology of, 908
  - prognosis of, 909
  - symptoms of, 907
  - treatment of, 909
- Pinto (Pinta), 907
- Piroplasmosis hominis (Rocky Mountain spotted fever), 432
- von Pirquet test, 78
- Pita (Tinea imbricata), 898
- Pituitary extract, 90
- Pityriasis circinata, 274
  - circiné et marginé, 274
  - disséminé, 274
  - lichenoides chronica, 270
  - linguæ, 1117
  - maculata et circinata, 274
  - pilaris, 280, 439
  - rosé de Gibert, 274
  - rosea, **274–279**
    - diagnosis of, 263, 278
    - etiology of, 277
    - pathology of, 277
    - symptoms of, 274
    - treatment of, 278
  - rubra, 186
    - aigu, 274
    - Crocker type of, 186
    - Hebra type of, 187, **193–196**, 286
      - diagnosis of, 195
      - etiology of, 194
      - pathology of, 195
      - prognosis of, 196
      - symptoms of, 193
      - treatment of, 196
    - pilaire, 280
    - pilaris, 279, **280–286**
      - diagnosis of, 236, 263, **286**, 440
      - etiology of, 285
      - pathology of, 285
      - prognosis of, 286
      - symptoms of, 280
      - treatment of, 286
  - simplex, 989
  - steatoides, 990
  - versicolor, 900
- Plaques blanches de la bouche, 1113
  - jaunâtres des paupières, 598
  - muqueuses, 729
  - ortiées, 55
- Plasma cells, 71
- Plasters, 98
- Plate cells, 71
- Plica neuropathica, **1045**
  - polonica, **1044**
- Pocken (Variola), 415
- Pocks (Variola), 415
- Podelcoma (Mycetoma), 909
- Poisons, animal, 324
- Poissonnade (Perlèche), 1125
- Poliosis (Canities), 1055
- Poliothrix (Canities), 1055
- Polydactyly, **1112**
- Polyidrosis (Hyperidrosis), 969
- Polypapilloma tropicum (Frambesia),830
- Polytrichia (Hypertrichosis), 1039
- Pomphi, 55
- Pompholyx, 340, 370
- Porokeratosis, **458–460**
  - etiology of, 460
  - pathology of, 460
  - symptoms of, 458
  - treatment of, 460
- Porrigo decalvans, 1069
  - favosa, 857
- Port-wine mark, 577
- Postmortem pustules, 324
  - tubercle, 665
- Potassium carbonate, 90
  - chlorate eruptions, 184
  - hydroxid, 100
  - iodid in syphilis, 769
- Pou de bois (Ixodes), 946
- Powders, **97**
- Pox (Syphilis), 709
- Prairie itch, 844
- Precancerous dermatoses, **653**
- Pregnancy, cutaneous disorders of, **65**
  - lineæ gravidarum, 523
- Prickle layer, 25
- Prickly heat, 981
- Primrose dermatitis, 166
- Primula dermatitis, 166
- Prognosis, **86**
- Protective function of skin, **49**
- Protozoan infections, **928**
- Proud flesh, 588
- Prurigo, **156–161**
  - agria, 156, 157
  - diagnosis of, 159, 236
  - etiology of, 157
  - ferox, 156, 157, 158
  - gravis, 156
  - Hebra's, 156, 157
  - hiemalis, 843
  - of infants and children, 156, 158
  - mitis, 156, 157
  - nodularis, **159–161**
    - etiology of, 161
    - pathology of, 161
    - prognosis of, 161
    - symptoms of, 160
    - treatment of, 161
  - pathology of, 158
  - prognosis of, 159
  - site of, 156
  - symptoms of, 156
  - treatment of, 159
  - winter, 843
- Pruritus, **841 851**
  - ani, 842
    - treatment of, surgical, 851

- Pruritus, bath, 844
  - diagnosis of, 846
  - essentialis, 841
    - local, 842
  - etiology of, 845
  - general, 841
  - hiemalis, 843
  - local, 842
  - narium, 843
  - pathology of, 846
  - prognosis of, 851
  - senile, 842
  - treatment of, 846–851
    - internal, 847
    - local, 848
    - surgical, 851
  - vulvæ, 843
    - treatment of, surgical, 851
- Pseudo-atheroma (Steatoma), 1012
- Pseudo-chromidrosis, 976
- Pseudo-colloid of lips (Fordyce's disease), 1123
- Pseudoleukemia cutis, **814–815**
    - etiology of, 815
    - pathology of, 815
    - symptoms of, 814
    - treatment of, 815
- Pseudoleukemids, 815
- Pseudo-pélade (Alopecia cicatrisata), 1080
- Pseudo-xanthoma elasticum, **607–608**
- Psora (Psoriasis), 249
- Psoriasis, **249–270**
  - acute, 256
  - age incidence of, 258
  - atypical forms of, 257–258
  - bacterial origin of, 260–261
  - buccal, 254, 1113
  - circinata, 251
  - diagnosis of, 236, 262–264, 278, 702
  - diffusa, 251
  - discoidea, 251
  - distribution of, 252–254
  - etiology of, 258
  - of face, 253
  - of feet, 253
  - figurata, 251
  - follicularis, 251
  - of genital region, 253
  - guttata, 251
  - of hands, 253
  - heredity in, 258
  - inveterata, 251
  - linguæ, 254, 732, 1113
  - metabolic disturbances in, 259
  - of mucous membrane, 254
  - of nails, 254, 1103
  - neurotic origin of, 259–260
  - nummularis, 251
  - orbicularis, 251
  - ostreacea, 257
  - parasitic origin of, 260
  - pathology of, 261
  - prognosis of, 269
  - punctata, 251, 255
- Psoriasis punctata unguium, 1103
  - rupioides, 252, 257
  - scaling in, 255
  - of scalp, 252, 262, 264
  - sequelæ of, 258
  - site of, 252
  - symptoms of, 250–258
  - temperature as etiologic factor in, 261
  - of tongue, 254, 732, 1113
  - treatment of, 264–269
    - general, 264
    - internal, 264
    - local, 266
    - radiotherapy in, 269
  - vaccinal, 261
  - verrucosa, 257
- Psorospermose folliculaire végétante (Keratosis follicularis), 446
- Psorospermosis, 446
  - cutaneous, 650
- Pterygium, **1096**
- Puce commune, 945
  - desable, 957
- Pudenda, sclerotizing granuloma of, 838
  - ulcerating granuloma of, 838
- Pulex irritans, **945–946**
    - treatment of, 946
  - penetrans, **957**
- Punaise des lits (Cimex lectularius), 939
- Purpura, **435–438**
  - annularis telangiectodes, **208–210**
      - diagnosis of, 210
      - etiology of, 210
      - pathology of, 210
      - prognosis of, 210
      - symptoms of, 208
      - treatment of, 210
  - cachectic, 436
  - etiology of, 437
  - fulminans, 437
  - hemorrhagica, 437
  - Henoch's, 436
  - infections, 435
  - mechanical, 436
  - neurotic, 436
  - pemphigoid, 436
  - rheumatica, 436
  - simplex, 436
  - symptomatic, 435
  - symptoms of, 435
  - toxic, 436
  - treatment of, 438
  - urticans, 436
  - urticata, 142
  - variolosa, 419
- Pusteln (Pustules), 56
- Pustulæ, 56
- Pustule, **56**
  - malignant, 325
  - maligne (Anthrax), 325
  - pathology of, 70
- Pyodermite végétante (Dermatitis vegetans), 314
- Pyoktanin-blue, 103
- Pyrogallic acid, 100

Q

- QUADDELN (Pomphi), 55
- Quijila (Ainhum), 532
- Quincke's edema (Angioneurotic edema), 153
- Quinin, 89
  - eruptions, 184
- Quinquaud's disease (Folliculitis decalvans), 1083
- Quirica (Pinta), 907

R

- RACE, etiologic influence of, **64**
- Radiodermatitis, **205–208**
  - pathology of, 207
  - symptoms of, 205
  - treatment of, 207
- Radiotherapy, **108–110**
- Radium, **111–113**
- Rash, canker, 405
  - scarlet, 405
  - wandering, 1117
- Rauber's layer, 47
- Raynaud's disease (Gangrene, symmetrical), 349
- Recklinghausen's disease, 557
- Red gum, 981
- Reedy nails, 1100
- Resistant maculo-papular scaly erythrodermias (Parapsoriasis), 270
- Resorcin, 89, **101**
- Respiration of skin, **52**
- Rete malpighii, 25
  - mucosum, 25–26
  - pegs, 23, 25
- Rhagades, **58**
- Rheumatism, erythema nodosum in, 136
- Rhinochoprion penetrans, 957
- Rhinophyma, 1032
- Rhinoscleroma, **655–657**
  - bacteriology of, 656
  - diagnosis of, 656
  - etiology of, 656
  - pathology of, 656
  - prognosis of, 657
  - symptoms of, 655
  - treatment of, 657
- Rhipicephalus annulatus, 946
- Rhus (Poison ivy), 166
- Ribbed tongue (Grooved tongue), 1116
- Ridges of skin, 18
- Rimæ (Rhagades), 58
- Ringelhaaren, 1049
- Ringworm (Trichophytosis), 865
  - bald, 875
  - of beard, 878
  - body, 865
  - Bowditch Island, 898
  - Burmese, 898
  - Chinese, 898
  - disseminated, 875, 887
  - eczematoid, of extremities, 869
  - honeycomb, 857
- Ringworm, India, 898
  - of scalp, 874
  - scaly, 898
- Risipola Lombarda (Pellagra), 393
- Ritter's disease (Dermatitis exfoliativa neonatorum), 196
- Rochelle salts, 90
- Rocky mountain spotted fever, **432–433**
  - etiology of, 433
  - pathology of, 433
  - prognosis of, 433
  - symptoms of, 432
  - treatment of, 433
- Rodent ulcer, **631**
- Roentgen rays, 108–110
  - dermatitis from, **205–208**
  - epithelial degeneration from, 70
- Rosa, La, 393
- Rosacea (Acne rosacea), 1030
  - acuminata, 1032
- Rose, La, 331
- Roseola, epidemic, 413
  - infantilis, 119
  - squameuse (Pityriasis rosea), 274
  - syphilitic, 719
  - vaccinosa, 430
  - variolous, 415
- Rosolia (Rubeola), 410
- Rötheln, **413–415**
  - diagnosis of, 413
  - etiology of, 413
  - pathology of, 413
  - prognosis of, 413
  - symptoms of, 413
  - treatment of, 415
- Rothlauf (Erysipelas), 331
- Rotzkrankheit (Equinia), 328
- Rougéole (Rubeola), 410
- Rouget (Leptus), 956
- Rubella (Rubeola), 413
- Rubeola, **410–413**
  - complications and anomalies of, 411
  - diagnosis of, 412, 413, 425
  - eruptive stage of, 410
  - etiology of, 411
  - notha, 413
  - prognosis of, 412
  - symptoms of, 410
  - treatment of, 412
- Rubéole (Rubeola), 413
- Rupia escharotica (Dermatitis gangrænosa infantum), 347

S

- SACCHAROMYCOSIS hominis (Blastomycosis), 916
- St. Anthony's fire (Erysipelas), **331**
- Salicylic acid, 100
  - eruptions, 176
- Salipyrin eruptions, 184
- Salol, 89
- Salvarsan, 772–779
- Salve-muslins, 98
- Salve-pencils, 98

- Sand flea (Pulex penetrans), 957
- Sandfloh (Pulex penetrans), 957
- Santonin eruptions, 184
- Sarcoid, multiple benign, **706–709**
    - diagnosis of, 709
    - etiology of, 708
    - pathology of, 708
    - prognosis of, 709
    - symptoms of, 706
    - treatment of, 709
  - nodularis, 706
  - Speigler-Fendt type of, 706
  - subcutaneous, 706
  - tumors, 138
- Sarcoïdes noueuses et nodulaires des membres (Sarcoid, multiple benign), 706
- Sarcoma capitis, **654–655**
  - cutis, **815–827**
    - diagnosis of, 817
    - etiology of, 817
    - generalized, 817
    - non-pigmented, 815
    - pathology of, 817
    - prognosis of, 817
    - treatment of, 818
    - types of, 815
  - fibrosarcoma, 817
  - idiopathic multiple hemorrhagic, **822–827**
      - etiology of, 825
      - pathology of, 825
      - prognosis of, 827
      - symptoms of, 823
      - treatment of, 826
    - pigmented, 299, 822
  - melanotic, **819–822**
    - diagnosis of, 822
    - pathology of, 821
    - prognosis of, 822
    - symptoms of, 819
    - treatment of, 822
  - non-pigmented, 815
  - recurrent fibroid, of skin, 817
- Sarcomatosis, 817
  - generalis (Granuloma fungoides), 798
- Sarcopsylla penetrans, 957
- Sarcoptes aucheniæ, 951
  - cameli, 951
  - canis, 951
  - capræ, 951
  - communis, 953
  - equi, 951
  - hominis, 953
  - ovis, 951
  - scabiei, 952–955
  - suis, 951
  - vulpis, 951
- Satyriasis (Lepra), 781
- Sauroderma, 488
- Savill's disease (Dermatitis exfoliativa epidemica), 198
- Scabies, **947–955**
  - animal, 951
  - diagnosis of, 954
- Scabies, etiology of, 952
  - Norvegica, 951
  - parasite, 951
  - pathology of, 953
  - prognosis of, 955
  - symptoms of, 947
  - treatment of, 954
- Scabrities unguium syphilitica, 1106
- Scabs (Crustæ), 57
- Scales, 56
- Scalp, eczema of, 223
  - endothelioma of, 654
  - favus of, 264
  - psoriasis of, 252, 262, 264
  - ringworm of, 874
  - sarcoma of, 654
  - seborrhea of, 989
  - trichophytosis of, 874
- Scar (Cicatrix), 58, 549
  - hypertrophic, 553
  - keloid, 553
- Scarf skin, 25
- Scarlatina, **405–410**
  - anginose, 406
  - diagnosis of, 408, 413
  - etiology of, 407
  - pathology of, 407
  - prognosis of, 409
  - septic, 406
  - symptoms of, 405
  - toxic, 406
  - treatment of, 409
- Scarlatine (Scarlatina), 405
  - foudroyante, 406
- Scarlatiniform typhus, 406
- Scarlatinoid erythema, 119
- Scarlatinoïde (Erythema scarlatiniforme), 119
- Scarlet fever, 405
  - rash, 120, 405
- Scars (Cicatrices), 58
- Schamberg's disease, **210–212**
- Scharlach (Scarlatina), 405
- Schimmelpilzmykose der Nägel, 1109
- Schleimhautpapeln (Syphilodermata, moist), 729
- Schmeerfluss (Seborrhea), 988
- Schönlein's disease, 436
- Schuppen (Squamæ), 56
- Schuppenflechte (Psoriasis), 249
- Schwarze Haarzunge (Tongue, hairy black), 1119
- Schweissdrüsenadenom mit Cystenbildung, 617
- Schweissflechte (Miliaria rubra), 981
- Sclerema adiposum, 504
  - adultorum, 495
  - edematosum, 505
  - neonatorum, **504–505**
    - etiology of, 504
    - pathology of, 504
    - prognosis of, 505
    - symptoms of, 504
    - treatment of, 505
  - of newborn, 504

Sclérème des nouveau-nés (Sclerema neonatorum), 504
Scleriasis (Scleroderma), 495
Sclerodactylia, 497
Scleroderma, **495–504**
  atrophic stage, 496
  circumscribed, 498
  diagnosis of, 502
  diffuse, 495
  etiology of, 497
  pathology of, 501
  symptoms of, 495
  treatment of, 503
  types of, 498
  Unna's card-like form of, 502
Sclérodermie (Scleroderma), 495
Sclerosis, initial (Chancre), 711
Sclerotizing granuloma of the pudenda (Granuloma, ulcerating, of pudenda), **838**
Scratching, etiologic influence of, 63
Scrofulide erythémateuse (Lupus erythematosus), 693
Scrofuloderma, **668–670**
  etiology of, 673
  pathology of, 676
  prognosis of, 683
Scrofulous gummata, 669
Scrotal tongue (Grooved tongue), 1116
Scrotum, elephantiasis of, 510
Scurvy, Alpine, 393
  land, 437
Seasons, etiologic influence of, 64
Sebaceous cyst, 1012
  flux, 988
  glands, **43**
    adenoma of, 610
    development of, 49
    disorders of, **988–1039**
    secretory function of, 50
  tumor, 1012
Sebiparous glands, 43
Seborrhagia (Seborrhea), 988
Seborrhea, **988–997**
  capitis, 989
  congestiva, 693
  corporis, **1004–1005**
  crusting form of, 990
  diagnosis of, 993
  etiology of, 991
  of eyelids, 990
  of face, 990
  furfuracea, 989
  genital, 990
  lichenoides, 1004
  milk crust, 990
  nigricans, 978
  oleosa, 988
  papulosa, 1004
  pathology of, 991
  pityriasiformis, 989
  prognosis of, 997
  scaling form of, 989
  sicca, 989
  simplex, 988
  treatment of, 993
Seborrhea of umbilicus, 990
  waxy form of, 990
Séborrhée (Seborrhea), 988
Seborrheids of face, 1004
Sebum, 44, 50
Secretions, internal, 65
Secretory function of skin, **50**
Sensation function of skin, **51–52**
Septum lucidum, 27
Serampion (Rubeola), 410
Serotherapy, 94
Serum eruptions, 184–185
  character of, 185
  frequency of, 184
Shedding of skin, **122**
Sheep camp fever (Rocky Mountain spotted fever), 432
Shingles (Herpes zoster), 355
Sifilide (Syphilis), 709
Silver nitrate eruptions, 185
Sleeping sickness, 963
Slipada (Mycetoma), 909
Small-pox (Variola), 415
Smoker's patches of mouth, 1113
Soaps, 95–96
Sodium biborate eruptions, 176
  cacodylate, 88
  carbonate, 90
  iodid in syphilis, 769
  sulphate, 90
Solid edema of face, 337
Sommersprosse (Lentigo), 535
Spargosis fibro-areolaris (Elephantiasis), 507
Spedalsked (Lepra), 781
Speigler-Fendt's sarcoids, 706
Sphaceloderma (Dermatitis gangrænosa), 344
Spider cancer, 581
  nevus, 581
Spirocheta aboriginalis, 840
  pallida, 756
    microscopic demonstration of, 756
    staining of, 757
  pertenuis, 830, 832
Spitzblattern (Varicella), 426
Spitzencondylom (Verruca acuminata), 475
Spitzenwarze (Verruca acuminata), 475
Splenic fever carbuncle (Anthrax), 325
Spongiasis, 232
Spoon nails, 1098
Sporisorium maydis, 400
Sporothrix, 931
  asteroïdes, 931
  Beurmanni, 931
  Dori, 931
  Gougeroti, 931
  indicum, 931
  Jeanselmei, 931
  Lesnei, 931
  Schencki, 931
Sporotrichosis, **928–932**
  diagnosis of, 931
  etiology of, 930

- Sporotrichosis, pathology of, 930
  - prognosis of, 932
  - symptoms of, 929
  - treatment of, 931
- Sporozoa furunculosa, 829
- Spots (Maculæ), 54
- Spotted disease of Central America, 907
  - fever, Rocky mountain, 432
  - sickness, 907
- Squamæ, 56
- Stains (Maculæ), 54
- Staphylococcus cereus albus, 1126
  - epidermidis albus, 232, 1075
- Staub's formula, 766
- Stearrhea (Seborrhea), 988
- Steatoma, **1012–1014**
  - diagnosis of, 1014
  - etiology of, 1013
  - pathology of, 1013
  - prognosis of, 1014
  - symptoms of, 1012
  - treatment of, 1014
- Stéatome (Steatoma), 1012
- Steatorrhea (Seborrhea), 988
- Steatozoön folliculorum, 964
- Stéréophlogose nodulaire et circinée, (Granuloma annulare), 138
- Stilli dilubiles, 98
  - unguentes, 98
- Stinking sweat, 974
- Stinkschweiss (Bromidrosis), 974
- Stomatitis, aphthous, **1126–1127**
  - symptoms of, 1127
  - treatment of, 1127
- Stomoxys calcitrans, 400
- Stramonium eruptions, 185
- Stratum corneum, 25, 27
  - disjunctum, 25, 28
  - filamentosum, 25, 26
  - germinativum, 25, 26
  - granulosum, 25, 26
  - intermedium, 25, 27
  - lucidum, 25, 27
  - mucosum, 25
  - subcutaneum, 18
- Straw itch (Grain itch), 940
- Strawberry tongue, 406
- Streptococcus, Fehleisen's, 510
  - plicatilis, 1125
- Striæ patellares, 524
- Strophulus (Milaria rubra), 981
  - albidus, 1009
- Strychnin eruptions, 185
- Subcutaneous tissue, anatomy of, **18–21**
    - of nail region, 42
- Subungual hemorrhage, 1096
  - tumor formation, 1097
- Sudamen, **980–981**
  - diagnosis of, 980
  - etiology of, 981
  - pathology of, 980
  - symptoms of, 980
  - treatment of, 981
- Sudamina alba, 980
  - crystallina, 980
  - rubra, 980
- Sudatoria (Hyperidrosis), 969
- Sudor sanguineosa, 979
- Sudoriparous glands, 45
- Suette miliaire (Miliary fever), 983
- Sukha pokla (Ainhum), 532
- Sulcated tongue (Grooved tongue), 1116
- Sulfarsenol in syphilis, 779
- Sulphonal eruptions, 185
- Sulphur, 89, **101**
- Sumach poisoning, 166
- Summer eruption, 387
- Superfluous hair (Hypertrichosis), 1039
- Susceptibility to skin diseases, 62
- Swamp itch, 844
- Sweat, **50**
  - bloody, 979
  - chemistry of, 50
  - colored, 976
  - ducts, 45
  - excessive, 969
  - fetid, 974
  - glands, 45–48, 50
    - adenoma of, 614
    - diseases of, **969–988**
    - fetal development of, 48
    - secretory function of, 51
  - phosphorescent, 979
  - pore, 45, 47, 48
  - secretion of, 51
  - stinking, 974
  - suppression of, 973
  - urinous, 978
- Sweating sickness, 983
- Sycose (Sycosis, coccogenous), 1087
- Sycosis, coccogenous, **1087–1092**
    - etiology of, 1090
    - pathology of, 1090
    - prognosis of, 1092
    - symptoms of, 1087
    - treatment of, 1091
  - frambœsiformis, 1084
  - hypogenous, 311, 878
  - lupoid, 1090
  - non-parasitic, 1087
  - nuchæ necrotisans, 1084
  - parasitica, 878
  - staphylogenes, 1087
  - tinea, 878
  - vulgaris, 1087
- Symptomatology of skin diseases, **53–61**
    - objective, 53–59
    - subjective, 53
- Symptoms, outline of, 84
- Synanthemata, 59
- Syndactyly, **1112**
- Synovial lesions of skin, **477–478**
    - treatment of, 478
- Syphilid. See Syphilodermata.
- Syphilide cornée, 733
- Syphilides. See Syphilodermata.
- Syphilis, **709–781**
  - alopecia areata in, 1074
  - chancre of, 711
  - congenital, 750–755
    - cutaneous manifestations 750

Syphilis, congenital, diagnosis of, 754
    prognosis of, 755, 781
    treatment of, 779–780
  diagnosis of, 759
  early lesions of, 716
  etiology of, 755
    microörganisms in, 756
  of feet, 732–735
  of hands, 732–735
  initial sclerosis of, 711
    excision of, 770
  iodids in, 769–770
  late lesions of, 746
  luetin test in, **81**, 760
  mercury in, 762–769
  mouth lotions in, 771
  mucous patches of, 59, 729
  of nails, **1105–1109**
  neuro-recurrences in, 779
  Noguchi's test in, **81**, 760
  palmar, 732, 735
  papillomatosa, 742
  pathology of, 757
  plantar, 732–735
  primary sore of, 711
  prognosis of, 780
  prophylaxis of, 780
  reinfection in, 750
  silvery spot in, 713
  skin manifestations of, 715–755
  spirocheta pallida in, 756
  subjective sensations in, 716
  superinfection in, 750
  symptoms of, concomitant, 718
    cutaneous, 715–755
  treatment of, 761–780
    arsphenamin in, 772–779
    cauterization in, 771
    general, 761
    hygiene in, 761
    iodids in, 769–770
    local, 770–772
    mercury in, 761, 762–769
    mixed, 769
      arsphenamin and mercury, 776
    neoarsphenamin in, 775
    ointments in, 771
    operative, 770
    sulfarsenol in, 779
    tonic, 763
  use of spirituous liquors in, 762
    of tobacco in, 762
  Wassermann reaction in, **78–81**, 759
Syphilitic circinate erythema, 720
  keratodermia, 734
  lichen, 723
  roseola, 719
Syphiloderm. *See* Syphilodermata.
Syphilodermata, **715–755**
  acneiform, 737
  acuminate miliary papular, 723
    pustular, large, 737
      small, 736
  annular papular, 726
  bullous, 741
Syphilodermata, bullous, congenital, 752
  career of, 716
  chancre, 711
    treatment of, 770
  characteristics of, general, 716
  classification of, 718
  color of, 716
  concomitant symptoms of, 718
  congenital, 750–755
    diagnosis of, 754
    prognosis of, 755
  configuration of, 716
  congenital, 750–755
  cornée, 733
  crusted lesions, treatment of, 771
  distribution of, 716
  ecthymaform, 739
  erythematous, 719
  of feet, 732
  follicular, 723
  frambesioides, 742
  grangrenous, treatment of, 771
  gummatous, 746–750
    congenital, 754
    diagnosis of, 750
    pathology of, 777
  of hands, 732
  impetigoform, 739
  lenticular papular, 725
  macular, 719–720
    congenital, 753
    diagnosis of, 720
    pigmentary, 720–723
  miliary papular acuminate, 723
    pustular, 736
  moist papules, 729–732
      diagnosis of, 732
  mucous patches, 729
      diagnosis of, 732
  nodular, 741–746
    congenital, 754
    diagnosis of, 745
  obtuse, 737
  opaline plaques, 731
  palmar, 732–735
    diagnosis of, 734
  papillomatous, 742
  papular, 723–734
    annular, 726
    congenital, 753
    corymbose, 725
    discoid, 725
    hypertrophic, 725
    large flat, 725
    lenticular, 725
    moist, 729–732
      diagnosis of, 732
    mucous patches, 729
    opaline plaques, 731
    small acuminate miliary, 723
        diagnosis of, 724
      flat, 725
    squamous, 732
  papulo-pustular, 725
  papulo-squamous, 726–728
  phagedenic, treatment of, 771

- Syphilodermata, pigmentary, 720–723
  - plantar, 732–735
    - diagnosis of, 734
  - polymorphism of, 718
  - pustular, 735, 739
    - congenital, 754
    - diagnosis of, 424
    - flat, 739
    - large acuminate, 737
      - diagnosis of, 737
    - small acuminate, 736
    - ulcerating, 740
  - pustulo-ulcerative, 740
    - diagnosis of, 741
  - serpiginous, 742, 744
  - site of, 717
  - squamous, 732–734
    - diagnosis of, 734
  - subjective sensations with, 716
  - ulcerative, pustular, 740
  - varicelliform, 735
  - varioliform, 737
  - vesicular, 735
- Syphilonychia sicca, 1106
- Syringocystadénome (Syringocystoma), 617
- Syringocystoma, **617–619**
  - diagnosis of, 619
  - pathology of, 618
  - prognosis of, 619
  - symptoms of, 618
  - treatment of, 619
- Syringoma (Syringocystoma), 617
- Syringomyelia, 529

T

- Tache de feu, 577
  - pigmentaire, 478
- Taches (maculæ), 54
  - bleuâtres, 938
  - ombrées, 938
- Tactile corpuscles, 32
  - sense, **52**
- Tænia echinococcus, 961
  - solium, 960
- Tar, 89, **103**
  - baths, 95
  - eruptions, 186
- Tarsonemidæ, 942
- Taschkat ulcer (Oriental sore), 827
- Tattooing, **542**
  - treatment of, 542
- Teigne faveuse, 857
  - tondante, 874
- Telangiectasia follicularis annulata (Purpura annularis telangiectodes), 208
- Télangiectasie verruqueuse (Angiokeratoma), 460
- Telangiectasis, **581–583**
  - faciei, 1030
  - multiple, 582
  - papillary ectasis, 582
  - treatment of, 582
- Telangiectatic granuloma, 587
- Telangiectatic lupus erythematosus, 693
  - lymphangitis, 510
- Temperature sense, 51
- Tenacetum eruptions, 186
- Texas mange, 844
- Thallium acetate alopecia, 186
- Therapeutics, **87–113**
  - external, 94–113
  - internal, 87–94
- Therapia sterilisans magna, 773
- Thigh, perforating granuloma of, 838
- Thyroid extract, 90
- Tic de l'épilation, 856
- Tick, cattle, 433
  - fever of Rocky Mountains, 432
  - wood, 946
- Tina (Pinta), 907
- Tinea barbæ, 878
  - circinata, 865
    - tropica, 898
  - decalvans, 1069
  - faveuse, 857
  - favosa, 857
    - precautions in treatment of, 897
    - unguium, 1109
  - imbricata, **898–900**
    - diagnosis of, 900
    - etiology of, 899
    - pathology of, 899
    - prognosis of, 900
    - symptoms of, 898
    - treatment of, 900
  - kerion, 876
  - nodosa, 1053
  - sycosis, 878
  - tondens, 874
  - tonsurans, 874
  - trichophytina cruris, **868–873**
    - unguis, 1109
  - versicolor, **900–905**
    - diagnosis of, 903
    - etiology of, 902
    - pathology of, 903
    - prognosis of, 905
    - symptoms of, 900
    - treatment of, 904
- Tingling sensation, 52
- Tique, 946
- Tobacco, use of, in syphilis, 762
- Tokelau ringworm (Tinea imbricata), 898
- Tonga (Frambesia), 830
- Tongue, atrophy of mucous membrane of, **1129**
  - black, 1119
  - chronic superficial excoriations of, **1120–1121**
    - etiology of, 1121
    - pathology of, 1121
    - prognosis of, 1121
    - symptoms of, 1120
    - treatment of, 1121
  - circinate eruption of, 1117
  - fluted, 1116
  - furrowed, 1116
  - grooved, **1116–1117**

- Tongue, hairy black, **1119–1120**
  - hyperkeratosis of, 1119
  - ichthyosis of, 1113
  - pityriasis of, 1117
  - psoriasis of, 254, 1113
  - ribbed, 1116
  - scrotal, 1116
  - sulcated, 1116
  - transitory benign plaques of, 1117–1119
    - treatment of, 1119
  - tylosis of, 1113
  - wrinkled, 1116
- Touch cells, Merkel's, 33
  - sense of, 52
- Toxituberculides (Tuberculides), 637
- Tragacanth jelly, 99
- Traumaticin, 99
- Trench mouth, 1127
- Treponema pallidum, 756
- Trichauxis (Hypertrichosis), 1039
- Trichiasis, **1044**
- Trichloracetic acid, 100
- Trichoclasia, 1046
- Trichoepithelioma papulosum multiplex (Multiple benign cystic epithelioma), 614
- Trichomycose nodulaire (Piedra), 1051
- Trichomycosis flava, 1054
  - nigra, 1054
  - nodosa, 1053
  - palmellina, 978, 1053
  - rubra, 1054
- Trichonodosis, **1050–1051**
- Trichonosis cana, 1055
- Trichopathophobia, 1073
- Trichophytie (Trichophytosis), 865
  - sycosique, 878
- Trichophyton acuminatum, 870, 885
  - album, 885
  - asteroides, 885, 891
  - caninum, 885
  - cerebriforme, 885
  - circumvolutum, 885
  - crateriforme, 884, 885
  - denticulatum, 885
  - discoides, 885
  - ectothrix, 890
    - megaspore, 891
  - effractum, 885
  - endothrix, 875, 883, 888
  - equinum, 885
  - exsiccatum, 885
  - farinulentum, 885
  - faviforme discoides, 886
  - fumatum, 885
  - glabrum, 885
  - granulosum, 885
  - gypseum, 867, 873, 885
    - asteroides, 886
  - lacticolor, 885
  - microïdes, 867
  - niveum, 867, 885
  - nodoformans, 870
  - ochraceum, 885
  - pilosum, 885
- Trichophyton plicatile, 868, 885
  - polygonium, 885
  - radians, 885
  - radiolatum, 885
  - regulare, 885
  - rosaceum, 885
  - sulphureum, 885
  - umbilicatum, 885
  - versicolor, 885
  - vinosum, 885
  - violaceum, 870, 885
- Trichophytosis, **865–900**
  - acute vesico-pustular, 869, 871
  - bacteriology of, 884
  - bald ringworm, 875
  - barbæ, **878–882**
    - diagnosis of, 881
    - etiology of, 883
    - symptoms of, 878
    - treatment of, 896
  - of beard, 878
  - black dot, 878, 888, 889
  - of body, 865
  - Bowditch Island ringworm, 898
  - Burmese ringworm, 898
  - capitis, **874–878**
    - diagnosis of, 877
    - etiology of, 883
    - symptoms of, 874
    - treatment of, 892
  - Chinese ringworm, 898
  - chronic intertriginous, 869
  - conglomerateor agminate folliculitis, 867
  - corporis, **865–874**
    - diagnosis of, 237, 264, 873
    - dry, 865
    - etiology of, 882
    - moist, 866
    - papular, 867
    - pustular, 867
    - symptoms of, 865
    - treatment of, 892
  - disseminated, 875, 887
  - dry, 865
  - ectothrix type, 883, 890
  - eczema marginatum, 870
  - eczematoid, of extremities, 869
    - treatment of, 893
  - endothrix, 883, 888
    - acuminate type, 888
    - crateriform type, 888
    - violaceum type, 889
  - etiology of, 882
  - of extremities, 869
    - treatment of, 893
  - glabrous skin, 868, 873
  - granuloma, 868
  - of hands, 869
  - histology of skin in, 891
  - hyperkeratotic, 869
  - India ringworm, 898
  - lichenoid, 873
  - microscopic examination of hair in, 877
  - microsporon audouini type, 886

- Trichophytosis, microsporon lanosum type, 887
  - moist, 866
  - of nails, 1109
  - palmar, 869
  - papular, 867
  - pathology of, 884
  - plantar, 869
  - precautions in treatment of, 897
  - pustular, 867
  - Sabouraud's classification of fungi, 884
  - of scalp, 874
  - scaly ringworm, 898
  - tinea kerion, 876
  - Tokelau ringworm, 898
  - treatment of, 892
    - precautions in, 897
  - types of, 866
  - unguium, **1109–1111**
    - diagnosis of, 1111
    - treatment of, 1111
- Trichoptilosis, 1046
- Trichorrhexis nodosa, **1046–1049**
  - etiology of, 1047
  - pathology of, 1047
  - treatment of, 1049
- Trichosporum giganteum, 1052
- Trichostasis spinulosa, **1054–1055**
- Trichotillomania, **856**
- Trombidæ, 956
- Trophedema, chronic hereditary, 507
- Tropical chlorosis, 962
- True skin, 21
- Trypanosoma gambiense, **963–964**
- Trypanosomiasis, 963
  - symptoms of, 963
  - treatment of, 964
- Tubercle, 55
  - anatomical, 63, 665
  - dissection, 665
  - postmortem, 665
- Tubercula, 55
- Tubercular gumma, 692
  - leprosy, 785
- Tuberculides, 658, **687–693**
  - acne cacheticorum, 691
    - scrofulosorum, 691
  - acnitis, 688, 689
  - etiology of, 691
  - folliclis, 688, 689
  - necrotic, 688
  - nodular, 689
  - papular, 689
  - pathology of, 692
  - symptoms of, 688
  - treatment of, 693
  - types of, **688**
- Tuberculin eruptions, 184
  - tests, **77–78**
    - hypodermic, 77
    - ophthalmic, 77
    - von Pirquet, 77
- Tuberculosis, acute, **672**
  - cutis, **657–682**
    - acute, 672
- Tuberculosis cutis, diagnosis of, 677
    - elephantiasis, 667
    - etiology of, 673
    - fibromatosis, 667
    - miliary, 672
    - orificialis, **671–672**
      - diagnosis of, 679
      - pathology of, 676
    - pathology of, 674–675
    - prognosis of, 682
    - treatment of, 679
    - verrucosa, **665–668**
  - exanthematous miliary, **672**
  - fungosa cutis, 670
  - hypodermic test in, 77
  - lymphangitis tuberculosa cutanea, 670
  - nodular, of hypoderm, 670
  - ophthalmo-tuberculin test in, 77
  - pruriginosa, 144
  - scrofuloderma, 668
  - ulcers, 671
  - verrucosa cutis, **666–667**
      - diagnosis of, 678
      - etiology of, 674
      - pathology of, 676
  - von Pirquet test in, 77
- Tuberculous eczema of nurslings, 221
- Tuberculum sebaceum, 1009
- Tuberosis cutis pruriginosa, 144
- Tumeurs (Tumor), 55
  - d'emblée, 801
- Tumor, 55
  - cavernosus, 575
  - multiple benign, 529
  - of nerves, 571
  - sarcoid, 138
  - sebaceous, 1012
  - subungual, 1097
- Tumores (Tumor), 55
  - benigni sarcoidei cutis, 138
- Turpentine, 89
  - eruptions, 186
- Tyloma (Callositas), 466
- Tylosis (Callositas), 466
  - linguæ, 1113
  - palmarum et plantarum, 451
- Typhus, scarlatiniform, 406
- Tyroglyphus longior, 943
- Tysonian glands, 44

U

- ULCER, **58**
  - cancroid, 631
  - crateriform, 636
  - oriental, 827
  - perforating, of foot, **528–529**
  - rodent, **631**
  - Taschkat, 827
  - tuberculous, 671
- Ulcères (Ulcer), 58
- Ulcus excedens, 631
  - grave, 909
  - neuroticum mucosæ oris, 1128

Ulerythema centrifugum, 693
  ophryogenes, **442**
  sycosiforme, 1090
Umbilicus, eczema of, 226
  seborrhea of, 990
Uncinarial dermatitis, **962–963**
    treatment of, 962
Uncinariasis, 962
Unguis incarnatus, **1096**
Universal congenital atrichia, 1058
Unna's balloon cell, 70
  ointment, 241
Uridrose (Uridrosis), 978
Uridrosis, 978–979
Urtica dioica, 146
  urens, 146
Urticæ, 55
Urticaria, **141–156**
  ab ingestis, 145
    treatment of, 148
  angioneurotic edema, 153
  annularis, 142
  bullosa, 142
  dermographism in, 143
  diagnosis of, 147
  edematosa, 153
  etiology of, 145
  evanida, 143
  factitia, 143
  figurata, 142
  giant, 153
  hemorrhagica, 142
  in infants and children, **144**
  mast cells in, 72
  papulosa, 142
  pathology of, 146
  perstans, 143
    verrucosa, 159
  pigmentosa, **149–153**
    diagnosis of, 153, 603
    etiology of, 151
    pathology of, 152
    prognosis of, 153
    symptoms of, 150
    treatment of, 153
  prognosis of, 148
  psychic, 145
  recidiva, 144
  solitaria, 143
  symptoms of, 141
  treatment of, 147
  tuberculosa, 142
  tuberosa, 144
    verrucosa, 144
  vesiculosa, 142
Urticarial autogram, 143
Ustilago maydis, 400

V

Vaccinal eruptions, 184, 430
  psoriasis, 261
  roseola, 430
Vaccination, **429**
  complications of, 431
  preparation of vaccine for, 429
  psoriasis following, 261
  sequelæ of, 431
Vaccine (Vaccinia), 429
  eruptions, 184, 430
Vaccines, preparation and standardization of, 92
Vaccinia, **429–432**
  dermatitis gangrenosa following, 348
  generalized, 430
  hemorrhagica, 430
Vaccinotherapy, 91–94
  external, 93
Vajuolo (Variola), 415
de Valagin's solution, 88
Varicella, **426–429**
  complications of, 427
  diagnosis of, 311, 424, 428
  etiology of, 428
  gangrenosa, 347
  pathology of, 428
  prognosis of, 429
  symptoms of, 426
  treatment of, 429
Varicelliform syphiloderm, 735
Variola, **415–426**
  complications of, 420
  confluent, 419
  cornea, 420
  crystallina, 420
  diagnosis of, 313, 424, 428
  eruption of, 415–419
  etiology of, 420
  hemorrhagic, 419
  initial rashes of, 415
  mild, 419
  nigra maligna, 419
  pathology of, 422
  pitting in, prevention of, 425
  prognosis of, 426
  pustulosa hæmorrhagica, 419
  red-light treatment of, 425
  sequelæ of, 420
  symptoms of, 415
  treatment of, 425
Variolette (Varicella), 426
Varioliform syphiloderm, 737
Varioloid, 420
Variolous erythema, 415
  roseola, 415
Varix lymphaticus (Lymph scrotum), 510
Varus (Acne), 1019
Vascular dilatation, acquired, 581
  system. See Bloodvessels.
Vaseline tumors, 566
Vater, corpuscles of, 30
Vegetable parasites, diseases due to, **857–932**
Veldt sore, **314**
    treatment of, 314
Venereal wart, 475. See also Syphilis.
Venerische granulom, 838
  warze, 475
Ver de Kutégal (Dracontiasis), 958

- Vergetures (Atrophia maculosa et striata), 523
- Verhärtung (Callositas), 466
- Vernix caseosa, 48, 990
- Vérole, 415
- Veronal eruptions, 186
- Verruca, **470–478**
  - acuminata, **475–477**
    - diagnosis of, 476
    - etiology of, 476
    - pathology of, 476
    - prognosis of, 477
    - symptoms of, 475
    - treatment of, 476
  - diagnosis of, 473
  - digitata, 471
  - etiology of, 472
  - filiformis, 471
  - necrogenica, 324, **665**
    - etiology of, 674
  - pathology of, 474
  - plana, 472
    - juvenilis, 471
  - plantaris, 471
  - prognosis of, 475
  - seborrheica, 472
  - senilis vel plana, 472
  - treatment of, 474
    - local, 474
  - venereal, 475
  - vulgaris, 470
- Verrue (Verruca), 470
  - telangiectasique (Angiokeratoma), 460
- Verrues charnues, 562
- Verruga peruana, **834–835**
  - diagnosis of, 835
  - etiology of, 835
  - prognosis of, 835
  - symptoms of, 834
  - treatment of, 835
- Vesicles, 55
  - pathology of, 70
- Vesico-bullæ, 56
- Vesico-papular lesions, 54
- Vesico-pustules, 56
- Vesiculæ (Vesicles), **55**
- Vespidæ, 947
- Vibices, 54, 435
- Vincent's disease, **1127–1128**
  - treatment of, 1128
- Vitiligo, **544–548**
  - acquisita syphilitica, 720
  - diagnosis of, 547
  - etiology of, 546
  - pathology of, 547
  - prognosis of, 548
  - symptoms of, 544
  - treatment of, 548
- Vitiligoid (Syphilodermata, pigmentary), 720
- Vitiligoidea (Xanthoma), 598
- Vleminckx's solution, 1035
- Voigt's boundary lines, 484
- Vulva, kraurosis of, 530
  - pruritus of, 843

W

- Wagner's corpuscles, 32
- Wandering edema, 153
  - rash, 1117
- Wart (Verruca), 470
  - moist, 475
  - Peruvian, 834
  - plantar, 471
  - postmortem, 665
  - soft, 562
  - venereal, 475
- Warze (Verruca), 470
  - venerische, 475
- Washerwoman's itch, 872
- Wasp stings, 947
- Wassermann reaction in syphilis, **78–81**, 759
- Wasserpocken (Varicella), 426
- Water, therapeutic use of, 94
- Waters, mineral, 90
- Wen (Steatoma), 1012
- Werlhof's disease, 437
- Wheals, 55. See also Urticaria.
  - giant, 153
- White spot disease, 499
- Whiteness of hair (Canities), 1055
- Whitlow, 1095
  - Hutchinson's melanotic, 821
- Wickham's striæ, 288
- Wilkinson's ointment, 245, 268
- Wilson's dermatitis, 187, **189**
- Winter prurigo (Pruritus hiemalis), 843
- Wood-beetle, 946
- Wood-tick (Ixodes), 946
- Wounds, diphtheria of, 350
- Wrinkled tongue (Grooved tongue), 1116

X

- Xanthelasma, 598, **603–605**
  - diagnosis of, 605
  - etiology of, 604
  - pathology of, 604
  - prognosis of, 605
  - symptoms of, 604
  - treatment of, 605
- Xanthelasmoidea (Urticaria pigmentosa), 149
- Xanthoerythrodermie perstans (Parapsoriasis en plaques), 272
- Xanthoma, **598–607**
  - cells, 602
  - diabeticorum, **605–607**
    - diagnosis of, 607
    - etiology of, 606
    - pathology of, 606
    - prognosis of, 607
    - symptoms of, 606
    - treatment of, 607
  - diagnosis of, 603
  - elasticum, 607
  - etiology of, 601
  - of eyelid, 603
  - multiplex, 598

Xanthoma, pathology of, 602
  planum, 601
  pseudo-elasticum, 607
  solitarium, 601
  symptoms of, 599
  treatment of, 603
Xeroderma, (Ichthyosis simplex), 487
  pigmentosum, **619–625**
    diagnosis of, 624
    etiology of, 622
    lentigo in, 535
    pathology of, 623
    prognosis of, 625
    symptoms of, 620
    treatment of, 625
Xerosis, 487, 1008
X-rays, 108–110
  dermatitis from, **205–208**
  epithelial degeneration from, 70

Y

YAWS (Frambesia), 830

Z

ZANDER'S cells, 47
Zinc chlorid, 100
Zona (Herpes zoster), 355
Zoster (Herpes zoster), 355
Zungenfratt, 1117

Xanthoma, 1
 planum,
 pseudo-
 solitariu
 sympto
 treatm
Xeroderma
 pigmen
  di
  et
  lc
  p
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  s
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Xerosis, 4

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